REPUBLIQUE DU CAMEROUN
Paix – Travail – Patrie
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UNIVERSITE DE YAOUNDE I
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FACULTE DE MEDECINE ET DES SCIENCES BIOMEDICALES

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THE UNIVERSITY OF YAOUNDE I
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FACULTY OF MEDICINE AND BIOMEDICAL SCIENCES

DEPARTMENT OF INTERNAL MEDICINE AND SPECIALTIES

Sleep Disorders among Stroke Survivors at Yaoundé Central Hospital: Prevalence, Associated Factors, and Impact on Functional Status

A thesis presented and publicly defended in partial fulfillment of the requirements for the award of the Doctorate degree in General Medicine

By

MBANGE LIKOWO GERMAINE

Registration number: 17M089

Director

Pr. NJAMNSHI Alfred Kongnyu

Professor in Neurology and Clinical Neurophysiology/Neuroscience

Co-Director

Dr. NGARKA Leonard

Senior Lecturer in Neurology

Academic year 2023- 2024

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Registration number: 17M089

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Academic year 2023- 2024

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DEDICATION

To

My beloved mother Madame BRIDGET NAMONDO NGOMBA and my father

Mr. MBOYA MBANGE JACOB of blessed memory

ACKNOWLEDGEMENTS

I wish to extend my sincere and heartfelt gratitude to:

- GOD Almighty, for His grace, strength, and direction throughout medical school and during the period of this work.
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DEPARTMENT OF PHARMACOTOXICOLOGY AND PHARMACOKINETICS			
258	**ZINGUE Stéphane (HOD)**	AP	Physiology and Pharmacology
259	FOKUNANG Charles	P	Molecular Biology
260	MPONDO MPONDO Emmanuel	P	Pharmacy
261	TEMBE Estella ép. FOKUNANG	AP	Clinical pharmacology
262	TABI OMGBA	SL	Pharmacy
263	NENE AHIDJO ép. NJITUNG TEM	L	Neuro-pharmacology
264	ANGO Yves Patrick	L	Chemistry of natural substances
DEPARTMENT OF GALENICAL PHARMACY AND PHARMACEUTICAL LEGISLATION			
265	**NNANGA NGA Emmanuel (HOD)**	P	Galenic Pharmacy
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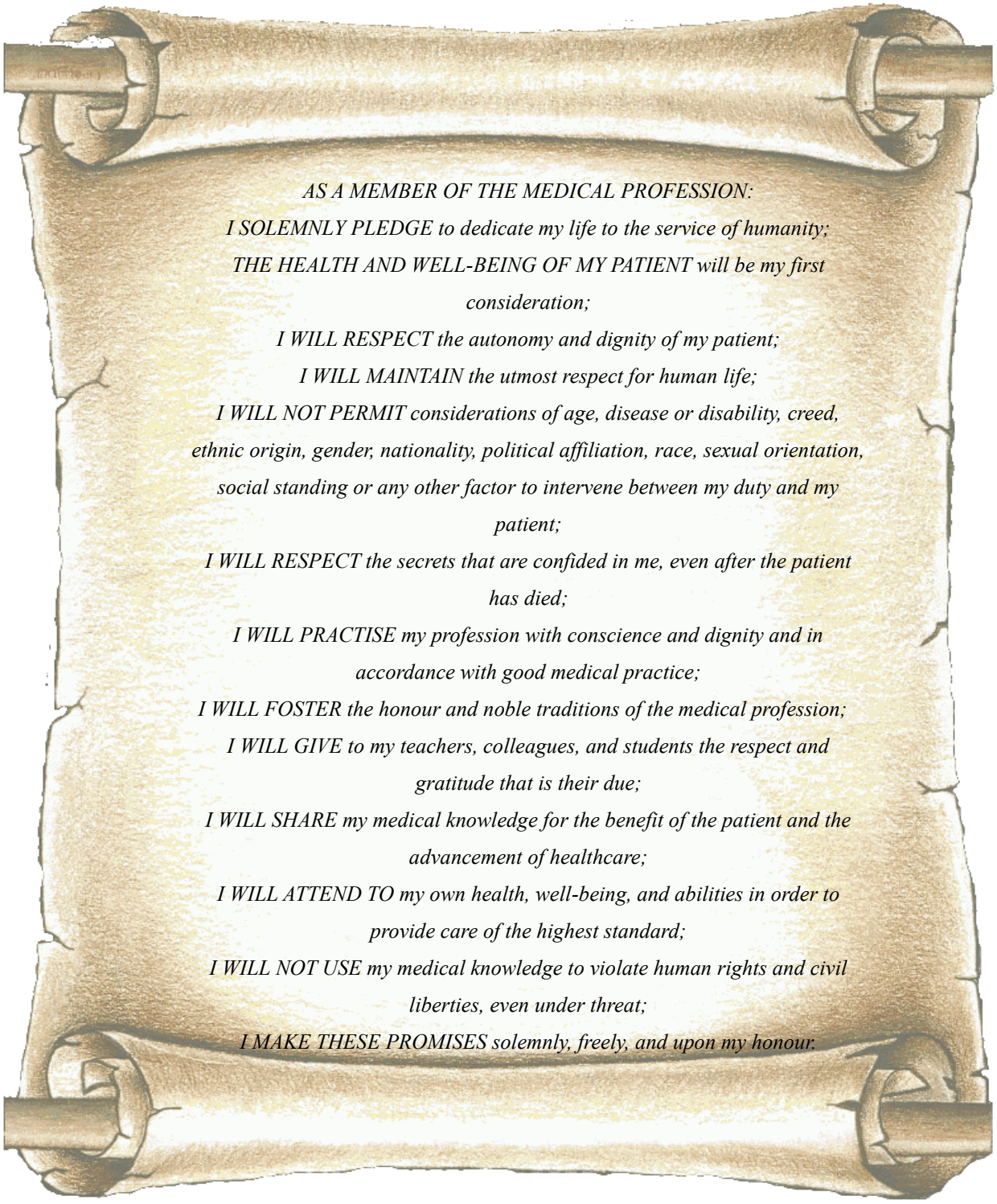
267	SOPPO LOBE Charlotte Vanessa	SL	Quality Control of Drugs
268	NYANGONO NDONGO Martin	SL	Pharmacy
269	MINYEM NGOMBI Aude Périne ép. AFUH	L	Pharmaceutical Regulations
270	ABA'A Marthe Dereine	L	Drug Analysis
271	FOUMANE MANIEPI NGOUOPIHO Jacqueline Saurelle	L	Pharmacology

KEY

HOD = Head of Department / **P**= Professor / **AP**= Associate Professor / **SL**= Senior Lecturer / **L**= Lecturer

THE PHYSICIAN'S OATH

Declaration of Geneva adopted by the Geneva Assembly of the World Medical Association in Geneva, Switzerland, September 1948 and amended by the 68th WMA General Assembly, Chicago, United States, October 2017



AS A MEMBER OF THE MEDICAL PROFESSION:

I SOLEMNLY PLEDGE to dedicate my life to the service of humanity;

THE HEALTH AND WELL-BEING OF MY PATIENT will be my first consideration;

I WILL RESPECT the autonomy and dignity of my patient;

I WILL MAINTAIN the utmost respect for human life;

I WILL NOT PERMIT considerations of age, disease or disability, creed, ethnic origin, gender, nationality, political affiliation, race, sexual orientation, social standing or any other factor to intervene between my duty and my patient;

I WILL RESPECT the secrets that are confided in me, even after the patient has died;

I WILL PRACTISE my profession with conscience and dignity and in accordance with good medical practice;

I WILL FOSTER the honour and noble traditions of the medical profession;

I WILL GIVE to my teachers, colleagues, and students the respect and gratitude that is their due;

I WILL SHARE my medical knowledge for the benefit of the patient and the advancement of healthcare;

I WILL ATTEND TO my own health, well-being, and abilities in order to provide care of the highest standard;

I WILL NOT USE my medical knowledge to violate human rights and civil liberties, even under threat;

I MAKE THESE PROMISES solemnly, freely, and upon my honour.

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LIST OF ABREVIATIONS

ADL	Activity of Daily Living Assessment
AASM	American Academy of Sleep Medicine
AHI	Apnea-Hypopnea Index
AHA/ASA	American Heart Association/The American Stroke Association
ATP	Adenosine triphosphate
ACA	Anterior cerebral artery
AChA	Anterior Choroidal Artery
BP	Blood Pressure
BI	Barthel's Index
CBT-1	Cognitive Behavioral Therapy for Insomnia
CSA	Central Sleep Apnea
CNS	Central Nervous System
CENAME	Centrale Nationale d'Approvisionnement en Médicaments et Consommables Médicaux Essentiels
COPD	Chronic Obstructive Pulmonary disease
CPAP	Continuous Positive Airway Pressure
CSF	Cerebrospinal fluid
DALYS	Disability Adjusted Life Years
DSM-5	Diagnostic and Statistical Manual of Mental Disorders, 5th Edition
ECG	Electrocardiogram
EEG	Electroencephalogram
EDS	Excessive daytime Sleepiness
ESS	Epworth Sleepiness Scale
GABA	Gamma-aminobutyric acid
GBD	Global Burden of Disease
HADS	Hospital Anxiety and Depression Scale
ICA	Internal carotid artery
ICSD-3-TR	International Classification of Sleep Disorders third edition text revision

ICH	Intracerebral Hemorrhage
ICP	Intracranial Pressure
IRLS	International Restless Legs Scale
ISI	Insomnia Severity Index
IVH	Intraventricular Hemorrhage
LDT	Laterodorsal tegmental nucleus
MCA	Middle Cerebral Artery
MEQ	Morningness-Eveningness Questionnaire
MS-SAS	Moderate to severe sleep apnea syndrome
NIHSS	National Institute of Health Stroke Scale
NREM	Non-rapid Eye Movement sleep
OSA	Obstructive Sleep Apnea
PCA	Posterior cerebral artery
PLMD	Periodic Limb Movements Disorder
PNS	Peripheral nervous system
PPT	Pedunculopontine tegmental nucleus
PSQI	Pittsburg Sleep Quality Index
PTSD	Post-traumatic stress disorder
RAS	Reticular Activating system
RBDs	Rapid Eye Movement Behavior Disorders
REM	Rapid Eye Movement sleep
RLS	Rapid Eye Movement Behavior Disorders
SAS	Sleep Apnea Syndrome
SAH	Subarachnoid Hemorrhage
SCN	Suprachiasmatic nucleus
SDB	Sleep disordered breathing
SREDs	Sleep-Related Eating Disorders
SSRI	Selective serotonin reuptake inhibitors
SSS	Standford Sleepiness Scale
SaO2	Oxygen saturation
SWS	Slow-wave sleep
VLPO	Ventrolateral preoptic nucleus

ABSTRACT

Introduction: Stroke is the second leading cause of death and the third leading cause of disability worldwide. Sleep disorders are common in stroke patients and can hinder rehabilitation and lead to poor functional outcomes if left undiagnosed and untreated.

Objectives: This study aimed to describe the epidemiological and clinical aspects of sleep disorders in stroke survivors at Yaoundé Central Hospital (YCH), determine the prevalence and types of sleep disorders, identify associated factors, and assess the influence of sleep disorders on functional status.

Materials and Methods: A descriptive and analytical cross-sectional study was conducted from November 2023 to May 2024. We assessed sociodemographic, clinical, radiological, and motor and functional disabilities. Sleep disorders were assessed using the Pittsburgh Sleep Quality Index (PSQI), Epworth Sleepiness Scale (ESS), and STOP-BANG Questionnaire. Epi Info version 7.2.4.0, was used to data, with statistical significance set at $P < 0.05$

Results: Among the 106 patients, (47.2% were males), the mean age was 60.9±13.1years. The overall prevalence of sleep disorders was 52.8%, with excessive daytime sleepiness being the most common type, affecting 28.3% of the participants. Multiple logistic regression revealed significant associations between age and sleep disorders and age and poor sleep quality ($P = 0.03$ and 0.04, respectively). Male sex, obesity, and prior stroke were significantly associated with a high risk of obstructive sleep apnea ($P = 0.002$, 0.02, and 0.02 respectively). Diabetes was significantly associated to excessive daytime sleepiness ($P = 0.04$), and sedative use was associated with poor sleep quality. Worse modified Rankin Scale scores were significantly associated with both the presence of sleep disorders and a high risk of obstructive sleep apnea ($P = 0.03$ and 0.02 respectively).

Conclusion: The prevalence of sleep disorders in stroke survivors was 52.83%. Identified sleep disorders included excessive daytime sleepiness, insomnia, high risk for sleep apnea, and restless leg syndrome. Factors associated with sleep disorders included age, sex, obesity, diabetes, prior stroke, and sedative use. The presence of sleep disorders was linked to poor functional status, potentially impairing rehabilitation outcomes.

Keywords: *sleep disorders, Stroke, post-stroke sleep disorders, Sleep quality, rehabilitation, associated factors, functional status.*

RESUME

Introduction: L'AVC est la deuxième cause de décès et la troisième cause d'incapacité dans le monde. Les troubles du sommeil sont fréquents chez les patients ayant subi un AVC et, s'ils ne sont pas diagnostiqués et traités, peuvent entraver la réhabilitation et entraîner de mauvais résultats fonctionnels.

Objectifs: Décrire les aspects épidémiologiques et cliniques des troubles du sommeil chez les survivants d'AVC à l'Hôpital Central de Yaoundé, déterminer leur prévalence, types et facteurs associés, ainsi que leur impact sur l'état fonctionnel.

Matériels et Méthodes : Une étude transversale descriptive et analytique a été menée de novembre 2023 à mai 2024, évaluant les caractéristiques sociodémographiques, cliniques, radiologiques, ainsi que les incapacités motrices et fonctionnelles. Les troubles du sommeil ont été évalués à l'aide de ; Indice de Qualité du Sommeil de Pittsburgh (IQSP), Échelle de Somnolence d'Epworth (ESE) et le Questionnaire STOP-BANG. Les données ont été analysées avec une signification statistique fixée à $P < 0,05$.

Résultats : Parmi les 106 patients (47,2 % d'hommes), l'âge moyen était de 60,9±13,1 ans. La prévalence globale des troubles du sommeil était de 52,8%, l'hypersomnie diurne étant le type de trouble du sommeil le plus courant, affectant 28,3 % des participants. L'analyse multivariée a révélé des associations significatives entre l'âge et les troubles du sommeil et la mauvaise qualité du sommeil ($P = 0,03$ et 0,04 respectivement). Le sexe masculin, l'obésité et un AVC antérieur étaient significativement associés à un risque élevé d'apnée obstructive du sommeil ($P = 0,002$, 0,02 et 0,02). Le diabète était associé à la somnolence diurne excessive ($P = 0,04$) et l'utilisation de sédatifs était associée à une mauvaise qualité du sommeil. Les scores modifiés de Rankin (mRs) plus élevés étaient significativement associés à la présence de troubles du sommeil et à un risque élevé d'apnée obstructive du sommeil ($P = 0,03$ et 0,02 respectivement).

Conclusion: Les troubles du sommeil post-AVC sont courants (52.83%), comprenant la somnolence diurne excessive, l'insomnie, l'apnée du sommeil et le syndrome des jambes sans repos. Leur présence est liée à un mauvais état fonctionnel, soulignant l'importance de leur identification et de leur prise en charge dans le processus de réhabilitation. Les facteurs associés aux troubles du sommeil incluaient l'âge, le sexe, l'obésité, le diabète, un AVC antérieur et l'utilisation de sédatifs.

Mots-clés : troubles du sommeil, AVC, troubles du sommeil post-AVC, qualité du sommeil, réhabilitation, facteurs associés, état fonctionnel.

CHAPTER I : INTRODUCTION

I.1. BACKGROUND

Stroke is an acute and focal neurological deficit resulting from an interruption in cerebral blood flow and perfusion by blockage or rupture of one or more blood vessels, lasting more than 24 hours [1]. Globally, Stroke ranks as the second-leading cause of death and the third-leading cause of disability [2]. As per the 2022 Fact Sheet from the World Stroke Organization, there are approximately 12.2 million new cases of stroke reported globally each year, resulting in roughly 6.5 million fatalities and over 143 million Disability-Adjusted Life Years (DALYs). In Africa, published data indicates an annual stroke incidence rate of 316 per 100,000, a prevalence of about 1,460 per 100,000, and a fatality rate exceeding 80%.[3]. Stroke poses a significant challenge in sub-Saharan Africa, including Cameroon. A study conducted in Cameroon found a 7.3% prevalence of stroke, with a mortality rate of 26.7% in the first month, increasing to 31.7% in the first three months following the stroke event.[4].

Stroke has a variety of complications known to affect patients, of which troubled sleep has been found to be among [5]. Sleep is a physiological process characterized by a reversible state of unconsciousness, with skeletal muscle inactivity and decreased response to external stimuli [6]. Sleep is important in improving various physiological functions, such as cognitive functions, memory consolidation, immune system response, emotions, physical abilities, and hormonal regulation [7]. It also has an established role in synaptic plasticity [8], which is essential in stroke recovery. Sleep disorders are classified as sleep-related breathing disorders, sleep-related movement disorders, insomnia, hypersomnia, circadian rhythm sleep-wake disorders, and parasomnias [9].

Studies have reported a bi-directional association between sleep and stroke. Sleep disorders like sleep-disordered breathing have been shown to be both a risk factor for stroke and also a consequence of stroke [10]. Frequently reported sleep disorders in stroke patients include Insomnia, hypersomnia, sleep apnea like OSA and CSA, sleep-related movement disorders like restless leg syndrome, and periodic movement [11]. Certain factors like age, sex, comorbidities, stroke severity, lesion location, and the presence of depression and anxiety have been linked to sleep disorders following a stroke [12–14].

Sleep disorders in stroke patients have been studied using subjective screening and objective methods such as polysomnography. In Africa, including Cameroon, there is relatively lower awareness and research interest in studying sleep disorders among stroke patients compared to the Western world. Available studies in Africa have predominantly focused on subjective

sleep assessments [13, 15]. Some research in Cameroon has revealed associations between sleep quality, sleep disorders, and comorbidities such as hypertension and cardiac conditions [16, 17]. However, there is a paucity of published information on the link between sleep disorders and stroke. Our study aims to raise awareness of sleep disorders in stroke patients in Cameroon.

I.2. JUSTIFICATION

The high prevalence of sleep disorders among stroke patients and their documented negative impact on rehabilitation has sparked increasing interest in sleep studies [18, 19]. The rise in global stroke cases and the subsequent high number of individuals coping with disabilities [2], as well as the substantial economic burden noted in Cameroon [20, 21], make it imperative to address the factors that negatively impact the functional status of stroke patients. This includes sleep disorders.

I.3. RESEARCH QUESTIONS

1. What is the prevalence of different sleep disorders among stroke patients?
2. What are the factors associated with the occurrence of sleep disorders in stroke patients?
3. How do post-stroke sleep disorders impact the functional status of Stroke?

I.4. RESEARCH HYPOTHESIS

1. Sleep disorders are highly prevalent in stroke patients.
2. There are factors associated with the occurrence of sleep disorders in stroke patients.
3. Sleep disorders have a negative impact on the functional status of stroke patients.

I.5. OBJECTIVES

I.5.1. General Objectives

This study aimed to describe the epidemiological and clinical aspects of sleep disorders in stroke patients at the Yaoundé Central Hospital in order to improve the recovery and quality of life of these patients.

I.5.2. Specific Objectives

1. Estimate the prevalence of sleep disorders in stroke patients followed up at the Yaoundé Central Hospital.
2. Identify the different types of sleep disorders in stroke patients.
3. Determine factors associated with sleep disorders in stroke patients.
4. Assess the influence of sleep disorders on the functional status of stroke patients.

I.6. OPERATIONAL DEFINITION OF TERMS

1. **Stroke:** In this study, we considered patients with a documented diagnosis of stroke by a neurologist.
2. **Sleep disorders**: Patients who presented with disorders of sleep as classified by the ICSD-3 ; Insomnia, sleep-related breathing disorders, central disorder of somnolence, circadian rhythm sleep-wake disorders, parasomnias, and sleep-related movement disorders [9].
3. **Post-stroke sleep disorders**: sleep disorders which occurred after the stroke event.
4. **Poor sleep quality**: In our study, poor sleep quality is considered a Pittsburg Sleep quality index of > 5 [15]
5. **Hypertension**: Patient was considered as hypertensive if was diagnosed in the past or if the patient was on antihypertensive medications over the last 15 consecutive days or if the patient had a systolic and/or diastolic blood pressure of 140/90 mmHg and above at the time of interview.
6. **Diabetic patient**: Participants were classified as having diabetes mellitus if they were taking hypoglycemic medications or if their fasting blood glucose levels were > 126mg/dl and/or HbA1c > 6.5%
7. **Cardiopathy**: Participants were said to have a cardiopathy if there was a documented disease on ECG or Echocardiography confirmed by a cardiologist or a known documented past disease. These diseases included myocardial infarction, rheumatic valvular heart disease, prosthetic heart valve, atrial fibrillation or flutter, heart failure, and dilated cardiopathy
8. **Obesity**: In this study participants with BMI $\geq$ 30 kg/m^2 were classified as obese individuals.

9. **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 and at most 6 months post-event [23].

CHAPTER II : LITERATURE REVIEW

II.1. SLEEP

II.1.1. Overview

- **Sleep**

Sleep is defined as a reversible state of unconsciousness, with reduced muscle activity and reduced metabolism [6]. Despite its reduced response to external stimuli, an individual can be aroused by external stimuli, distinguishing it from coma, characterized rather by a state of unconsciousness that cannot be reversed in response to external stimuli [6]. The amount of sleep required for 24 hours varies with age. According to the American Academy of Sleep Medicine, infants need 12 – 16 hours of sleep for optimal health, children need an average of 10 – 13 hours, teenagers need an average of 8 -10 hours, and adults need an average of 7 hours of sleep. Sleep is necessary for the overall productivity and health of an individual. Polysomnography is a primary tool in evaluating the electrophysiological aspects of sleep and wakefulness. Its profile aids in defining 2 stages of sleep: Rapid eye movement sleep (REM) and non-REM sleep. Sleep improves brain function and various other physiologic functions, such as cognition, memory consolidation, learning, and many others[7].

- **Wake** [24]

This is a period of alertness which makes up at least two-thirds of the 24 hour-day. It is characterized by open eyes, movements, conversations, and various other activities. These activities wind down within the course of the day, and people recline and close their eyes. Brain brainwaves are now slowed down to a stable posterior dominant rhythm. This rhythm lies between wake and sleep, and further slowing down the rhythm brings an individual to sleep. The sleep-wake cycle is controlled by 2 biological processes: sleep/wake homeostasis and the circadian biological clock.

II.1.2. Anatomic review of the Nervous system[25]

The nervous system is divided into the central and peripheral nervous systems. The CNS consists of the brain and spinal cord, while the PNS consists of the somatic and autonomic nervous systems. The brain consists of two hemispheres, left and right, which are in constant communication, and each is responsible for different actions. The left hemisphere is usually the dominant hemisphere and is responsible for logic, calculations, and language. On the other hand, the right hemisphere is dominant for creative thinkers like artists.

The Brain consists of the cerebrum, brainstem (midbrain, pons, medulla), and cerebellum.

The brain can equally be divided based on embryologic origin into

1. The telencephalon (also known as the cerebrum) contains the two cerebral hemispheres which are joined by a group of fibers called corpus callosum. The telencephalon includes the following structures

- Cortex
- Subcortical structures (closely related structures called the basal ganglia; the caudate nucleus, putamen, globus; amygdala; hippocampus)

2. The diencephalon consists of the thalamus, epithalamus (which contains the pineal gland), hypothalamus, and subthalamus. The telencephalon and Diencephalon stem from the prosencephalon, also called the forebrain.
3. The mesencephalon consists of; structures surrounding the cerebral aqueduct like the periaqueductal gray, the reticular formation, the cerebral peduncles, superior and inferior colliculi, substantia nigra, some cranial nerve, red nucleus projection of sensory and motor pathways
4. The metencephalon consists of; the pons and cerebellum.
5. The myelencephalon consists of the medulla, projection of sensory and motor pathways, and some cranial nerve nuclei. The mesencephalon's caudal end develops into the spinal cord.

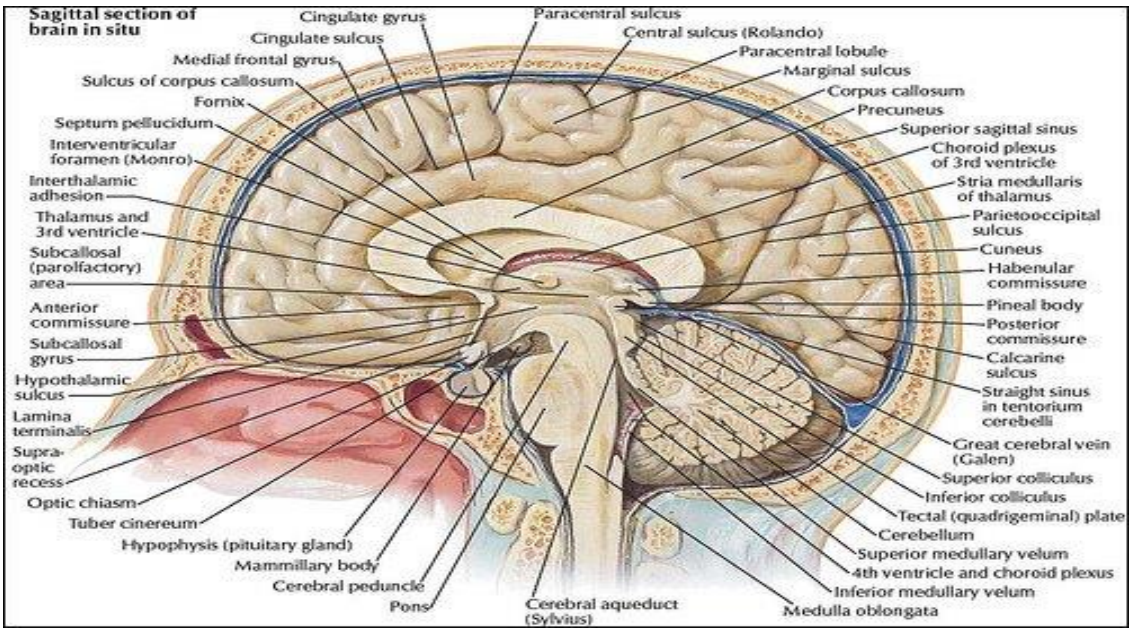

Figure 1: Sagittal section of the brain in situ. Source: Atlas of Human Anatomy, Sixth Edition Frank H. Netter, M. D [25]

II.1.3. Neuroanatomy and Neurobiology Sleep-wake cycle[26]

The sleep-wake cycle is made up of a bistable, on/off circuit. This circuit consists of

- An ascending arousal pathway and
- A sleep-inducing Ventrolateral preoptic nucleus pathway (VLPO).

Each half of the circuit strongly inhibits the other half, leading to a stable on-and-off pattern. Injury to either side of this circuit leads to an unwanted instability between sleep and wake states regardless of the affected site.

The ascending arousal pathway consists of major cell populations that are involved in prompting arousal: pedunculopontine and laterodorsal tegmental nucleus (PPT/LDT) made up of cholinergic neurons, locus coeruleus made up of noradrenergic neurons, dorsal and median raphe nucleus entailing serotoninergic and dopaminergic neurons respectively, and tuberomammillary nucleus made up of histaminergic neurons. The sleep-inducing pathway consists of the ventrolateral preoptic nucleus (VLPO), which comprises sleep-active γ-aminobutyric acid (GABA)-ergic and galaninergic neurons.

➢ **<u>Ascending arousal system</u>**

It originates in the upper brainstem adjacent to the junction point between the pons and the midbrain projecting towards the diencephalon, then the cortex. There are two major branches of this system (Fig .2. A) ;

Cortical arousal occurs through 2 branches;

- The first branch contains PPT/LDT neurons, which are cholinergic structures in the brain stem and basal forebrain. They innervate the thalamus, activating relay neurons and reticular nucleus, which are essential for thalamocortical transmission and leading to wakefulness.
- The second branch of the ascending arousal system, which projects into the lateral hypothalamus, basal forebrain, and cerebral cortex, consists of monoaminergic cell populations such as noradrenergic, serotoninergic, dopaminergic, and histaminergic neurons. Its neurons have broad action potentials, discharging rapidly during wakefulness, slowing during NREM sleep, and showing little activity during REM sleep. Additional cerebrocortices afferents have been identified: basal forebrain nuclei containing GABA or acetylcholine and lateral hypothalamic peptidergic neurons containing melanin-concentrating hormone or

orexin/hypocretin[26]. Orexin-containing neurons in areas close to histamine neurons project extensively to most brain areas and are linked to arousal[27]. Orexin deficiency has been connected to daytime sleepiness and unplanned episodes of sleep[28].

- **Sleep structural pathways**

γ-aminobutyric acid (GABA) and galanin are found in the ventrolateral preoptic nucleus (VLPO) and initiate sleep. These neurotransmitters are activated by the anterior hypothalamus, which provides circadian input and endogenous chemical signals (such as adenosine) that build up in response to awake time, leading to sleep-wake homeostatic information. VLPO neurons aggregate densely and diffusely innervate the brainstem and hypothalamus's monoaminergic systems, which are involved in cortical arousal regulation (Fig. 1 B)[26, 28]. The mammalian brain's activity in calming the ascending monoaminergic arousal system during sleep has been linked to VLPO efferent, with the help of inhibitory neurotransmitters galanin and GABA[26, 27, 29]

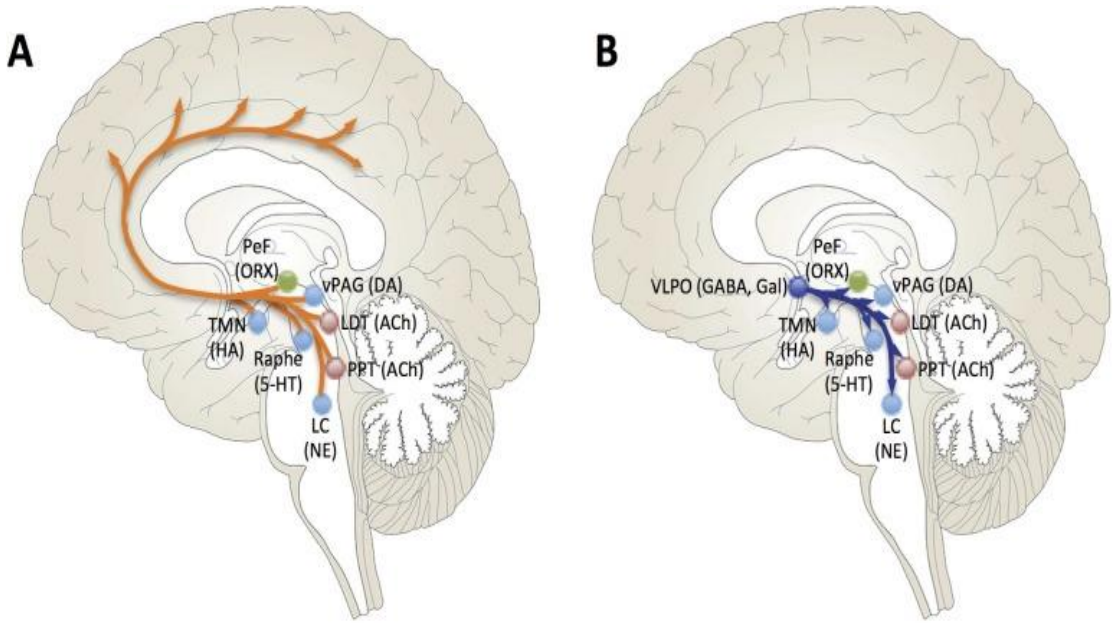

Figure 2: Brain networks regulate sleep and wakefulness [28]

Panel A depicts key elements of the ascending arousal systems, with diffuse excitatory projections to the cortex. Panel B shows pathways arising from the hypothalamus that inactivate the ascending arousal system during sleep. ACh, acetylcholine; DA, dopamine; GABA, gamma amino-butyric acid; Gal, galanin; HA, histamine; LDT, laterodorsal tegmentum; NE, norepinephrine; ORX, orexin; PeF, perifornical region; PPT,

pedunculopontine tegmentum; TMN, tuberomammillary nucleus; vPAG, ventral periaqueductal gray matter; 5-HT, 5-hydroxytryptamine[28]

II.1.4. Normal Sleep Architecture [28]

➢ **Sleep Stages**

There are two major phases of sleep: non-rapid eye movement (NREM) and rapid eye movement (REM) sleep. NREM is further divided into N1, N2, N3. They are all associated with particular brain waves and neural activity. A normal night's sleep involves many cycles through all stages of non-REM and REM sleep, with progressively longer and deeper REM episodes happening in the early hours of the morning. Sleep cycles average four to five cycles per night, with the following order of sleep stage progression: N1, N2, N3, N2, REM. It takes 90–110 minutes to go through a whole sleep cycle. In the first sleep cycle, REM sleep is short. REM sleep becomes progressively longer throughout the night while the duration of NREM sleep reduces.

- **NREM SLEEP**

It is divided into 3 N1, N2, N3.

✓ **N1 sleep** – light sleep (5%)

EEG recording: low-voltage theta waves of frequency 4-7 hz.

This stage of sleep is the lightest and starts the sleep cycle when low-amplitude mixed-frequency (LAMF) activity replaces more than 50% of the alpha waves, which are more characteristic of the awake stage. It marks the transition from wakefulness to sleep. Here, skeletal muscle tone is present (which eventually decreases progressively throughout the cycle), and breathing happens at a regular pace. This phase, which accounts for 5% of the overall sleep duration, lasts for one to five minutes[6, 28].

✓ **N2 sleep – Deeper sleep (45% of sleep cycle)**

EEG recording: sleep spindles and K complexes.

This stage is characterized by a decrease in body temperature and heart rate, which indicates deeper slumber. K-complexes, sleep spindles, or both may be present, which is what distinguishes it. The thalamus, anterior cingulate, superior temporal gyri, and insular cortices all experience short, intense bursts of neuronal firing during sleep, which cause calcium to

enter cortical pyramidal cells[28]. It is thought that this process is essential for synaptic plasticity. Sleep spindles are implicated in memory consolidation, particularly in declarative and procedural memory, according to several studies[28].

K-complexes are the longest and most unique brain waves; they are long delta waves lasting around a second. K-complexes have been demonstrated to help memory consolidation and maintain sleep [30]. The first cycle of stage 2 sleep lasts roughly 25 minutes, and it gets longer with each subsequent cycle until it makes up roughly 45% of the entire amount of sleep[28].

✓ **N3 sleep – Deepest Non-REM Sleep (25% of sleep cycle)**

EEG recording: delta waves - lowest frequency, highest amplitude (0.3 – 5 Hz).

N3 can also be referred to as **slow-wave sleep (SWS**). It is characterized by delta waves, which are signals with significantly lower frequencies and higher amplitudes and are indicative of the deepest state of sleep. This is the hardest stage to wake up from, and some people are not even awakened by loud noises (over 100 dB). People often spend more time in stage N2 sleep and less time in this sluggish, delta-wave sleep as they get older. Even though this stage has the highest arousal threshold, if someone is awakened during it, they will experience sleep inertia, which is a brief period of mental disorientation. According to cognitive testing, people who awaken during this stage typically have mild impairment in their mental function for a duration of thirty to sixty minutes. During this phase, the body creates bone and muscle, heals and regenerates damaged tissues, and fortifies the immune system. Additionally, bedwetting, sleepwalking, and night terrors happen during this phase[28].

- **REM Sleep** – 25% of total sleep

EEG recording: beta waves which are likened to brain waves during wakefulness

REM is not thought of as a restful sleep state; instead, it is connected to dreaming.

✓ This phase begins about 90 minutes after you go to sleep, during which time your REM periods lengthen over the course of the night. Usually, the first phase lasts 10 minutes, while the last one might last up to an hour

✓ Except for the eyes and diaphragmatic breathing muscles, which are still functioning, the skeletal muscles are atonic and immobile despite the EEG being comparable to that of an awake person.

✓ Here, respiration starts to get increasingly erratic and irregular.

✓ Dreaming, nightmares, and penile/clitoral tumescence happen during REM sleep.

✓ Compared to slow wave sleep (SWS), a person is harder to arouse using sensory inputs.

✓ During REM sleep, the brain is very active.[28].

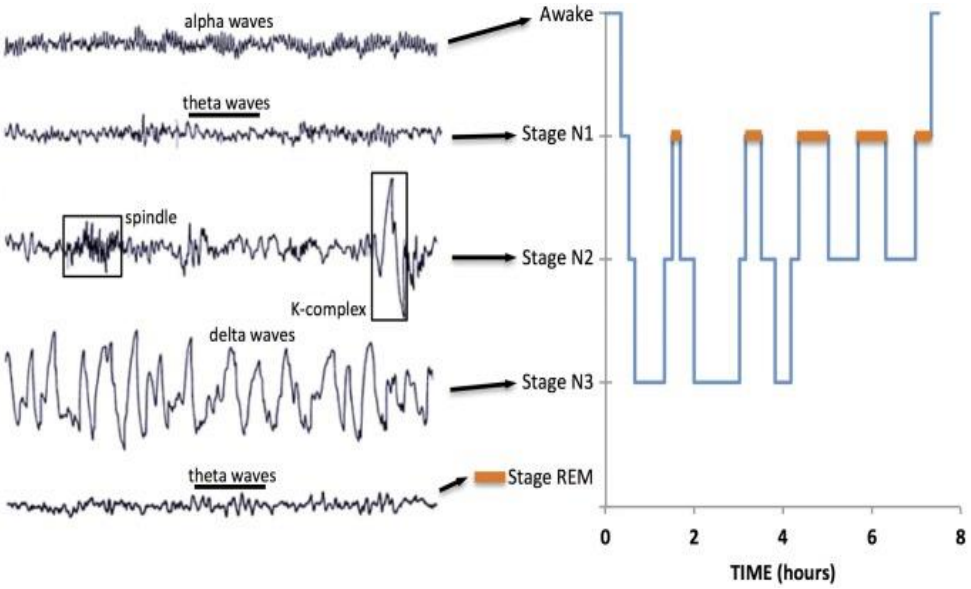

Figure 3: EEG features of sleep/wake stages (left) and typical temporal organization [28]

- **Physiologic modifications of sleep** [6, 28]

Table I: Physiologic modifications of sleep

	NREM SLEEP	REM SLEEP
Respiratory system		
Pharyngeal muscle	Reduced activity	Activity markedly reduced
Respiratory rate	Regular and decreases	variable with apneas
Cardiovascular system		
Blood pressure	Decreases	Variable increases slightly
Heart rate	Decreases	Variable
Endocrine system		
Growth hormone	Peak secretion	No peak in secretion
Renin angiotensin system	Increase in activity	Activity increases
Cortisol	Activity decreases	Activity increase
Thyroid-stimulating hormone	Decreases	Increases slightly
Melatonin	Increases	decreases
Body temperature	Is regulated at a lower set point from wakefulness	Not regulated, temperature drifts towards that of the local environment
Brain	Decreases	Increases and variable
Brain activity	Conceptual	Hallucinatory
Mental activity	Conserved N1 sleep	Decreases significantly
Responsiveness to external stimuli	Decreases in SWS	
awakening	easy (light sleep i.e., N1)	Easy
	unpleasant (N3 sleep)	
Reproductive system	Sexual arousal occurs infrequently	Penile erections
		Increased vaginal blood flow

➢ **Sleep regulation**[26]

Sleep is governed by two separates but overlapping systems: sleep/wake homeostasis and the circadian system.

- **Homeostatic control of sleep**

Sleep homeostasis is a physiological process that occurs during prolonged wakefulness, where sleepiness and tension increase. Adenosine, an endogenous factor produced in neurons and glia, is a key mediator of sleep homeostasis. It accumulates in the extracellular space and can regulate the sleep-wakefulness cycle circuits. Caffeine, an adenosine receptor antagonist, promotes alertness by blocking the adenosine A2A receptor. Nitric oxide, a neuromodulator, is synthesized by inducible nitric oxide synthase in the basal forebrain during prolonged wakefulness. Persistent wakefulness lengthens the cycle of neuronal activity in various brain areas, increasing energy consumption. Sleep homeostasis may be linked to energy exhaustion due to prolonged neuronal activation during prolonged wakefulness. Recovery sleep after sleep restriction improves less in older animals, and increases in adenosine, nitric oxide, and lactate concentrations are blunted in older individuals, suggesting that the main regulators of sleep homeostasis and homeostatic sleep responses are affected by aging[26].

- **Circadian regulations of the human sleep-wake cycle**

The regulation of sleep and wakefulness is influenced by the circadian clock, which is regulated by the suprachiasmatic nucleus (SCN). The SCN generates circadian rhythms in clock gene expression, which are higher during the day and lower at night[26].

Daytime SCN electrical activity is higher, whereas nighttime activity is reduced. The circadian rhythm in the SCN is regulated by hormones and neurotransmitters. The SCN, for instance, harmonizes and adjusts the endogenous rhythm of melatonin release, a hormone produced and secreted by the pineal gland during the night under dark conditions. One of melatonin's primary physiological functions is to tell bodily structures about the daily cycle of light and dark[31]. The physiological processes that respond to changes throughout exposure to light are harmonized with this knowledge, and SCN neurons project to specific brain regions to control various physiological activities. The sleep-wake cycle progresses from intervals of wakefulness to NREM sleep to REM sleep, reflecting the functioning of various neuronal systems. Activating neurons containing glutamatergic nitric oxide synthase 1 promotes

wakefulness while activating GABAergic neurons in the ventral tegmental area promotes long-lasting, non-REM-like sleep.

A two-process model has been proposed for the regulation of sleep and wakefulness, with process S representing sleep pressure and process C regulated by the circadian clock[32].

II.1.5. Sleep evaluation

There are various methods to explore sleep[33–35]:

- **Subjective methods**

- Sleep Diary: A personal written account of one's waking and sleep patterns over several weeks or months is called a sleep diary or sleep log. Patients keep a thorough sleep diary that includes information on when they go to bed, how long it takes them to fall asleep, how often they wake up, and when they take naps. This sleep schedule will have to be completed every morning for a week. It allows the discussion of the patient's habits but does not provide information about sleep cycles and their durations.
- Tests/questionnaires: assess the quality of sleep, daytime drowsiness, and severity of the disorders. The most commonly used are the Pittsburgh index (PSQI)[36] and the Epworth index to assess daytime sleepiness. There are many questionnaires, and most are assigned to evaluate various types of sleep disorders: the berlin questionnaire and STOP-BANG questionnaire(sleep apnea assessment), PSQI (sleep quality assessment and insomnia assessment), International Restless Legs Scale, IRLS (Restless Legs syndrome), Morningness-Eveningness Questionnaire (MEQ) – assessing circadian rhythm, Insomnia Severity Index(ISI) – for assessing insomnia, Epworth Sleepiness Scale (ESS) and Standford Sleepiness Scale (SSS) – for assessing excessive daytime sleepiness, Cataplexy questionnaire (for assessing narcolepsy) [36–39].

- **Objective methods**

- Polysomnography (reference method): recording of EEG, eye movements (EOG), muscle tone (EMG of chin muscles), breathing, snoring, SaO2, leg movement ± video.
- Ventilation polygraphy: recording of breathing, snoring, and SaO2.
- Actigraphy: it complements the sleep agenda data. An actigraph gadget is worn on the wrist, much like a watch, during this test. Movement triggers the signals, while periods of inactivity or sleep result in very few or no signals being recorded. Its measurements,

sometimes inadequate, allow us to study the total duration of the period of inactivity and awakening during the night. By measuring circadian rhythm or sleep-wake cycles over a lengthy period of time, this gadget can identify advanced or delayed sleep phase syndrome.

- Multiple sleep latency testing (MSLT): This objective test assesses the level of drowsiness[40]. Many refer to this examination as a nap study. The patient is instructed to sleep for eight to ten hours the day after an overnight PSG exam, taking four or five naps. Every nap lasts roughly twenty minutes. These examinations aid in determining the reasons behind excessive daytime sleepiness, which is a symptom of a number of illnesses, including narcolepsy, sleep apnea, and hypersomnia.

II.1.6. Sleep disorders[9]

The most widely used classification scheme for sleep disorders is the International Classification of Sleep Disorders (ICSD) [9]**.** There are six main types of sleep disorders in the third edition of the ICSD (ICSD-3-TR) text revision, which are;

✓ **Insomnia Disorders**: (Chronic insomnia disorder, short-term insomnia disorder)

✓ **Sleep-Related Breathing Disorders**: (Central sleep apnea disorders, Obstructive sleep apnea (OSA) disorders, Sleep-related hypoventilation disorders, sleep-related hypoxemia disorder)

✓ **Central Disorders of Hypersomnolence:** (Narcolepsy type 1, Narcolepsy type 2, Idiopathic hypersomnia, Kleine-Levin syndrome, Hypersomnia due to a medical disorder, Hypersomnia due to a medication or substance, Hypersomnia associated with a psychiatric disorder, Insufficient sleep syndrome).

✓ **Circadian Rhythm Sleep-Wake Disorders**: (Shift work disorder and jet lag disorder, Delayed sleep-wake phase disorder, Advanced sleep-wake phase disorder, Irregular sleep-wake rhythm disorder, non-24-hour sleep-wake rhythm disorder, Circadian sleep-wake disorder.

✓ **Parasomnias: (**non-rapid eye movement (NREM)-related parasomnias, rapid eye movement (REM)-related parasomnias, and other parasomnias).

✓ **Sleep-related movement Disorder** (Restless legs syndrome, Periodic limb movement disorder, Sleep-related cramps, Sleep-related bruxism (teeth grinding), Sleep-related rhythmic movement disorder, Benign sleep myoclonus of infancy, Propriospinal myoclonus at sleep onset, Sleep-related movement disorder due to a medical disorder, Sleep-related movement

disorder due to a medication or substance, Sleep-related movement disorder, unspecified).

II.1.6.1. Insomnia [41]

- **Definition**: Insomnia is defined as difficulty falling or staying asleep accompanied by daytime impairments [41].
- **Epidemiology**:

Most common sleep-wake disorder (global prevalence ~ 10%). Prevalence is higher in women, shift workers, and people with physical or mental disorders or disabilities. [42]

- **Etiology**: intricate and poorly understood [41]

Risk factors include:

✓ A persistently high level of both physiological and cognitive alertness

✓ Medical comorbidities: Insomnia has been found to be associated with quite a good number of medical conditions or related to medications targeted to treat these conditions, some of which include pulmonary diseases like COPD, hypertension, diabetes, Cancer, Chronic pain, heart disease, Neurologic conditions like Parkinson disease, dementia

✓ Psychological disorders: anxiety, depression, substance use disorders, and post-traumatic disorders.

✓ Intrinsic or genetic factors: older age, Female sex (especially peri- and postmenopausal), Previous episode of insomnia, Family history of insomnia, Predisposition toward being more easily aroused from sleep, Trait sleep reactivity (i.e., the propensity for exaggerated sleep disruption in response to stressful events).

✓ Medication and substances - Central nervous system stimulants, such as caffeine, methylphenidate, amphetamine and modafinil; Respiratory stimulants, such as theophylline; Appetite suppressants; Antidepressants, Monoamine oxidase inhibitor antidepressants, Selective serotonin reuptake inhibitors, such as fluoxetine, are associated with insomnia in 5 to 35 percent of patients; Norepinephrine and dopamine reuptake inhibitors, such as bupropion; Serotonin and norepinephrine reuptake inhibitors, such as venlafaxine; Most tricyclic antidepressants are sedating, but some (e.g., protriptyline); Beta-blockers, such as propranolol, metoprolol, and pindolol, can produce sleep-onset insomnia, increased awakenings, and dreams; Glucocorticoids, such as prednisone and dexamethasone[57]; Alcohol, caffeine, and tobacco; Over-the-counter medications, such as nasal decongestants and cold medicine.

✓ Precipitating (acutely triggering) factors: for example, stressful situations (either acute or chronic)Perpetuating factors: e.g., Bad sleeping habits.

- **Clinical features** [41]

✓ Difficulty getting to sleep or initiating, staying asleep, or waking up early.

✓ Compromised day-to-day performance fatigue, cognitive impairment disorder in mood difficulties in social, scholastic, or professional functioning, behavioral problems like impulsivity, hyperactivity, reduced motivation or energy.

✓ Non-refreshing sleep.

✓ Negative effects on health; (Suicide risk and mood disorder development, Injuries sustained at work, decreased quality of life; cognitive function and performance impairment; cardiovascular risk and mortality

- **Diagnosis** [43]

Diagnostic criteria for insomnia disorder

Table II: Diagnostic criteria for insomnia disorder 5th edition.

	DSM-5 criteria
Nighttime symptoms **(presence of ≥ 1 feature)**	• Difficulty initiating sleep (initial or sleep-onset insomnia) • Difficulty maintaining sleep (middle or sleep-maintenance insomnia), i.e., frequent or prolonged awakening from sleep • Early-morning awakening with inability to return to sleep (late or sleep-offset insomnia)

Additional considerations	• Symptoms occur despite adequate time and environment for sleep.
	• Symptoms cannot be attributable to:
	  ○ Use of a substance or medication
	  ○ Medical or psychiatric comorbidities or other sleep disorders

Interpretation	• Symptoms occur ≥ 3 times per week
	  ○ Episodic insomnia: 1–3 months
	  ○ Persistent insomnia: ≥ 3 months

	DSM-5 criteria
	○ Recurrent insomnia: Patient meets criteria for episodic insomnia at least 2 times within 1 year

- **Management**[44]

- **All patients**
  - ✓ Counselling on sleep hygiene education.
  - ✓ Ensure optimal management of coexisting conditions
- **Short-term insomnia**
  - ✓ Managing triggers.
  - ✓ A brief course of pharmacotherapy for insomnia
- **Chronic insomnia**
  - ✓ First line: multicomponent cognitive behavioral therapy for insomnia (CBT-I)
  - ✓ When CBT-I is not successful:
  - ✓ Reassessment of the diagnosis (e.g., considering other sleep-wake disorders), comorbidities, and worsening factors.
  - ✓ Pharmacotherapy for select patients.

➢ **Nonpharmacological management**

A variety of nonpharmacological strategies can enhance sleep, and their combined effects are more potent.

Table III: Behavioral and cognitive therapies for insomnia.

	Goal	**Techniques**
Sleep hygiene	• Prevention of triggering factor or exacerbating factors of insomnia.	• Reserving the bed/bedroom only for sleep or sex. • Refraining from alcohol, caffeine, and nicotine close to bedtime. • Exercising regularly, avoiding doing so 3–4 hours before bedtime. • Avoiding daytime naps. • Avoiding heavy meals and fluid intake in the evenings. • Avoiding bright lights (including electronic screen use) before bedtime.
Stimulus control	• Maintaining an association between bed/bedroom and sleep • Creating a consistent sleep/wake schedule.	• Avoid engaging in other activities in bed. • Maintaining a regular sleep/wake cycle (wake up at the same time every day, avoid naps). • Going to bed until you feel sleepy. • If unable to fall asleep within 20 minutes: Leave the bedroom to sit somewhere quiet and only return when sleepy.
Cognitive therapy	• Identifying and reframing unhealthy sleep-related beliefs.	• Psychotherapy and education • Paradoxical intention • Cognitive behavior therapy, which helps patients with persistent insomnia by lowering their stress and anxiety levels when they can't sleep. In order to benefit from this treatment, the patient is encouraged to maintain good sleep hygiene, which includes avoiding stimulants close to bedtime, exercising, and getting as much light and darkness as possible during the day. • Journaling, which includes mental notes
Sleep restriction	• Avoid delaying sleep	• Suggest to the patient that bedtime should only be used for sleeping and that bedtime should be increased gradually.
Relaxation training	• Diminish anxiety and physical and cognitive arousal	• Examples include: o Progressive muscular relaxation o Visual visualization o Biofeedback

➢ **Pharmacotherapy for insomnia** [45]

- Overall, the data showing the advantages of medication in treating insomnia is not very strong.
- Commonly used medications include, for instance:

✓ Sleep-onset insomnia
  - Melatonin, ramelteon
  - Z-drugs (eszopiclone, zaleplon, zolpidem)
  - Benzodiazepines (preferably short-acting benzodiazepines like triazolam)
  - Suvorexant (orexin antagonist)

✓ Sleep-maintenance insomnia: Z-drugs (eszopiclone, zolpidem), doxepin, suvorexant

✓ Early-morning awakening: doxepin, suvorexant

✓ Older adults (> 65 years old): doxepin, melatonin, ramelteon

✓ Comorbid depression: doxepin, mirtazapine, trazodone[45]

II.1.6.2. Hypersomnolence[46]

- **Definition:**

The International Classification of Sleep Disorders, Third Edition, Text Revision (ICSD-3-TR) defines **Excessive daytime Sleepiness** (EDS) as the inability to maintain wakefulness and alertness during the major waking episodes of the day, with sleep occurring unintentionally or at inappropriate times almost daily for at least three months[9].

In the ICSD-3-TR, **hypersomnolence** describes symptoms including excessive sleepiness and increased sleep duration, while hypersomnia refers to specific disorders characterized by hypersomnolence.

Idiopathic hypersomnia is a central disorder of hypersomnolence, characterized by at least three months of uncontrollably strong sleep demands or daytime lapses into sleep or sleepiness.

A subjective loss of physical or mental energy is referred to as **fatigue**. Clinical fatigue varies depending on the patient and is dependent on three factors: reduced ability to sustain activity (easy fatigability); difficulty focusing, remembering details, and maintaining emotional stability; and difficulty initiating activity (perception of generalized weakness in the absence of objective findings[47].

- **Epidemiology**:

In Africa, excessive daytime sleepiness, or EDS, is a prevalent issue. Numerous populations have high rates of EDS, according to studies. It was discovered that 22.1% of patients with atopic dermatitis (AD) also have EDS [48]. EDS was prevalent in 62.78% of hypertensive sub-Saharan Africans [17]. EDS was prevalent in patients with chronic renal disease in 56% of cases [49]. In Burkina Faso, the overall adult population had a prevalence of EDS at 9.6%[50]. These results demonstrate the substantial impact of EDS in Africa and the necessity of additional studies and initiatives to deal with this problem.

- **Etiology**
  - ✓ Genetic (may be autosomal dominant)
  - ✓ Head trauma
  - ✓ Insufficient sleep
  - ✓ Depression
  - ✓ Medications: benzodiazepines, nonbenzodiazepine sedatives, antihistamines, anticonvulsants opioid analgesics, alcohol abuse, narcotics .
  - ✓ Comorbid medical and psychiatric disorders: e.g., Parkinson's disease, hypothyroidism.
  - ✓ Other sleep disorders like sleep-related breathing disorders, circadian rhythm disorders.
- **Classification**
  - ✓ Acute: < 3 months
  - ✓ Chronic: ≥ 3 months
- **Clinical features**

Clinical features

From History, there will be a complaint of fatigue, low energy or weakness.

- ✓ Excessive sleep (with decreased sleep quality)
- ✓ Difficulty awakening from sleep
- ✓ Sleep inertia (impaired alertness or excessive fatigue after waking)
- ✓ Automatic behaviors (with no memory of the episode after waking

- ✓ Patients could feel sleepy in low-stimulus activities like long drives, watching television, and completing desk work.

On physical examination; Findings like yawning, difficulty in maintaining, eyes open, poor concentration.

- **DSM-5 diagnostic criteria**[43]

1. Excessive sleepiness despite ≥ 7 hours of sleep with at least one of the following:
   - ✓ Recurrent periods of sleep on the same day
   - ✓ > 9 hours of sleep that is nonrestorative
   - ✓ Impaired alertness after awakening
2. Symptoms occur ≥ 3 days/week for ≥ 3 months
3. Symptoms cause functional impairment or distress
4. Symptoms not caused by an underlying substance or medication use
5. Symptoms occur despite having enough time to sleep
6. No underlying or coexisting psychiatric or medical disorder that explains symptoms.

Epworth Sleepiness Scale: This is a subjective test equally used to diagnose EDS. a one-page survey asking participants to rate how likely they are to have recently dozed off or fallen asleep in eight sedentary scenarios, such as reading while sitting, spending an hour in a car without stopping, or relaxing after lunch without drinking.

- **Treatment**
  - ✓ **Encourage good sleeping habits**
  - ✓ Regularly scheduled naps
  - ✓ First-line therapy: modafinil or methylphenidate [51]
  - ✓ Second-line therapy: atomoxetine

II.1.6.3. Parasomnias[52]

- **Definition**

A parasomnia is an unwanted physical occurrence (complex movements, behaviors) or experience (emotions, perceptions, dreams) that happens just before, during, or right after falling asleep[9]

- **Classification**

The International Classification of Sleep Disorders, 3rd text revision, classifies parasomnia as divided into non-rapid eye movement (NREM)- related parasomnias, rapid eye movement (REM)- related parasomnias, and other parasomnias.

Table IV: Classification of parasomnias according to the International Classification of Sleep Disorders-3[52]

A	NREM-related parasomnia
1.	Confusional arousals
2.	Sleepwalking
3.	Sleep terrors
4.	Sleep-related eating disorder
B	REM-related parasomnia
1.	REM sleep behavior disorder
2.	Recurrent isolated sleep paralysis
3.	Nightmare disorder
C	Other parasomnias
1.	Exploding head syndrome
2.	Sleep-related hallucinations
3.	Sleep enuresis
4.	Parasomnia due to a medical disorder
5.	Parasomnia due to medical or substance abuse
6.	Parasomnia, unspecified

NREM: non-rapid eye movement, REM: rapid eye movement

- **Clinical features of different types of parasomnias**[52]

➢ **NREM-related parasomnias**—NREM-related parasomnias are disorders of arousal. They consist of sleepwalking, sleep terrors, confusional arousal, and sleep-related eating disorders.

✓ **Confusional Arousals**

Confusional arousals, also known as sleep drunkenness, Elpenor's syndrome, or morning sleep inertia, are partial awakenings that occur during the third stage of slow-wave sleep. This results in a confused and disoriented person exhibiting automatic behaviors, such as opening their eyes or murmuring,

without any motor activity or sympathetic hyperactivity (which differentiates it from sleep terrors and sleepwalking). This state can last several minutes to many hours, and the individual may completely forget about the incident. Alcohol misuse or the use of sedative-hypnotic drugs can trigger confusional arousal. Children experience confusional arousal more frequently than adults [52, 53].

✓ **Sleep Walking**

Sleepwalking, also known as somnambulism, is a type of arousal disorder that causes a person to engage in ambulatory behavior during stage three of sleep. During these episodes, the person may appear disoriented and may walk aimlessly, play an instrument, perform inappropriate actions such as urinating in the closet, drive or leave the house, or even harm themselves by walking off a balcony. These episodes can be alarming and may interfere with the sleep of anyone sharing the same bed. [52, 53]. Adult sleepwalking is often caused by a range of factors such as sleep disorders (such as apnea and restless legs syndrome), head trauma, encephalitis, fever, vitiligo, migraines, stroke, and chronic pain syndrome. Moreover, certain medications can also lead to sleepwalking in adults as a side effect. These medications include paroxetine, amitriptyline, bupropion, benzodiazepine receptor agonist (which can result in forgetfulness related to zolpidem), propranolol, metoprolol, topiramate, montelukast, and fluoroquinolones.

✓ **Sleep Terror**

The terms "sleep terror," "night terror," or "pavor nocturnus" describe sudden episodes of arousal accompanied by intense motor activity and increased autonomic activity, such as shouting and crying out of fear. Autonomic hyperactivity symptoms include dilated pupils, sweating, rapid breathing, and rapid heartbeat. Patients who experience these episodes are

often scared and upset and may have difficulty remembering the incident clearly. These episodes can also disrupt the sleep of family members and bedmates. [52, 53].

✓ **Sleep-related eating disorders**

Sleep-related eating disorders (SREDs) are a type of disorder in which people experience recurrent episodes of compulsive binge eating after partially waking up from sleep. These episodes involve consuming various food items, such as chocolates, carbohydrates, raw meat, or pet food, while losing memory of the events. These episodes can result in abnormal lipid levels, dental cavities, weight gain, injuries, and diabetes. It's important to distinguish SREDs from other eating disorders like binge eating disorder and nocturnal eating syndrome, which involve large food consumption before bedtime. Medical conditions such as encephalitis, narcolepsy, autoimmune hepatitis, quitting smoking, substance addiction, and certain medications such as anticholinergics, lithium, zolpidem, mirtazapine, and quetiapine may also cause SRED [53].

➢ **REM-related parasomnias** — REM-related parasomnias include the exaggeration of REM sleep characteristics (e.g., nightmare disorder), the incursion of REM sleep features into awake (e.g., sleep paralysis), or abnormalities of REM sleep physiology (e.g., lack of atonia as reported in REM sleep behavior disorder).

✓ **REM Sleep Behavior Disorder**

Dream enactment behavior is a condition that causes an increase in motor activity during REM sleep, which is a characteristic of Rapid Eye Movement Behavior Disorders (RBDs). This can involve kicking, punching, shouting, jumping, or engaging in other violent conduct that is consistent with the dream, which could hurt the bedmate or oneself. Most of the time, the dream is either vaguely remembered or the folks can recall the dream. In contrast to disorders of arousal, patients with RBD typically wake up abruptly at the end of an episode. There are numerous neurologic conditions, such as multiple sclerosis, narcolepsy, cerebral tumor, subarachnoid hemorrhage, pontine stroke, and α-synucleinopathies, that have been linked to RBDs. RBD can also be brought on by drugs such as biperiden, monoamine oxidase inhibitors, cholinergic medicines, tricyclic antidepressants, selective serotonin reuptake inhibitors (SSRI), abrupt withdrawal of sedative-hypnotic medications, and abrupt alcohol withdrawal. Although RBDs are more prevalent in adults, recent research indicates that RBD may be the initial indication of pediatric narcolepsy. [52].

✓ **Nightmare**

Nightmares are often characterized by complex and frightening dream patterns that can lead to sudden awakening and vague recollection of the dream. Recurrent nightmares can be caused by stressful or traumatic situations, as well as the use of certain drugs like beta-blockers, levodopa, and acetylcholinesterase inhibitors. Abrupt withdrawal of REM suppression medications can also contribute to the occurrence of nightmares[52]

✓ **Recurrent Isolated Sleep Paralysis**

Recurrent isolated sleep paralysis is a benign illness that causes loss of voluntary muscle tone and incapacity to move upon awakening. Prolonged REM sleep muscle atonia upon awakening may be the fundamental mechanism responsible for sleep paralysis. Circadian rhythm abnormalities, such as shift work problems or jet lag, can cause sleep paralysis or sleep deprivation.

➢ **Other parasomnias**

There is no particular connection between other parasomnias and sleep stages.

They consist of exploding head syndrome, sleep-related hallucinations, sleep enuresis. These include sleeping-related hallucinations, exploding head syndrome, enuresis, parasomnia linked to medical conditions, parasomnia due to a medication , and unspecified parasomnia.

✓ **Exploding Head Syndrome**

"Exploding head syndrome" is a benign condition that involves a sudden, loud bang or explosive crashing sound in the head during wake-sleep transitions or while awakening in the middle of the night. This can be accompanied by light flashes or myoclonic jerks, and can be frightening for the person experiencing them. While patient reassurance is the main form of care, medications such as topiramate, nifedipine, and clomipramine may also be helpful.[52].

✓ **Catathrenia**

Catathrenia, also known as sleep-related groaning, is a condition characterized by recurrent episodes of groaning during sleep without any underlying otolaryngologic or vocal cord disorders. These episodes typically occur during the expiration phase of the REM sleep cycle and can interfere with the ability to sleep, leading to complaints from bed companions. Continuous positive airway pressure (CPAP) has been found to be an effective treatment

✓ **Sexsomnia**

Unusual sexual actions during sleep are the hallmarks of sexsomnia. Sexual activity, assault, masturbation, vocalizations, and affectionate touching between bed partners are a few examples of these occurrences. They occur during partial arousals in slow-wave sleep (stage three) and are classified as NREM parasomnia. Sexsomnia may be triggered by shift work and certain drugs, such as SSRIs. Parkinson's disease patients who have an impulse control problem may experience severe forms of these episodes. There is little information available on treating sleeplessness, but clonazepam has been demonstrated in certain studies to alleviate symptoms, and using CPAP to treat underlying obstructive sleep apnea should also be considered[52, 54].

- **Diagnosis**

The first step in evaluating parasomnias is having the patient and, if feasible, their bed companion provide a thorough history of their sleep. To identify the precise cause of the parasomnias, clinicians should additionally ask about the patient's underlying medical history, family history, history of substance misuse, and current medications taken. It is imperative to exclude further plausible differential diagnoses, including nocturnal seizures, psychiatric conditions such as post-traumatic stress disorder (PTSD), panic attacks, and psychogenic spells that may resemble parasomnias. Video electroencephalography (EEG) and polysomnography (PSG), often known as overnight sleep studies, are helpful in identifying parasomnias and identifying any underlying sleep disorders that may be causing sleep fragmentation and maybe parasomnia.

- **Management**

The initial step in managing parasomnias involves identifying and treating any comorbid sleep disorder or medical condition, as well as terminating inducing agents like benzodiazepine receptor agonists, antidepressants, and antipsychotics. Most childhood parasomnias are benign and can be outgrown without medical intervention. For adult parasomnias, it is crucial to educate patients and their bed partners about environmental safety methods, such as removing firearms, sharp objects, and furniture near the bed area. Locking windows and bedroom door alarms can be helpful for sleepwalkers. In patients with Restless Sleep Disorders (RBDs), environmental safety techniques are essential due to higher chances of injury to self or bed partners. Patients should use extra padding or pillows on the sides of the bed or padded armrests on the bedsides to prevent falls and injury. Bed partners should be made aware of the risk of injury and advised to sleep in a separate bed if violent behaviors occur. Psychotherapy

can be helpful in most NREM parasomnias, with benzodiazepines being the mainstay of management[52].

II.1.6.4. Circadian rhythm sleep-wake disorders[55]

- **Definition**:

Circadian rhythm sleep-wake disorders are Defined as persistent or recurrent sleep disturbances due to changes in the circadian system or imbalance between an individual's sleep-wake cycle and their surroundings.[9]. To diagnose a circadian rhythm sleep-wake disorder, certain general criteria must be met, including:

✓ A prolonged or frequent disruption of the sleep-wake rhythm resulting from a change in the endogenous circadian timing system and the necessary or desirable sleep-wake schedule

✓ The existence of a sleep-wake disorder, such as insomnia or extreme tiredness

✓ Associated discomfort or deficiency

Except for jet lag disorder, every disorder necessitates a minimum three-month symptom duration. In an ideal world, actigraphy and/or biomarkers, like dim-light melatonin onset, would be used to objectively document circadian timing. While they are not required, validated surveys, such as the morningness-eveningness questionnaire to determine chronotype, can be useful in diagnosing circadian rhythm sleep-wake problems**.**

- **Disorders of the circadian rhythm** include;

✓ **Delayed sleep phase disorder**

It is defined as a sleep-wake condition marked by a persistent delay in the onset of sleep and the time when people wake up. A typical patient has difficulty falling asleep at a conventional bedtime and awakens late in the morning.

Sleep is often normal once it is initiated. However, some patients will develop a comorbid chronic insomnia problem, which may result in difficulty initiating or maintaining sleep, even on a delayed schedule. This disorder is most prevalent in adolescents and younger adults

Puberty, stimulant use (e.g., caffeine), and irregular sleep patterns are risk factors.

In the majority of patients, clinical history supplemented by sleep logs and/or actigraphy is sufficient to make the diagnosis.

Treatment options include morning phototherapy, melatonin receptor agonist (such as ramelteon, taken at night), the use of chronotherapy[56–58]

✓ **Advanced sleep phase disorder**

Definition: A sleep-wake condition that causes earlier than expected sleep onset and awakening times.

Risk factor: linked to advanced age.

The diagnosis is confirmed by the demonstration of an advanced circadian rhythm on sleep log or actigraphy and the exclusion of alternative diagnoses such as depression or insomnia.

Treatment options include; Reassuring the patient and phototherapy in the evening [9, 56].

✓ **Jet lag disorder**

It is a circadian rhythm sleep disorder characterized by insomnia or hypersomnia resulting from travel across time zones. Risk factor includes sleep deprivation before travel. Treatment options include spontaneous resolution and exposure to sunlight in the new time zone can accelerate the recovery.

✓ **Shift-work sleep disorder**

A sleep-wake condition is defined by a misaligned circadian rhythm, which is often caused by working at night and experiencing sleep deprivation. The risk factors include working shifts that last longer than 16 hours or working during the night. Modafinil is prescribed in severe cases, and bright light treatment at night can help adjust to shift work.

✓ **Non-24-hour sleep-wake disorder**

It is a circadian rhythm sleep disorder in which an individual is unable to synchronize with the 24-hour cycle of their surroundings. Risk Factors include blindness and reduced sensitivity to light. Management options include a combination of scototherapy and phototherapy can help the patient's circadian clock resynchronize. Also tasimelteon is a melatonin receptor agonist is a treatment option.

✓ **Irregular sleep-wake rhythm disorder**

It is characterized by the absence of a clearly defined circadian rhythm for sleep and wakefulness. This problem is often associated with developmental abnormalities in children and adults who suffer from neurodegenerative diseases such as Alzheimer's, Parkinson's, and Huntington's disease.

II.1.6.5. Sleep-related breathing disorders [59–61]

- **Obstructive sleep apnea**

- **Definitions**[59–61]:

✓ **Obstructive sleep apnea** (OSA) is a disorder that is characterized by obstructive apneas, hypopneas, and/or respiratory effort-related arousals caused by repetitive collapse of the upper airway during sleep.

✓ **Sleep Apnea Syndrome (**SAS) is a disorder characterized by frequent episodes of apnea and hypopnea associated with symptoms of excessive daytime sleepiness and cardiovascular mortality and morbidity. It englobes a broad spectrum of sleep-disordered breathing from central to mixed, OSA and hypopnea

✓ **Apnea:** complete or nearly complete (≥ 90%) cessation of inspiratory airflow for ≥ 10 seconds.

✓ **Hypopnea:** airflow reduction by ≥ 30% of the pre-event baseline for ≥ 10 seconds in combination with either desaturation by ≥ 3% or arousal from sleep

✓ **Respiratory effort-related arousal:** arousal from sleep due to increased respiratory effort or reduced airflow for ≥ 10 seconds without significant hypopnea or apnea.

- **Epidemiology**

Global estimates using five or more events per hour suggest rates of 936 million people worldwide with mild to severe OSA and 425 million people worldwide with moderate to severe OSA between the ages of 30 and 69 years of age [62] . In sub-Saharan Africa, precisely Cameroon, a study done in an adult population found a prevalence of OSA at 28.5% [63], aside from this population-based study, hospital-based or clinical studies showed higher rates, with one of such studies showing a prevalence of 57.7% of OSQ in a tertiary referral hospital in Cameroon [64].

- **Etiology and risk factors**

OSA is caused by Obstruction of the upper airways due to the collapse of the pharyngeal muscles during sleep. Risk factors for obstructive sleep apnea include[60, 61] ;

✓ Obesity, especially around the neck (short, wide “bull neck”)

✓ Structural abnormalities that impair respiratory flow, including:

- ✓ Adenotonsillar hyperplasia (especially in children); nasal septum deviation; previous upper airway surgery; enlarged uvula, tongue, or soft palate (especially in adults); overbite with a small chin; hypertrophied pharyngeal muscles; nasal polyps
- ✓ Alcohol consumption before sleep
- ✓ Intake of sedatives and/or beta-blockers before sleep
- ✓ Smoking
- ✓ Family history
- ✓ Acromegaly
- ✓ An increased neck circumference (> 40 cm) is a very important risk factor.

- **Pathophysiology**

When the upper airways are obstructed, it can lead to apnea, which in turn can cause a decrease in the amount of oxygen in the arterial blood (PaO2) and an increase in the amount of carbon dioxide (PaCO2), also known as hypercapnia. This can cause several negative effects, including increased hypoxic pulmonary vasoconstriction, which can lead to pulmonary hypertension and cor-pulmonale. It can also cause an increase in sympathetic activity, which can result in secondary hypertension. Additionally, respiratory acidosis can occur, which triggers renal compensation. This leads to an increase in HCO3 retention and a decrease in chloride reabsorption [65].

- **Clinical features**
  - ✓ Restless sleep with waking, gasping, or choking
  - ✓ Loud, irregular snoring with apneic episodes (third-party reports)
  - ✓ Excessive daytime sleepiness (e.g., patient falls asleep, microsleep while seated)
  - ✓ Morning headaches
  - ✓ Signs of complications, including Impaired cognitive function (e.g., impaired concentration, memory loss) [60]; Depression [66]; Decreased libido; Hypertension with increased pulse pressure; Obstructive sleep apnea is one of the most common causes of secondary hypertension[67]

- **Diagnosis**

Diagnosis is made with the help of a;

- ✓ Detailed sleep history, including third-party reports (e.g., interviewing the sleep partner about snoring and witnessing apneas).

Also, evaluation for comorbidities, including complications of OSA and risk factors for OSA.

✓ Screening options are available to assess the risk of OSA; they include the STOP-BANG questionnaire and the Berlin Questionnaire**.**

✓ To confirm diagnosis overnight, polysomnography is the gold standard; it is an in-laboratory test that measures the frequency of obstructed breathing events — apneas and hypopneas — during sleep. Collectively, the number of apneas and hypopneas per hour of sleep is termed the apnea-hypopnea index (AHI), in which the presence of obstructive sleep apnea is defined as an AHI of 5 or more events per hour. The AHI is used to categorize disease severity; persons with an AHI of 5 to 15, 16 to 30, or more than 30 events per hour are considered to have mild, moderate, or severe obstructive sleep apnea, respectively.

✓ There are other diagnostic options, which include portable monitoring devices, such as the Home-Sleep apnea test, which is a portable device worn at night that monitors your breathing and oxygen levels to detect and measure pauses in breathing, which are known as apneas. The test calculates an OSA severity score by calculating the average number of lapses in breathing per hour in bed. Most at-home tests do not measure sleep quality. Comparatively, polysomnography offers a more holistic overview of your sleep quality and sleep patterns in addition to the apnea-related metrics measured by at-home tests.

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- **Treatment** [61]

Treatment includes

✓ Lifestyle modifications like weight loss, sleep hygiene measures, avoiding alcohol, sedatives like benzodiazepines, nicotine

✓ Supportive measures like treating associated conditions and complications of OSA, such as high blood pressure and concomitant nasal obstruction with nasal steroids

for allergic rhinitis, and surgery in case of nasal valve collapse. Other surgeries include uvulopalatopharyngoplasty, which entails surgically removing the uvula and tissue from the soft palate to create more space in the oropharynx.

- ✓ **Positive airway pressure therapy** is the gold standard treatment for adults.

Other low-cost and portable devices like the

- **Oral appliances:** They are used by patients who cannot tolerate or unwilling to use CPAP i.e., continuous positive airway pressure. These are appliances which are being worn during sleep to keep mandibular advancement and prevent airway collapse.
- **Position therapy**: This entails using devices to keep patients in a lateral rather than the usual supine position.[68]

II.2. STROKE

II.2.1. Introduction

According to The American Heart Association/The American Stroke Association (AHA/ASA), in the simplest form, Stroke is defined as an episode of acute neurological dysfunction presumed to be caused by ischemia or hemorrhage, persisting ≥24 hours or until death. Despite this definition, the AHA/ASA provides a comprehensive definition of stroke, taking into consideration advanced information on clinical and imaging elements

Stroke ranks as the second leading cause of death worldwide and is a major contributor to disability [2]. Stroke imposes a considerable financial burden due to the costs associated with prehospital, hospital, and posthospital care [20]. Studies reveal that 85% of strokes are ischemic, primarily caused by cerebral small vessel disease, cardioembolism, and large artery atherosclerosis-related thromboembolism, while 15% are intracerebral hemorrhage and 80% result from cerebral small vessel diseases. Notably, strokes are distinguished from transient ischemic attacks, which are defined as a brief period of neurological impairment without acute infarction or tissue damage brought on by focal brain, spinal cord, or ocular ischemia [69]. Symptoms usually last less than 24 hours.

Stroke is one of the most frequent pathologies encountered in most health facilities worldwide and in Cameroon in particular [70]. Hence, a proper understanding of its epidemiology, etiologies, clinical aspects, management, and proper rehabilitation measures are necessary in order to significantly reduce the burden of stroke.

II.2.2. Epidemiology

As per the latest estimates from the Global Burden of Disease (GBD), the number of incident cases of stroke was approximately 12.2 million in 2019, with 143 million DALYs lost and 6.6 million deaths worldwide. Thus, making stroke the third most common cause of disability and the second leading cause of death globally.

In Africa, studies within the past decade have revealed a yearly incidence of stroke cases of up to 316 per 100,000, a prevalence of up to 1,460 per 100,000 and a 3-year fatality rate greater than 80%. Also, an approximate value of 70% of deaths from stroke and 87% of stroke-related disability have been recorded in low-income and middle-income countries. Recent studies witness an exponential increase in the burden of stroke in Africa, with several factors being attributed to this increase. They include utero and early-life undernutrition, which are associated with increased cardiometabolic risk factors in mid-life, increasing exposure to indoor and outdoor particulate air pollution, changes in dietary habits, and population aging [3, 71].

In Cameroon, a prevalence rate of stroke of 7.3% and a mortality rate of 26.7% during the first month and 31.7% in the first three months after the stroke event has been recorded[4]. A study conducted on stroke patients in Douala, Cameroon, revealed that the male sex made up 61.8% of their study population. It equally enumerated hypertension as the most prevalent risk factor and a case fatality rate of 26.8% [70].

II.2.3. Pathophysiology of stroke[72]

- **Ischemic stroke**

An inadequate blood supply to a specific region of brain tissue is the first step in the pathogenesis of ischemic stroke. The area of infarction, or center core of damaged tissue, advances toward irreparable destruction in a matter of minutes. However, the surrounding tissue called the penumbra, does not suffer from instantaneous cell death and may recover if early reperfusion is accomplished.

Reduced blood flow regions have an imbalance between adenosine triphosphate (ATP) generation and consumption, which leads to a decrease in ATP storage. Ionic imbalances, electrical disruptions, and a series of ischemia-related alterations result from this. Nitric oxide and reactive oxygen species are produced at higher rates as a result of these modifications.

Through processes like necrosis or apoptosis, the pathophysiological cascade gradually ruins cell membranes, causes cell lysis, and causes cell death.

Microglia are quickly activated in the ischemic area after an ischemic stroke and spread to the penumbra area. Their activation can last for several weeks and peaks 48 to 72 hours after the stroke occurs. Proinflammatory cytokines such as reactive oxygen species, nitric oxide, interleukin-1β, and tumor necrosis factor-α are increased when activated microglia are present. On the other hand, they also release neurotrophic factors such as basic fibroblast growth factor, glial cell line-derived neurotrophic factor, and brain-derived neurotrophic factor, as well as anti-inflammatory cytokines.

- **Intracerebral hemorrhage**

Following tiny artery rupture from hypertension alterations, coagulopathies, other vasculopathies, and growing hematoma mass effect, perihematomal edema, and other harm mechanisms are the main causes of ICH. Increased intracranial pressure (ICP) is caused by the expanding hematoma volume and edema, which may lessen cerebral perfusion and ischemic damage. Furthermore, patients run the risk of developing a hernia and intraventricular hemorrhage (IVH).

Like ischemic stroke, ICH swings through pro- and anti-inflammatory stages afterward. The disruption of the blood-brain barrier by secondary mechanisms of damage, such as blood-related cytotoxicity, excitotoxicity, and oxidative stress, leads to a substantial loss of brain cells and the formation of potentially fatal cerebral edema**.**

- **Subarachnoid hemorrhage**

SAH is mostly caused by a ruptured cerebral aneurysm. It's important to remember, though, that cerebral aneurysms can cause brain damage even in the absence of a rupture. The aneurysm's compressive stresses have the potential to harm nearby brain tissue and impair the blood flow to distant regions.

Aneurysm ruptures cause arterial blood to burst into the subarachnoid space, where it quickly permeates the CSF and raises intracranial pressure. Additionally, the cerebral parenchyma and intraventricular area may be reached by arterial blood.

A number of conditions, including delayed cerebral ischemia, hydrocephalus, ICH, IVH, and ICP, can result in secondary brain injury.

II.2.4. Recall on the vascularization of the central nervous system[25]

II.2.4.1. Arterial system

The brain receives its blood supply from the internal carotid arteries (ICA) on both sides, which is known as the anterior circulation. On the other hand, the vertebral-basilar arterial system, originating from branches of the aortic arch, makes up the posterior circulation. The anterior and posterior circulatory systems combine at the central cranial base to form a circular anastomotic ring referred to as the "circle or polygon of Willis." The sides of the polygon of Willis comprise the anterior cerebral arteries (ACA), the posterior cerebral arteries (PCA), and the anterior communicating branch, which bridges both ACA and the posterior communicating arteries, which link ICA and PCA on each hemibrain.

Each Internal Carotid Artery (ICA) branches into 3: Anterior Cerebral Artery (ACA), Middle Cerebral Artery (MCA), and Anterior Choroidal Artery (AChA). Posterior Cerebral Arteries (PCAs) are terminal branches of the Vertebrobasilar Artery.

Each of these branches is further subdivided:

- ACA: (A1-Horizontal or pre-communicating starts at the carotid bifurcation and ends at the level of ACom A; A2- Vertical or post-communicating segment or pre-callosal begins at the anterior communicant artery and ends at the junction rostrum-genu of the corpus callosum; A3- pre-callosal; A4- Supra-callosal; A5 - Postero-callosal

(A3, together with A4 and A5, are collectively called peri-callosal artery).

- MCA is subdivided into: M1- Sphenoidal; M2 – Insular; M3 – Opercular; M4 – Cortical.
- PCA is subdivided into: P1- Pre-communicating; P2- post-communicating;

P3-Quadrigeminal; P4 – Calcarine.

ACA, MCA, and PCA contain perforating branches that supply vital structures like the pituitary gland, infundibular stalk, optic chiasm, hypothalamus, thalamus, midbrain, and basal ganglia.

Each of the brain territories is supplied by a respective artery. Listed below are the main cerebral arteries, their perforating branches, and related supplied territory.

➢ **Anterior Cerebral Artery, its Branches**

- The recurrent artery (of Heubner) is the largest artery arising from A1 or proximal A2. This artery goes through the anterior perforated substance and then moves upward and laterally to the optic chiasm. It supplies the anterior section of the putamen, the anterior head of the caudate nucleus, the uncinate fasciculus, the anteroinferior region of the anterior limb of the internal capsule, and, in rare cases, the anterior hypothalamus.
- The basal perforating arteries, which are also known as medial lenticulostriate arteries, arise from the anterior cerebral and anterior communicant arteries. These arteries supply various parts of the brain including the anterior perforated substance, the dorsal surface of the optic nerve and optic chiasm, the optic tract, the suprachiasmatic portion of the hypothalamus, the rostrum of the corpus callosum, the lower surface of the frontal lobe, and the medial part of the Sylvian fissure.
- The Peri-callosal artery (made up of pre-callosal, supra-callosal, and postero-callosal branches of the ACA) is the distal part of the artery surrounding the corpus callosum.

It gives rise to its largest branch: the callosomarginal artery. The callosomarginal artery is easily identifiable as it courses in or near the cingulate sulcus.

Both callosomarginal and distal peri-callosal arteries give 5 main cortical branches. They are orbitofrontal, frontopolar, internal frontal, paracentral and parietal arteries. The cortical branches vascularize the superior frontal gyrus, the anterior two-thirds of the medial hemisphere (together with the pre-central, central, and post-central gyri), and the basal surface of the frontal lobe. Some distal ACA may supply part of the contralateral hemisphere.

➢ **Anterior Choroidal Artery**

Just above the origin of the posterior communicant artery, on the posteromedial side of the supra-clinoid internal carotid artery, there is a small but relatively constant channel called the anterior choroidal artery (AChA). The AChA can be found either as a plexus of small blood vessels or as a single trunk. The AChA region may vary but usually includes the optic tract, the posterior limb of the internal capsule, the cerebral peduncle, the choroid plexus, and the medial temporal lobe.

➢ **Middle Cerebral Artery**

- The M1-Sphenoidal proximal segment travels laterally to the optic chiasm, where it meets the Sylvian fissure's medial entrance medially. Pre and post bifurcation are the two sections it describes along the way. Mainly bifurcating, but occasionally trifurcating, is this trunk.
- The lateral lenticulo-striate arteries, which are perforating branches of M1, supply the majority of the basal ganglia, internal capsule, and caudate nucleus. The temporal lobe's anterior pole is supplied via its cortical branches.
- The insular segment (M2) consists of 6 to 8 main arteries which lie over the insula and terminate on top of the circular sulcus.
- The distal segments M3 (Opercula) and M4 (Cortical) extend from the lateral cerebral fissure's surface to the majority of the brain's lateral surface. The orbitofrontal, parietal, angular, temporal, pre-frontal, pre and postcentral sulcus and temporal-occipital arteries are its cortical branches. Nearly all of the brain's lateral surface is covered by the M3 and M4 territories.
- Watershed areas occur on the cortical surface that lead to vulnerable brain areas during hypotensive episodes, despite the development of anastomoses between the ACA, MCA, and PCA arterial domains

➢ **Posterior Cerebral Artery**

The proximal segment (P1 - pre-communicating artery) of the posterior cerebral artery (PCA) has numerous significant perforating branches that nourish the brainstem, thalamus, oculomotor, and trochlear nuclei. This segment runs from the basilar bifurcation to the junction with the posterior communicant artery.

P2(post-communicating artery) travels between the junction and the posterior aspect of the midbrain where it gives thalamoperforating, thalamogeniculate, peduncular perforating, posterior choroidal and posterior temporal arteries.

The distal part of PCA (P3 and P4) courses from the quadrigeminal plate to the calcarine fissure where they supply the occipital lobe, part of the parietal and temporal lobe and the posterior third of the medial brain hemisphere.

➢ **Vertebrobasilar System**

The vertebral artery's intradural portion courses anteromedially through the foramen magnum and runs superomedially toward the midline, where the two vertebral arteries unite to form the basilar artery. On its way to the basilar artery, the vertebral artery gives the posteroinferior cerebellar artery, the anterior spinal artery (which supplies the upper spinal cord, lateral medulla, tonsils, the vermis), and the inferior cerebellar hemisphere. From the confluence of the vertebral arteries, the Basilar courses superiorly in front of the medulla, pons, and bifurcate at the junction pons/mesencephalon where it gives the posterior cerebral arteries. Along its path, BA gives the anterior inferior cerebellar arteries, pontine perforating branches, and the superior cerebellar arteries, supplying the brainstem, and midbrain.

II.2.4.2. Venous system of the brain [73]

➢ **Superficial cerebral veins**

Superficial veins are veins that drain the white matter and are linked to a bridging vein, which connects the superficial vein to the dural venous sinuses.

- **Superior cerebral veins:** It has as bridging vein, the Superior anastomotic vein and it drains into the Superior sagittal sinus
- **Middle cerebral veins**: which have as bridging vein, Inferior anastomotic vein, which drains in to cavernous sinus
- **Inferior cerebral veins** which drain into Cavernous and transverse venous sinuses.
- **Deep cerebral veins:** Deep cerebral veins drain the cerebral medulla and drain into the straight sinus.
- Medullary veins: They drain the gray matter.
- Subependymal veins: They receive blood from the medullary veins.
- Basal vein (vein of Rosenthal): They are paired paramedian veins that receive blood from the temporal lobe and drain into the great cerebral vein.
- Great cerebral vein (vein of Galen): It receives blood from the deep veins.

➢ **Dural venous sinuses**[73]

The dural venous sinuses drain blood from cerebral veins and CSF from the arachnoid granulations into the internal jugular vein. The sinuses are located intracranially between the two layers of the dura mater (endosteal layer and meningeal layer). They include

- **Superior sagittal sinus** which is located at the midline and terminates at the confluence of sinuses and becomes the right transverse sinus. It has the function of draining blood from the cortical veins of the cerebral hemispheres. The main location of cerebral fluid return via arachnoid granulations.
- **Inferior sagittal sinus** is located at the midline and is joined by the great cerebral vein of Galen before draining into the straight sinus. It drains blood from the medial surface of the cerebral hemispheres.
- **Straight sinus** is located at the midline and terminates at the confluence of sinuses. Variations in structure may happen as it may unite with the left transverse sinus.
- **Occipital sinus** is located posteriorly and it drains into the confluence of sinuses.
- **Confluence of sinuses** is located posteriorly. It is formed by the union of the superior sagittal sinus, straight sinus, and occipital sinus. It drains into the left and right transverse sinus.
- **Superior petrosal sinus**(paired): It is located laterally it drains blood from the inner ear structures via the labyrinthine vein into the transverse sinus.
- **Transverse sinus** is paired and is located laterally along the edge of the tentorium cerebelli. It drains into the sigmoid sinus.
- **Inferior petrosal sinus**(paired): It is located laterally. As concerning its drainage, it drains the cavernous sinus into the internal jugular vein. It equally drains blood from the medulla, pons, and inferior surface of the cerebellum.
- **Sigmoid sinus (paired):** It is Laterally located. It is the continuation of the transverse sinus that arches downward in an S-shaped groove into the internal jugular vein.
- **Sphenoparietal sinus** (paired): It is anteriorly located and it drains into the cavernous sinus.
- **Cavernous sinus** (paired): It is located anteriorly on each side of the Sella turcica (pituitary fossa). Structures running through these sinuses include:

- Medially: internal carotid artery with postganglionic sympathetic fibers and abducens nerve (CN VI)
- Laterally: oculomotor nerve (CN III), trochlear nerve (CN IV), ophthalmic nerve (CN V1), and maxillary nerve (CN V2)

It receives blood from the superior ophthalmic vein (blood from the eye and superficial cortex), drains into the petrosal sinuses, and then enters the internal jugular vein.

- **Basilar venous plexus** (paired): It is located over the basilar part of the occipital bone (the clivus). It is connected with the cavernous and petrosal sinuses and the internal vertebral (epidural) venous plexus.

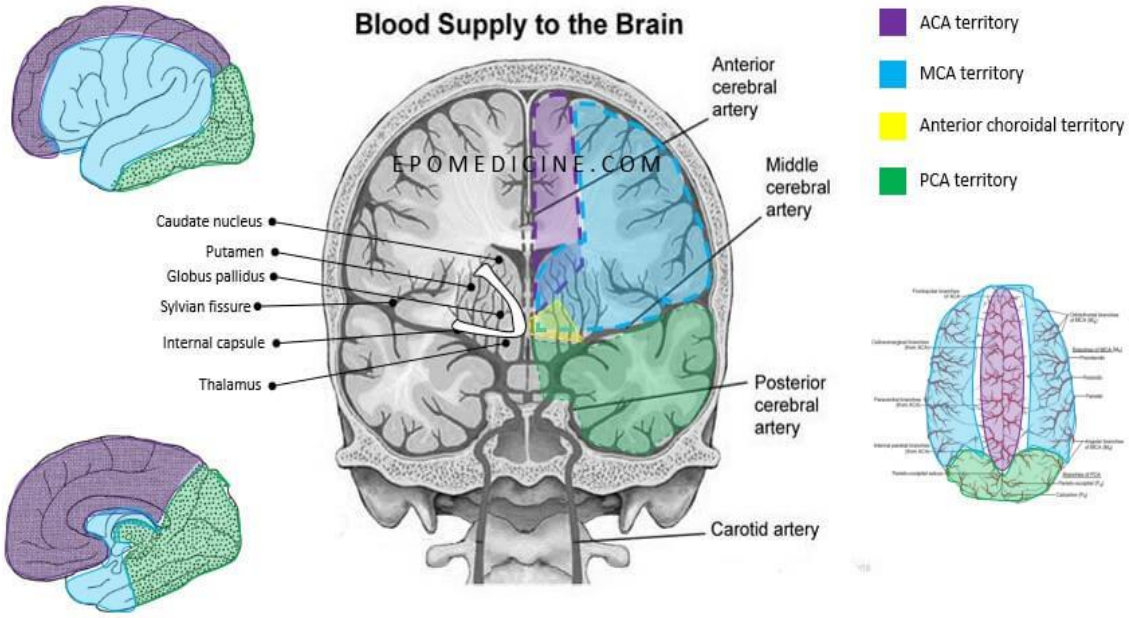


Figure 4: Blood supply to the brain [25]

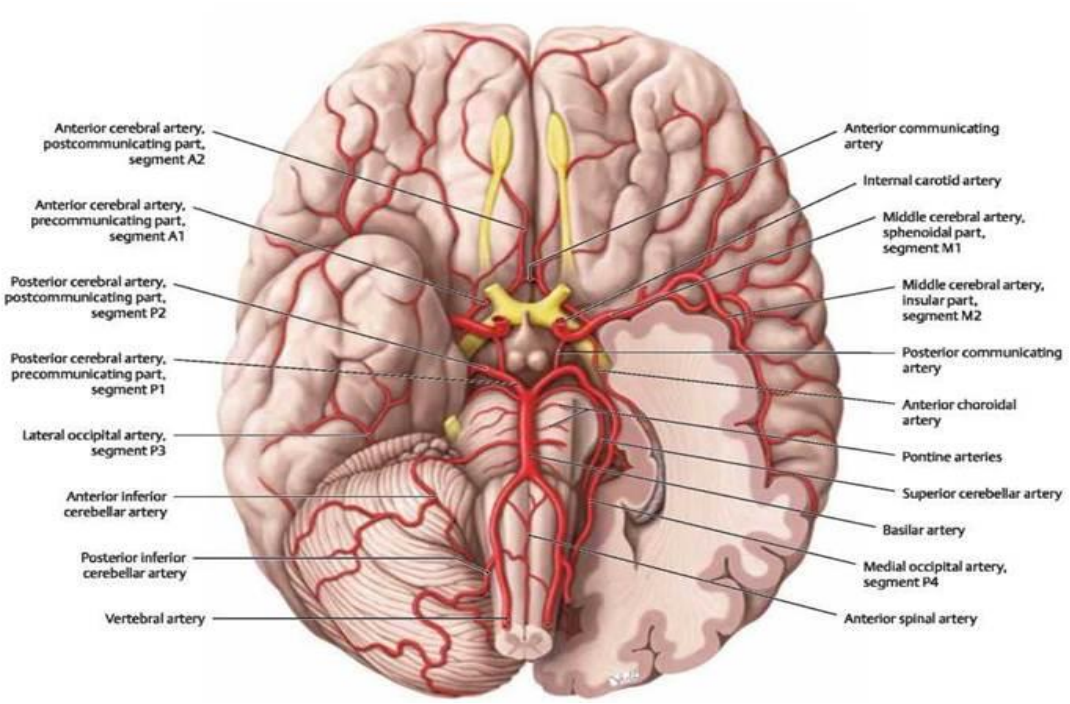


Figure 5: Blood Vessels of the Brain - Atlas of Anatomy. Head and Neuroanatomy. Michael Schuenke[74].

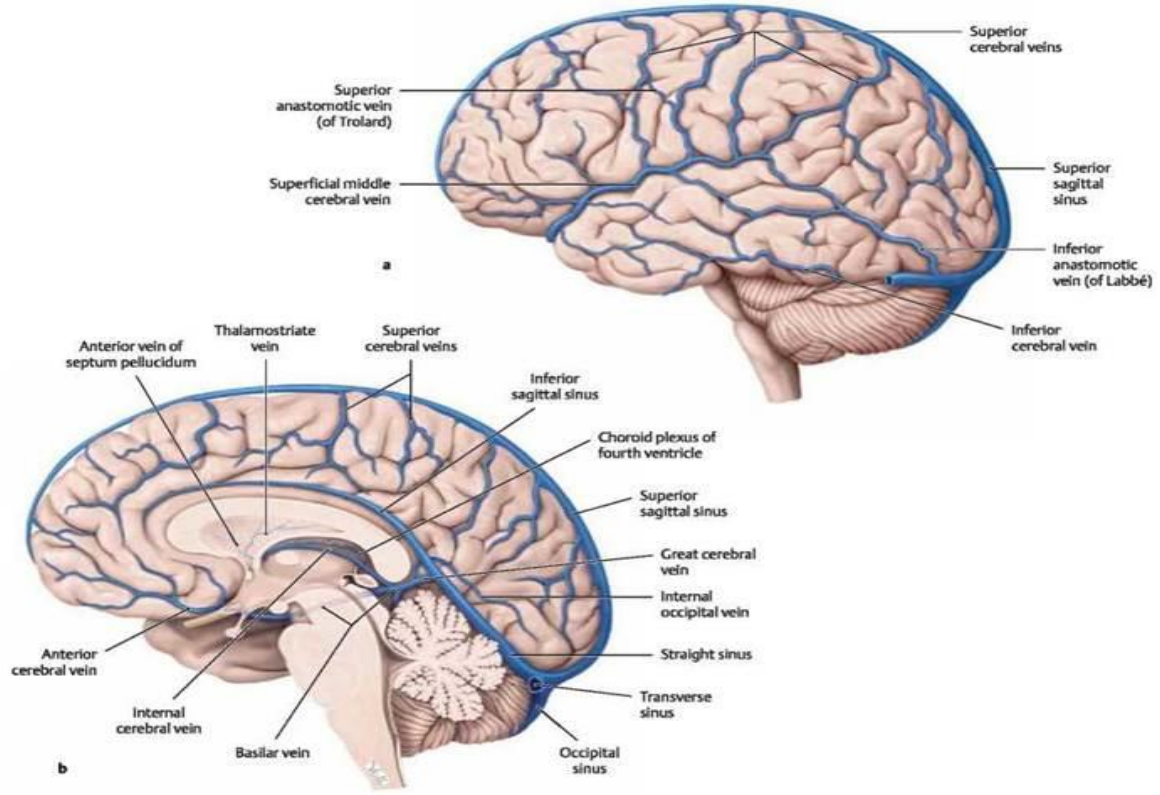


Figure 6: Superficial and deep veins of the brain - Atlas of Anatomy. Head and Neuroanatomy. Michael Schuenke [74]

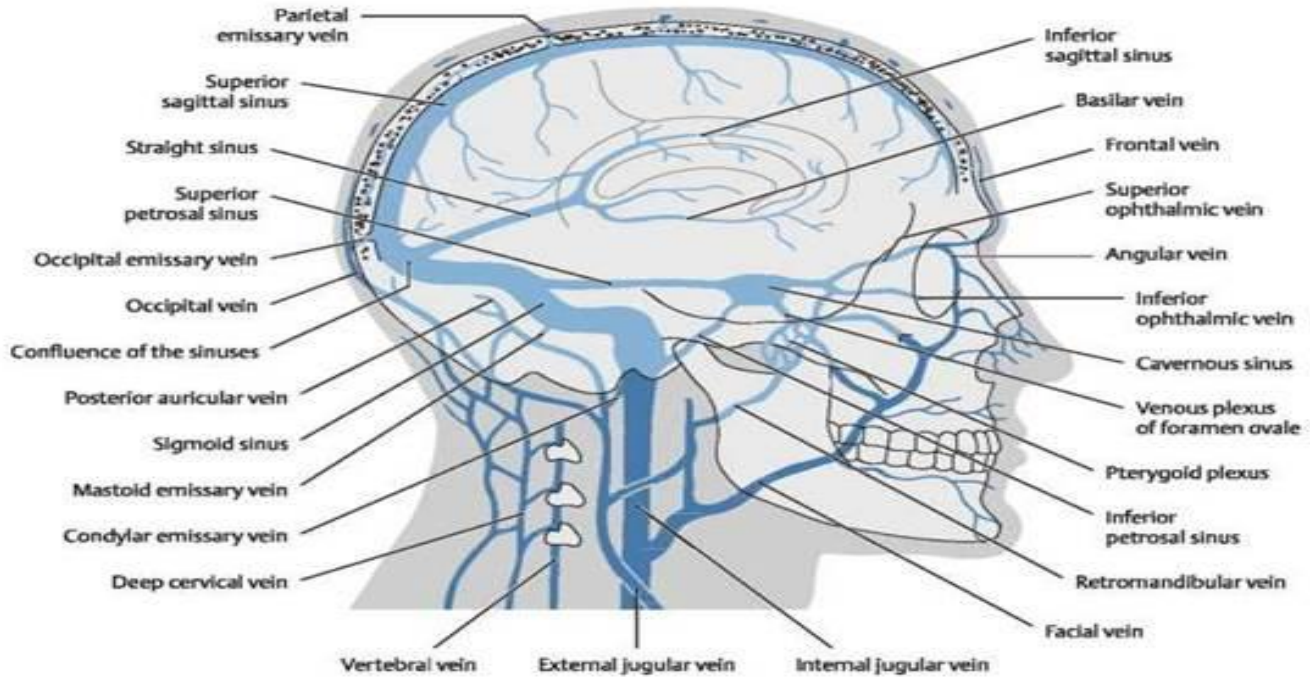


Figure 7: Superficial and deep veins of the brain - Atlas of Anatomy. Head and Neuroanatomy. Michael Schuenke[74]

II.2.5. Risk factors and etiologies of stroke [1, 72]

➢ **<u>Risk factors</u>**

Certain individuals are more susceptible to stroke than others. By being aware of the risk factors and taking action against them, a stroke may be avoided. Stroke risk factors can be classified as either non-modifiable or modifiable.

- ✓ Non-modifiable risk factors—those that cannot be altered or controlled—include age, gender, race or ethnicity, and family history.
- ✓ Modifiable risk factors and medical conditions, like high blood pressure, high cholesterol, and smoking, can be controlled with medical care and lifestyle modifications.

➢ **Unmodifiable risk factors**

- **Age**: People of all ages are susceptible to strokes, including young adults, kids, newborns, and unborn children. However, the risk of stroke rises with age, with the incidence doubling every decade after the age of 55 years.
- **Sex:** As a person gets older, their risk of developing a stroke increases. Gender also plays a role in the risk of stroke. Generally, males are at a higher risk of stroke in their younger

and middle ages, as shown by a study conducted in Douala [70], Cameroon. However, this risk evens out as they get older. Although fewer females have strokes than males, they are more likely to die from them. This may be because females are generally older when they experience a stroke.

- **Family history**: An individual's risk of stroke increases if they have a parent, grandparent, or sibling who has experienced a stroke. In some families, there may be a genetic component to the risk of stroke. While lower genetic contributions sometimes come from family members who may have a genetic or inherited inclination for stroke risk factors, such as high blood pressure, diabetes, or heart disease, some genetic abnormalities carry a higher risk of stroke. It is also plausible that modifiable behavioral characteristics within a family account for an elevated risk of stroke.
- **Race:** There are variations in the risk of stroke among different racial and ethnic groups. Black and Hispanic Americans have nearly double the rate of stroke in comparison to White people. Strokes usually happen at a younger age in Black and Hispanic Americans. Additionally, Black people have a higher stroke fatality rate compared to other populations.
- **Prior stroke or heart attack**: A person is more likely to experience another stroke or heart attack after one. People who have experienced a heart attack are also more likely to have a stroke [75].

➢ **Modifiable risk factors**[72]

- **High blood pressure** Hypertension, also known as high blood pressure, is the leading risk factor for stroke. People with high blood pressure are two to four times more likely to have a stroke before the age of 80 compared to those without high blood pressure. Hypertension can cause damage to blood vessels and promote atherosclerosis, which is the main cause of blood vessel narrowing. This narrowing can lead to both heart attack and stroke. Hence, it is essential to diagnose and control hypertension to prevent strokes from occurring. A good number of studies in Cameroon have shown hypertension to be the most prevalent modifiable risk factor in stroke patients [75, 76]
- **Hyperlipidemia**: High cholesterol, a vital body product, can contribute to stroke risk. It is primarily produced by the liver and is essential for hormone production and cell membranes. Excessive LDL can build up in blood vessels, leading to stenosis and atherosclerosis. A person's LDL level should be less than 130 mg/dL. High HDL levels can prevent stroke. Genetics may also play a role in high cholesterol levels. A healthy diet and

regular exercise can help lower cholesterol levels, and statin drugs can significantly reduce stroke risk in high-cholesterol individuals

- **Atrial fibrillation**: Cardioembolic infarction, primarily caused by atrial fibrillation, is the most severe ischemic stroke subtype, causing high disability and mortality. Atrial Fibrillation increases with age, causing 20-25% of strokes in patients over 80. Anticoagulation is effective in preventing stroke in Atrial Fibrillation patients, with a two-thirds reduction in risk. Atrial fibrillation causes blood stagnation in the atria, leading to blood clots that can cause an ischemic stroke. Atrial Fibrillation affects over 9 percent of people over 65.
- **Diabetes:** Diabetes increases stroke risk due to impaired glucose transport from blood to cells. hence, glucose accumulates in the blood vessels, where it leads to damage to both the blood vessels and tissue. High blood pressure is common among diabetics, contributing to stroke risk. Obesity is the greatest modifiable risk factor for diabetes. Controlling blood pressure, diabetes, dietary changes, and weight loss can lower stroke risk. High blood glucose levels during a stroke can cause more severe brain damage. However, controlling blood sugar does reduce the risk of recurrent stroke.
- **Smoking:** An individual's risk of having an ischemic stroke is nearly doubled by smoking alone, even in the absence of additional risk factors. Smoking increases blood clotting factors, aneurysm development, and atherosclerosis. Two years after quitting smoking, the risk of stroke significantly reduces; five years later, the risk drops even more to the level of nonsmokers. Individuals with cerebral aneurysms who smoke also have a higher chance of rupture and subarachnoid hemorrhage.
- **Obesity**: Being overweight is a medical condition that is closely associated with three additional stroke risk factors: high blood pressure, diabetes, and heart disease. Therefore, it is undoubtedly considered a stroke risk factor. Moderate exercise and weight loss have been found to be effective in reducing high blood pressure and improving heart health. However, no studies have yet been conducted to examine their effects on the risk of stroke.
- **Alcohol and drug abuse**: There is a curvilinear pattern in the link between the risk of stroke and alcohol intake, with the risk increasing with daily alcohol consumption. Alcohol consumption that is low to moderate ($\leq$2 standard drinks per day for males and $\leq$1 for women) lowers the risk of stroke, while high intake raises it. On the other hand, even moderate alcohol use increases the risk of hemorrhagic stroke. Frequent use of illegal drugs, such as amphetamines, cannabis/marijuana, phencyclidine, cocaine, heroin, lysergic acid diethylamide, or cocaine, is linked to an elevated risk of strokes of all subtypes.

II.2.6. Etiologies of stroke[1]

Stroke can be caused either by ischemia resulting from blockage of a blood vessel (ischemia), or a rupture of a blood vessel (hemorrhagic stroke). Ischemic stroke accounts for 87% of all strokes [72].

- **Ischemic stroke**

The Trial of Org 10172 in Acute Stroke Treatment (TOAST) classification system identifies 3 primary causes of ischemic stroke: large vessel disease, small vessel disease (lacunar), and cardioembolism.

Large vessel disease refers to various conditions that affect major arteries, such as atherosclerosis, arterial dissection, and artery-to-artery embolism. These conditions can cause specific syndromes due to the reduced blood flow to certain brain regions, which is associated with corresponding examination findings. Large vessels are composed of intracranial arteries (including the circle of Willis and its proximal branches) and extracranial arteries (including common carotid, internal carotid, and vertebral arteries).

Cardioembolism, as an etiology of stroke (ischemic) can be caused by arrhythmia, valvular heart disease, bioprosthetic and mechanical heart valves, and cardiomyopathy.

Lacunar strokes are a type of stroke that is often caused by small vessel diseases. The main culprits behind these strokes are lipohyalinosis and atherosclerosis. Lipohyalinosis occurs when the small cerebral vessels thicken and narrow due to the buildup of a substance called hyaline. This can lead to blockages in the penetrating arteries, which can cause a stroke. Atherosclerotic plaques in the parent arteries, especially those near the perforating branches, can also cause blockages. Additionally, microatheromas can obstruct small penetrating arteries, leading to lacunar stroke.

Ischemic stroke is mainly associated with several important risk factors, including advanced age, hypertension, diabetes, hyperlipidemia, cigarette smoking, arrhythmia, and cardiac disease.

Intracerebral Hemorrhage (ICH)

The second most prevalent type of stroke is intracerebral hemorrhage (ICH). A number of diseases, including coagulopathies, cerebral amyloid angiopathy hypertensive vasculopathy,

and other vasculopathies, can produce tiny artery rupture (ICH). While lobar ICH is more frequently related with cerebral amyloid angiopathy, hypertensive vasculopathy is mostly linked to non-lobar ICH, which can develop in areas such the brainstem, thalamus, cerebellum, and basal ganglia. ICH is associated with a number of risk factors, such as advanced age, hypertension, cerebral amyloid angiopathy, smoking, heavy alcohol use, sympathomimetic medications, anticoagulants, and antiplatelet medications.

- **Subarachnoid Hemorrhage (SAH)**

Approximately 5% of all strokes are caused by spontaneous subarachnoid hemorrhage (SAH) due to a ruptured aneurysm, which accounts for 85% of SAH cases. Other causes of spontaneous SAH may include drug use (such as amphetamines and cocaine), coagulopathy, a ruptured arteriovenous malformation, and vessel rupture due to a dural venous sinus thrombosis. There are several risk factors associated with SAH, including smoking, hypertension, excessive alcohol consumption, advancing age, personal history of another type of aneurysm or SAH, and family history of an intracranial aneurysm.

II.2.7. Clinical features

Clinical features [1, 77, 78]

The diagnosis is placed with the help of a thorough history, and a physical exam.

The history is very important as it helps to eliminate ischemic stroke mimics like hypoglycemia, seizures, and migraines.

In the history, information about when they were last known to be well, time of onset of symptoms, any risk factors, medications, and other relevant details regarding a possible underlying illness needs to be obtained,

Thereafter a rapid assessing vital signs, conduction of a targeted neurological examination using the National Institutes of Health Stroke Scale (NIHSS) is recommended. The National Institutes of Health Stroke Scale (NIHSS) is most commonly used to measure the severity of the stroke and has 11 categories and a score that ranges from 0 to 42. The 11 categories include the level of consciousness, which incorporates level of consciousness questions evaluating best gaze, visual, facial palsy, motor arm, motor leg, limb ataxia, sensory, best language, dysarthria, and extinction and inattention. The stroke scale should be performed in the order listed. Each score is based on the patient's action on the exam, and it is not a prediction of what the patient can do.

As concerns ischemic strokes, certain findings on physical exams help the physician to predict the territory of the brain vasculature that could be affected. Symptoms manifest as follows with respect to the vessel injured;

- **Anterior cerebral artery**
  - ✓ Leg more than arm involvement with hand sparing
  - ✓ Urinary incontinence
  - ✓ Gait apraxia
  - ✓ Akinetic mutism
- **Middle cerebral artery**
  - ✓ Homonymous hemianopia/quadrantanopia (involvement of inferior division)
  - ✓ Face–arm–leg involvement
  - ✓ Aphasia (Broca's = superior division; Wernicke's = inferior division)
  - ✓ Inattention
  - ✓ Gaze paralysis (usually indicates a large area of frontal damage)
- **Vertebrobasilar**
  - ✓ Occipital lobe: homonymous hemianopia, cortical blindness, other cortical visual deficits
  - ✓ Cerebellum: ataxia, nystagmus
  - ✓ Brainstem cranial nerve palsies: diplopia, facial numbness/weakness, vertigo, dysphagia, dysphonia
  - ✓ Spinal tracts: hemiparesis and hemisensory loss
- **Lacunar stroke syndromes** (due to occlusion of deep perforating small arteries)
  - ✓ Pure motor hemiparesis
  - ✓ Pure sensory stroke
  - ✓ Sensorimotor stroke
  - ✓ Ataxic hemiparesis.

- The clinical presentation of **hemorrhagic stroke** is not very different from that of ischemic stroke, but certain studies have found some symptoms to be more attributed to hemorrhagic stroke and vary by size and location of the hematoma.
  - ✓ Headache, which is more frequent in a large hematoma.

- ✓ Vomiting is a common symptom for 50% of **hemispheric stroke,** a sign of raised intracranial pressure and very particular for **cerebellar hemorrhage.** Cerebellar hemorrhage equally has symptoms like bradycardia and lethargy, which are also signs of raised intracranial pressure.
- ✓ Coma is brought on by the involvement of **the reticular activating system of the brainstem**. Worsening of the neurological state usually happens as the hematoma with enlargement of the hematoma and an increase in edema.
- ✓ Aphasia, seizures, and hemianopia are seen in **lobar hemorrhage**. A prodrome of weakness, numbness, and tingling may also be present in patients with lobular bleeding. Seizures mostly occur at the onset of bleeding or within the first 24 hours.
- ✓ The hallmarks of **basal ganglia and thalamic bleeding (supratentorial hemorrhage)** include contralateral sensorimotor deficits, while in infratentorial **ICH**, signs of **brainstem** dysfunction manifest as ocular motor or other cranial nerve abnormalities and contralateral motor deficits.
- ✓ Loss of all sensory modalities is the principal symptom of **thalamic hemorrhage**.
- ✓ Ptosis, unresponsive pupil, and vertical gaze palsy can all result from **thalamic hematoma extension into the midbrain.**
- ✓ Usually, **pontine** hematoma produces coma and quadriparesis.

- Clinical symptoms of subarachnoid hemorrhage include
  - ✓ severe headache described as a thunderclap
  - ✓ Vomiting
  - ✓ Syncope
  - ✓ Photophobia
  - ✓ Nuchal rigidity
  - ✓ Seizures,
  - ✓ Decreased level of consciousness.
  - ✓ Signs of meningismus such as the Kernig sign (pain on straightening the knee when the thigh is flexed to 90 degrees) and Brudzinski sign (involuntary hip flexion on flexing the neck of the patient) may be positive.

II.2.8. Diagnosis of Stroke[79]

After a detailed clinical assessment, the diagnosis is made with imaging,

- Further neurovascular imaging is considered depending on the type of stroke

- **Non-contrast head CT** is the first-line of imaging and allows for the detection of acute hemorrhage. It equally allows for the detection of ischemic changes that occur after 6–24 hours (cannot be used to reliably identify earlier ischemia). Indicated in all patients suspected of having an acute stroke to rule out intracranial hemorrhage before administering thrombolytic therapy
- **Diffusion-weighted MRI**: Allows identification of ischemia earlier than a CT (within 3–30 minutes after onset) [29]. It also allows the detection of hyperacute hemorrhage. It evaluates the reversibility of ischemic injury
- **Perfusion-weighted imaging**: visualizes reduced perfusion and allows quantification of perfusion parameters, e.g., mean transit time, cerebral blood flow and cerebral blood volume
- **Perfusion-diffusion mismatch MRI:** allows identification of the penumbra (or "tissue-at-risk")
- **Magnetic resonance imaging** helps in diagnosing subacute hemorrhage as the hematoma is isodense to brain tissue. MRI equally aids in distinguishing between primary hemorrhage and the hemorrhagic transformation of an infarct. MRI can also detect underlying causes of secondary hemorrhages, such as vascular malformations, including tumors, cerebral vein thrombosis, and cavernomas

- **Other diagnostic tests** are considered to look for etiologies and complications;

Troponin, complete blood count, electrolytes, blood urea nitrogen, creatinine, and coagulation factors are among the tests that can be performed. Stroke is frequently linked to coronary artery disease; hence, an ECG and troponin are recommended. A full blood count may indicate an infection or be used to check for anemia. Any irregularities in electrolytes should be rectified. Contrast investigations have the potential to affect renal function, hence it is important to monitor blood urea nitrogen and Creatinine. Additionally, coagulation variables such as partial thromboplastin time (PTT), prothrombin time (PT), and International normalized ratio (INR) should be measured because increased values may indicate a potential cause of hemorrhagic stroke.

II.2.9. Treatment / Management

The possibility of complete neurological recovery reduces with every minute an acute stroke remains untreated. This forms the "time is brain" concept foundation, highlighting the importance of promptly diagnosing and managing acute stroke cases.

➢ **Ischemic Stroke**[80]

- Airway, breathing, and oxygenation: Supplemental oxygen is administered to patients to maintain the oxygen saturation above 94%. However, it is not recommended for nonhypoxic patients.

- Blood pressure (BP): Elevated BP is carefully reduced to a systolic BP below 185 mm Hg and diastolic BP below 110 mm Hg before initiating IV fibrinolytic therapy. Following the treatment, the BP should be maintained below 180/105 mm Hg for at least 24 hours. Suppose a mechanical thrombectomy is planned, and the patient has not received IV fibrinolytic therapy. In that case, it is reasonable to maintain their BP at or below 185/110 mm Hg before the procedure and at or below 180/105 mm Hg during and for 24 hours after the procedure.

- Temperature: In patients with acute ischemic stroke, hyperthermia (body temperature >38°C or 100.4°F) should be addressed by administering antipyretic medication.

- Blood glucose: Both hypoglycemia (blood glucose level <60 mg/dL) and hyperglycemia (blood glucose levels within the range of 140 to 180 mg/dL) should be treated in patients with acute ischemic stroke.

- IV alteplase: IV alteplase is recommended for patients with acute ischemic stroke at a dosage of 0.9 mg/kg, with the initial 10% given as a bolus over 1 minute (maximum dosage is 90 mg over 60 minutes). Eligible patients include those who meet the criteria within 3 hours or 3 to 4.5 hours of acute ischemic stroke witnessed symptom onset or when the patient was last known well or at baseline.

- Other IV fibrinolytics: In patients without contraindications who are eligible for mechanical thrombectomy, a single IV bolus of tenecteplase at 0.25 mg/kg (maximum 25 mg) can be administered instead of IV alteplase. However, IV defibrinogenating or IV fibrinolytic agents, other than alteplase and tenecteplase, are not recommended.

- Mechanical thrombectomy: Patients eligible for IV alteplase should receive it, even if mechanical thrombectomy is being considered. However, if a patient fulfills all 6 of the following criteria, they should undergo mechanical thrombectomy using a stent retriever or direct aspiration: (1) pre-stroke modified Rankin Scale score of 0 to 1; (2) AIS caused by an occlusion in the internal carotid artery or MCA segment 1 (M1); (3) age 18 or older; (4) NIHSS score of 6 or higher; (5) Alberta Stroke Program Early CT Score (ASPECTS) of 6 or higher; and (6) treatment can be initiated within 6 hours of symptom onset.

- Antiplatelet treatment: Aspirin administration is recommended within 24 to 48 hours after symptom onset. However, aspirin administration is typically delayed until 24 hours after treating a patient with IV alteplase. For patients diagnosed with minor, non-cardioembolic ischemic stroke who have not received IV alteplase, it is appropriate to initiate dual antiplatelet therapy with aspirin and clopidogrel within 24 hours after symptom onset.

➢ **Intracerebral Hemorrhage**[81]

Initial treatment constitutes reducing elevated BP within 2 hours of ICH onset and achieving the target BP within 1 hour in order to prevent complications and improve outcome and improve outcomes.

- In patients with mild-to-moderate ICH and an initial SBP between 150- and 220-mm Hg, the target SBP is 140 mm Hg to maintain the systolic blood pressure between 130- and 150-mm Hg. It is noteworthy that if these patients present with an initial systolic blood pressure above 150 mm Hg, rapidly lowering the SBP to below 130 mm Hg may cause potential harm.
- Anticoagulation should be discontinued immediately in patients with anticoagulant-associated ICH, and rapid reversal should be performed as soon as possible.
- Platelet transfusion may be considered in patients treated with aspirin who require emergency neurosurgery. However, platelet transfusion should not be administered for patients treated with aspirin who do not require emergency neurosurgery due to potentially harmful effects.
- Surgical management has reduced mortality for specific patients compared to medical management alone. Surgical options for managing ICH include minimally invasive hematoma evacuation with endoscopic or stereotactic aspiration, external ventricular drain insertion, and craniotomy.

➢ **Subarachnoid Hemorrhage (SAH)**

According to the latest guidelines from the European Stroke Organization (ESO) and AHA/ASA, early treatment of aneurysms is recommended to decrease the risk of rebleeding. The ESO recommends intervention within 72 hours after symptom onset. However, a recent meta-analysis on the timing of endovascular treatment in SAH indicates a lack of evidence regarding the optimal timing in SAH patients.

- Short-term antifibrinolytic therapy was considered a strategy to reduce the rebleeding risk; however, the routine use of tranexamic acid after SAH cannot be recommended.
- Between the onset of SAH symptoms and the obliteration of the aneurysm, AHA/ASA recommends maintaining SBP below 160 mm Hg. On the contrary, ESO suggests maintaining the SBP below 180 mm Hg until the ruptured aneurysm has been coiled or clipped. Complete obliteration of the aneurysm is the recommended goal, with endovascular coiling being the preferred treatment when a ruptured aneurysm is considered suitable for either coiling or clipping.
- In case of a patient experiencing a seizure associated with SAH, treatment with antiepileptic drugs is recommended. Short-term seizure prophylaxis may also be considered during the immediate posthemorrhagic period. Additional treatment goals include pain control, euvolemia, normothermia, and normoglycemia.

II.3. REVIEW OF STUDIES ON SLEEP AND STROKE

Sleep disorders and stroke are closely linked conditions that can have significant impacts on an individual's health and well-being. Sleep disorders are commonly observed in stroke patients and can have significant negative impacts on their overall health and recovery. Various studies have highlighted the bidirectional relationship between sleep and activity in the context of stroke recovery [19].

➢ **Prevalence of sleep disorders in stroke patients**

The prevalence of sleep disorders in stroke patients has been extensively studied, with findings consistently indicating a high frequency of sleep disturbances in this population. Research has shown that sleep disorders such as sleep-disordered breathing, insomnia, restless legs syndrome, circadian rhythm disorders, and periodic limb movement disorders are prevalent in stroke patients, with estimates ranging from 20% to 78% [82–84]. Additionally, sleep-related

breathing disorders were found in 40-70% of patients with acute stroke and are associated with worse recovery outcomes[85].

- **Sleep disorders associated with the risk of stroke**[86–89]

Evidence from meta-analyses and prospective studies has consistently demonstrated that sleep disorders, such as sleep-disordered breathing, insomnia, and restless legs syndrome, are associated with an increased risk of stroke[86–88].

OSA is a known risk factor for cerebrovascular diseases. Studies have shown that approximately 4-7% of the adult OSA population suffers from cerebral ischemia and cardiovascular death, and approximately 60% of stroke patients suffer from OSA. Obstructive upper airway induces paroxysmal hypoxia and leads to OSA during the process of sleeping. This in turn causes stroke due to hypoxia, which causes alterations in intrathoracic pressure, blood pressure fluctuations, and sympathetic activation, with possible mechanisms of endothelial dysfunction, oxidative stress, atherosclerosis, cardiac arrhythmia, hypercoagulation, paradoxical embolisms, and heart failure.

Furthermore, the impact of specific non-apnea sleep disorders or sleep problems on stroke risk has been highlighted, wherein REM sleep behavior disorders and insomnia, despite questioning of its overall association with the incidence of stroke, were shown to be associated with incident stroke in certain studies, providing novel targets for stroke prevention [87]. A study found that after controlling for other factors, short sleep (less than 5 to 6 hours per night) can accurately predict the occurrence of stroke [86]. In a cohort study, which included 21,438 insomniacs and 64314 non-insomniacs, insomniacs were shown to have a 54% higher risk of stroke compared to non-insomniacs. This difference was more evident in younger adults. It is still unclear why insomnia could lead to the development of stroke, but neuro-inflammation may play a role. Also, chronic stress caused by lack of sleep can increase the risk of stroke [11, 89]. Hypersomnia also affects stroke, with studies revealing that sleeping over nine hours per night strongly predicts stroke events after eliminating confounding factors. It has also been reported that excessive sleep is associated with a higher risk of stroke than short sleep [11, 86]. Circadian rhythm disorders, which involve disruptions in the sleep-wake cycle, have been identified as potential risk factors for stroke. During physiologic sleep, there is a decrease in sympathetic activity and an increase in parasympathetic activity, which causes a reduction in blood pressure, heart rate, cardiac output, and respiratory rate. Upon awakening, the sympathetic activity increases, leading to an increase in blood pressure and heart rate.

Disruption of the circadian rhythm can lead to alterations in alterations in blood pressure, heart rate, and other physiological processes, which may increase the risk of stroke.

Additionally, the association between sleep duration and stroke risk has been a focus of investigation, with evidence suggesting that both short and long sleep durations may be associated with an increased risk of stroke, highlighting the complex nature of this relationship [86, 90].

➢ **Pathophysiology and mechanism of sleep disorders in stroke patients** [11, 91]

The pathophysiology of sleep disorders in stroke patients is a complex and multifaceted area of study, with numerous factors contributing to the development and impact of sleep disturbances in this population.

- Insomnia in stroke has been attributed to several mechanisms. Studies have shown that stroke patients who suffer from certain psychological diseases like depression and anxiety usually have trouble sleeping and suffer from insomnia [92]. Also, insomnia is common in the acute phase of stroke and is usually a result of environmental factors like hospital environment, noise, and lights. Additionally, insomnia on stroke has been directly associated with brain injury [93]. Insomnia is caused by strokes in the thalamic and brainstem regions, leading to inversion of the sleep-wake cycle. Ponto-mesencephalic strokes have been reported to cause complete sleep loss, while thalamic strokes result in brain wave lack. Supratentorial, left hemispheric, or paramedian thalamic strokes have equally been reported to decrease non-REM (NREM), and right hemispheric strokes decrease REM sleep. In addition, insomnia was also associated with damage to some specific areas of the cerebral cortex in patients with penetrating brain damage. Furthermore, sleep disorders have been found to result or worsen as a result of certain medications used in treating comorbidities in stroke patients, like hypertensive patients on medications such as beta-blockers, clonidine, or diuretics, which can disrupt REM sleep, induce insomnia, and lead to early morning awakening, bad dreams or painful calf cramps during sleep. For stroke patients with psychiatric symptoms, who are placed on selective serotonin reuptake inhibitors (SSRIs) such as sertraline or paroxetine, which reduce REM sleep and enhance exhaustion throughout the day.
- Post-stroke hypersomnia is defined as exacerbated sleep propensity with excessive daytime sleepiness, increased daytime napping, or lengthened nighttime sleep following a cerebrovascular accident. Hypersomnia in stroke has been attributed to a reduction in the activation threshold due to damage to the ascending reticular system (ARS). Bilateral thalamic

lesions, thalamic-mesencephalic lesions, upper pons lesions, and lesions to the medial pontomedullary region—that is, the region where RAS fibers are concentrated—are among the lesions that are likely to cause marked hypersomnia. The activating system is less impacted by cortical and subcortical lesions, with the exception of thalamic lesions, because RAS projections are more dispersed in the cortex unless a very large lesion compresses the upper brainstem as a result of edema. One of the most common forms of excessive daytime sleepiness is stroke in the paramedian thalamus. Also, large lesions in the left hemisphere and anterior regions result in increased hypersomnia. Most frequently, hypersomnia is associated with memory and cognitive disturbances and lack of attention due to impairment of specific physiologic functions in the sleep-wake cycle. Hypersomnia has been found to improve within months of stroke, but other defects like cognitive disturbances have been found to persist.

- Breathing–related sleep disorders have been found to be very frequent and severe, with high mortality in acute cerebrovascular diseases, though improvement has been recorded with stroke recovery. Sleep disordered breathing (SDB) relationship with stroke is the most frequently studied. A bi-directional relationship has been enumerated, with SDB shown as a risk factor for stroke and as a consequence of stroke. Acute stroke patients frequently experience abnormal breathing patterns, which are frequently brought on by the site of the lesion or pre-existing SDB. Patients with medullary infarcts, severe deficits, sleep difficulties, and reduced consciousness are more likely to exhibit these patterns. breathing abnormalities may arise from ictal lesions that impact the breathing muscles. Respiratory apraxia may result after a stroke in the internal capsule, basal ganglia, or frontal brain. Numerous respiratory patterns, such as neurogenic hyperventilation, apneustic respiration, ataxic respiration, central apnea syndrome, Ondine's curse, and hypoventilation, can be brought on by brainstem strokes. Both automatic and voluntary breathing may be hampered by medullary strokes at the C1 level. Several respiratory patterns are not connected with a prognostic significance, with the exception of tachypnea with low CO2, which is associated with poor prognosis.
- Parasomnia is mainly defined as motor or sensory activity that occurs during sleep, whether during the REM or NREM phases. Cases of tegmental pontine stroke have been described in association with behavior disorders during REM sleep (RBD). Most of the frequent parasomnia is REM- sleep behavior disorders and restless leg syndrome (RLS), which is often accompanied by periodic limb movements, which are involuntary movements during NREM sleep. Stroke lesions in the basal ganglia and brainstem infarcts have been linked to RLS in stroke patients. Furthermore, strokes affecting the thalamus, temporal, parietal, and

occipital lobes may cause a syndrome known as dream-reality confusion and an increase in dreaming and nightmares.

- Circadian rhythm disorders have been identified as potential risk factors for stroke and have also been reported to result from stroke. Circadian rhythms are altered when infarcts affect growth hormone and melatonin secretion, and right insular infarcts may increase post-infarct morbidity and mortality.

➢ **Impact of stroke on sleep** [19]

Sleep disorders can negatively affect recovery, increase the risk of recurrence and death, and hinder patients' rehabilitation progress. Sleep plays an important role in the learning of new motor skills. Consolidation of procedural memory occurs in REM sleep and enables long-term potentiation and neuroplasticity. Most studies use the modified Rankin Scale(mRs) or Barthel's index to measure functional status in stroke patients. Other measures of functional activity include the Functional Independence Measure (FIM), Utrecht Scale for Evaluation of Rehabilitation (USER), Functional Ambulation Categories (FAC), Berg Balance Scale (BBS), 9-hole peg test, and Purdue Peg test. Measures of participation included the 36-item Short Form Survey (SF-36), EuroQol-5, 8-item Short Form Survey (SF-8), and the Stroke Specific Quality of Life (SSQoL).

A meta-analysis conducted in 2020 [19], studied the impact of sleep apnea (SDB), Restless leg syndrome, Insomnia, and Excessive daytime sleepiness. Conclusions drawn were: People with stroke and a concomitant sleep disorder may have poorer outcomes in terms of functional recovery and participation compared to those without a sleep disorder. Also, sleep disorders have a negative impact on the overall health and quality of life of stroke survivors, potentially hindering their ability to regain functional abilities and participate in daily activities. In addition, addressing sleep disorders in stroke rehabilitation programs may be important for improving outcomes and promoting better recovery and participation among stroke survivors.

Furthermore, sleep disorders in stroke patients are associated with cognitive impairment, reduced quality of life, increased risk of stroke recurrence, and undesirable effects on neurological recovery and functional outcomes [19, 94, 95]. Patients who suffer from stroke and experience sleep disturbances are often associated with other conditions, such as anxiety,

depression, memory loss, focus problems, muscle cramping, cold sensitivity, and dry skin, in addition to fatigue. [92, 96].

- **Diagnosis and management of sleep disorders in stroke patients**[11]

Accurate diagnosis of sleep disorders in stroke patients is crucial for effective treatment and management. Several assessment tools and methods are used to diagnose sleep disorders in stroke patients, including polysomnography, actigraphy, and questionnaires such as the Pittsburgh Sleep Quality Index and Epworth Sleepiness Scale.

Polysomnography is the gold standard for diagnosing sleep disorders and provides comprehensive information about different sleep stages, breathing patterns, and movements during sleep. Actigraphy is a non-invasive method that uses a wrist-worn device to monitor sleep-wake patterns over multiple days, offering valuable insights into sleep efficiency and circadian rhythms.

Additionally, questionnaires such as the

- The Pittsburgh Sleep Quality Index, which can assess different domains of sleep, including sleep duration, disturbances, and overall quality, Pittsburg Sleep Quality Index (PSQI) [36]. This is a self-rated questionnaire designed to evaluate sleep quality and sleep disturbances over a one-month interval. It contains 24 questions aimed at evaluating seven main areas: sleep latency, sleep duration, sleep quality, habitual sleep efficiency, sleep disturbances, sleep medication use , and daytime medication. Every area is being graded on a scale of 0- 3, wherein 3 is extreme negativity. Questions rated by the roommate or bed partner are included in clinical purposes and are not scored. Total score range 0-21. A high score indicates poor sleep quality. A global score above or equal to 5 indicates poor sleep quality, while a score below or equal to 4 indicates good sleep quality.

- Epworth Sleepiness Scale measures daytime sleepiness [97] The ESS is a subjective measure of a patient's sleepiness and consists of 8 situations by which the client or patient rates his tendency to be sleepy. Each of these situations is scored on 3. A scale of 0 indicates no chance of dozing, and 3 indicates a high chance of dozing, with a total score range of 0 to 24. Interpretation;

- ✓ 0-7: Patient is unlikely to be abnormally sleepy.
- ✓ 8- 9: Patient has an average amount of daytime sleepiness.
- ✓ 10-15: Depending on the situation, the patient may be excessively sleepy and may want to consider seeking medical attention.
- ✓ 16-25 Patient is excessively sleepy and should consider seeking medical attention.

EDS is an ESS score ≥ 11. Mild excessive daytime is a score of [11-14], Moderate excessive daytime sleepiness is an ESS score of [15-17] and Severe excessive daytime sleepiness is a score of [18-24].

- STOP-BANG and Berlin questionnaires for OSA are used to evaluate the risk for obstructive sleep apnea. The STOP-BANG is a highly specific tool used in screening for the risk of obstructive sleep apnea [39, 98]. It assesses 8 items related to clinical factors of sleep apnea: snoring, tiredness, observed apnea, high BP, BMI, age, neck circumference, and male gender. Each of the 8 items has a dichotomous grading (yes or no) with a total score range of 0 to 8. This score was simple to use for screening and had a high diagnostic accuracy.

Low risk of OSA: Yes 0-2

Intermediate risk of OSA: Yes 3-4

High risk of OSA: Yes 5-8

- Berlin questionnaire which is less specific and more sensitive as compared to the STOP-BANG questionnaire [98, 99] This tool permits the identification of patients likely to have sleep apnea. It includes 1O questions that focus on 3 categories of apnea signs and symptoms: snoring, daytime sleepiness, and obesity/high blood pressure. A score of 0 = never or almost never, 1 = 1 to 2 times a month, 2 = 1 to 2 times a week, 3 = 3 times a week, and 4 = almost every day.Patients can be classified as high risk and low risk based on the responses to the individual items in the various categories.
  - ✓ High Risk: if there are 2 or more categories where the score is positive.
  - ✓ Low Risk: if there is only 1 or no categories where the score is positive.

These tools are most used in prevalence studies of sleep disorders in stroke patients [12, 17, 84, 100]. These tools can help identify specific sleep disorders, such as obstructive sleep apnea, central sleep apnea, insomnia, and periodic limb movements in sleep. Implementing these

assessments in clinical practice can aid in accurately diagnosing and effectively managing sleep disorders in stroke patients. With a comprehensive understanding of the specific sleep disturbances experienced by each patient, healthcare providers can tailor treatment plans and interventions to address their individual needs and challenges.

Studies have revealed certain factors to be associated with the occurrence of stroke.

➢ **Management**[11]

Treatment of sleep disorders is essential for stroke patients' recovery and general health. Stroke patients frequently experience sleep disturbances, such as insomnia and sleep-disordered breathing (SDB), which have been linked to poor recovery results. Research indicates that treating sleep disturbances in stroke patients may enhance their recovery from the condition, emphasizing the importance of treating these disorders in stroke patients' care.

- **Treatment of insomnia in stroke patients**: Treatment options include

  ✓ Non-pharmacological measures like prevention from light and noise at night, increased exposure to daylight, and acupuncture (a technique that involves inserting needles into the skin to stimulate certain areas of the body) have recently been proven by several randomized control trials to help in the treatment of insomnia.

  ✓ Pharmacologic measures include the use of antidepressants like mianserin, which has been reported to improve insomnia after stroke. In addition, Zolpidem has been seen to improve stroke prognosis by increasing brain-derived neurotrophic factor secretion and protection of neurovascular unit in acute stroke. Zolpidem (a hypnotic) should be used with care as it is reported to be related to ischemic stroke in high doses. Benzodiazepines are not recommended in the treatment of insomnia in stroke patients due to the possibility of aggravating breathing-related sleep disorders and triggering the reappearance of new motor deficits.

- **Treatment of hypersomnia in stroke patients**

The management of hypersomnia in stroke patients is challenging. Notwithstanding, certain measures have been recorded to improve hypersomnia symptoms in stroke patients.

  ✓ Methylphenidate and levodopa

  ✓ Anti-depressants also ameliorate hypersomnia symptoms

- **Breathing-related sleep disorders treatment in stroke patients**

Continuous positive airway pressure (CPAP) therapy is commonly used to treat obstructive sleep apnea (OSA) in stroke patients, as it helps maintain an open airway during sleep.

Other treatment options for OSA in stroke patients include oral appliances, positional therapy, surgical options like hypoglossal nerve stimulation, upper airway surgery, nasal reconstruction, mandibular advancement (especially in people unable to tolerate CPAP), and weight loss interventions.

For central sleep apnea (CSA) in stroke patients, adaptive servo-ventilation therapy is often recommended to stabilize breathing patterns during sleep. In some cases, supplemental oxygen therapy may be used to treat breathing-related sleep disorders in stroke patients. It is important to address any underlying causes or risk factors for breathing-related sleep disorders, such as obesity or medication side effects, as part of the treatment approach.

Treatment decisions should be individualized based on each stroke patient's specific needs and characteristics, and close monitoring of treatment effectiveness is necessary. Benzodiazepines and anti-depressants should be avoided as they may negatively influence CSA.

- **Treatment of parasomnias in stroke patients**

Options include treatment of RLS and RBD

✓ General measures: Avoiding medications, including alcohol, stimulants, SSRIs, and selegiline, which have been found to worsen RBD. Antidepressants and diazepam should be used with care due to their adverse effects on RLS.

✓ Pharmacotherapy includes;

- Administering before sleep clonazepam at doses ranging from 0.25-2.0mg.
- Clonazepam and melatonin can decrease violent attacks and improve dream enactment.
- Fluoxetine has also been found to be effective in stroke-associated RBD.
- Levodopa, dopamine agonists (pramipexole and ropinirole), and gabapentin can also be used to treat stroke-related RLS/PLMS patients.

- **Conclusion**

Sleep disorders are highly frequent among stroke patients, with a range of sleep disturbances such as hypersomnia, insomnia, parasomnias, periodic limb movements in sleep, and sleep-related breathing disorders. In addition to hindering successful recovery, these sleep disturbances can have a substantial negative influence on stroke patients' general health and quality of life by limiting their ability to participate in rehabilitation exercises Moreover, poor sleep quality has been connected to depression, anxiety, cognitive impairment in stroke patients, as well as a detrimental effect on their ability to recuperate functionally and return to work.

Thus, studies on sleep disorders in stroke victims are essential to enhancing stroke prevention, optimizing rehabilitation, improving post-stroke outcomes, and lowering the burden of comorbidities linked to sleep disturbances.

- **Review of studies on sleep disorders in stroke patients in Africa**

✓ In a cross-sectional study done by Mansour et al. in Egypt [15], to determine the prevalence of sleep disorders in stroke patients at the outpatient clinic at Ain Shams University Hospital from January 2015 to December 2015, a total of 75 patients were enrolled. Out of these patients, 45.3% were males, and 54.7% were females. The mean age ± standard deviation (SD) was 59.3 ± 5. In this study, the prevalence rate of sleep disorders was 70.6%. The common sleep disorders were Insomn**ia** (14.7%), excessive daytime sleepiness (17.3%), parasomnias (Nightmares- 9.3%, arousal confusion- 6.7%, sleep talking – 6.7%, sleep paralysis – 4%), sleep-disordered breathing (29.5%). Also, sleep disorders were more prevalent in moderate severity of stroke by NIHSS and in the first 1-4 weeks, though it wasn't statistically significant.

✓ Another cross-sectional study assessed the prevalence of sleep apnea risk and its clinical correlates and predictors among Ghanaian stroke survivors attending the neurology clinic of the Komfo Anokye Teaching Hospital, a tertiary medical center in Kumasi, Ghana [101]. A total of 200 stroke patients were included in the study. Results revealed that 52.5% were males and 47.5% females. The median age was 62, with a range of (52 -72). 49.5% of the patients were identified as having a high risk of sleep apnea with the help of the Berlin questionnaire and STOP-BANG questionnaire, which classified 26 (13%), 137 (68.5%), and 37 (18.5%) subjects as low, intermediate, and high risk for sleep apnea respectively. A high

risk of sleep apnea was found to be associated with advanced age, excess alcohol intake, and poor ability to perform daily activities despite their NIHSS scores being lower than those who had a low risk of sleep apnea [101].

✓ Furthermore, another study by Iwouzo et al. was carried out in the neurology outpatient clinics of two tertiary hospitals in North Central, Nigeria, respectively, from February 2021 to January 2022. Its aim was to investigate the frequency and associated factors of sleep disturbances among stroke survivors. A total of 110 stroke survivors were included in the study [13]. In this descriptive cross-sectional hospital-based study, Iwouzo et al. noted that the prevalence of sleep disorders among stroke patients was found to be 33.6%. In this study, the majority of the cases were ischemic stroke; 72.7 % were males, and 27.3% were females. The commonest sleep disorders included insomnia (19, 17.3%), hypersomnia (10, 9.0%), sleep-disordered breathing (5, 4.5%), and sleep-related movement disorder (3, 2.7%), respectively. Using the ESS score, 22 (20.0%) had mild, 10 (9.0%) had moderate, and 7 (6.4%) had severe ESS scores, respectively. The mean age of the patients was 60.9 ± 2.9. Univariate analysis showed depression to be significantly associated with ESS (P = 0.006), whereas multivariate analysis revealed age and sex as significant associated factors (P = 0.008 and P = 0.009) of ESS.

➢ **Review of sleep studies in Cameroon**

Sleep has been studied in Cameroon and linked to various medical conditions, but studies in its link or association to stroke are rare.

A study was conducted by Tcheukam et al. in 2023 to assess sleep quality among school-going teenagers in urban and semi-urban environments. The study included 952 participants, 309 from students in semi-urban areas and 643 from those in urban areas. The mean age of the study population was 16.33 ± 1.70 years, with extremes of 11 and 19 years. Most of them were aged between 14 and 16 (53.4%) and female (52.0%). The study found a higher frequency of the use of psychoactive substances and illegal drugs in urban teenagers than in semi-urban teenagers. Sleep quality was found to be poor in 41.0% of students, 46.3% in urban areas, and 29.3% in semi-urban areas, with a statistically significant difference between the two groups ($p< 0.001$). Insomnia was the most prevalent sleep disorder in the study population, at 19.4%, followed by excessive sleepiness, at 15.5%. The independent risk factors for poor sleep quality in the students who lived in an urban area were, being aged between 17 and 19 years, being

female, being enrolled in the first or final year of secondary school, taking legal drugs, and having definite anxiety and depression.

A cross-sectional study was done by Legoum et al in Cameroon [102] , to evaluate the effect of sleep duration and sleep quality on glucose metabolism in an urban and rural population in Cameroon. Out of 249 rural and 250 urban community dwellers, 39.1% were males and 59.9% were females. The mean age ± standard deviation (SD) was 36 ± 12. In this study, the frequency of poor sleep quality was 50.3% and was similar in urban and rural groups. Short SD was present in 30.5% of subjects and was more frequent among urban dwellers. This study noted a significant association between short sleep duration as well as the combination of short sleep duration and poor sleep quality with type 2 diabetes prevalence.

Another study by Massongo et al. [16] , evaluated Sleep apnea syndrome (SAS) prevalence and comorbidity with other non-communicable diseases and HIV in hospitalized patients in Yaoundé Cameroon. The study cross-sectional study was conducted in the cardiology, endocrinology, and neurology departments of the Yaoundé Central Hospital. This study showed that the mean age of 110 patients who presented a valid sleep monitoring report was 58 ± 12.5 (28–87) years, and 53.2% were female. The prevalence of SAS was 55.0 %, and the one of moderate to severe SAS was 34.2 %. The majority pattern was obstructive, accounting for 90.2% of SAS and 86.8% of MS-SAS. Among particular comorbidities, the prevalence of SAS varied from 52.2% to 75.0%. Higher percentages of SAS patients than SAS-free patients had a history of stroke (36.7% vs. 32.0%, p = 0.756), hypertension (75.4% vs. 48.0%, p = 0.005%), heart failure (23.0% vs. 12.0%, p = 0.213), and combined cardiovascular comorbidity (80.3% vs. 52.0%, p = 0.003).

Furthermore, a case-control study by Njamnshi et al. [104] assessed the sleep patterns of hypertensive patients in Cameroon with the aid of clinician-friendly, readily available, less costly, and easy-to-use sleep questionnaires in order to generate preliminary data on the likelihood (risk) of obstructive sleep patterns (and consequent daytime sleepiness) in hypertensive patients compared to normotensive participants. The study included 50 hypertensive participants, age and sex-matched with 54 normotensive participants at the outpatient cardiology and neurology departments of the Yaoundé Central Hospital. The prevalence of snoring was higher in participants with hypertension compared to normotensives (58.0% versus 44.0%, respectively), though not significantly (p = 0.167). Nevertheless,

compared to the controls, the hypertensive cases (average age 54.78 ± 8.79 years, mean time since diagnosis 4.46 ± 4.36 years) had a significantly higher likelihood of having obstructive sleep apnea (OSA) (OR = 5.03; 95% CI, 1.90–13.33, p = 0.001), but there was no significant increase in daytime sleepiness as a result (p = 0.421). There was no discernible pattern found in the relationship between OSA risk, daytime drowsiness, and hypertension severity. Participants with managed hypertension exhibited lower rates of OSA risk than those with uncontrolled hypertension (50.0% versus 63.2%, p = 0.718), albeit the difference was not statistically significant.

Conclusion

With the information in this section, sleep disorders are directly or indirectly associated with vascular diseases. Its study in cerebrovascular diseases, particularly stroke, is rare in Cameroon and is much needed for a better understanding and management of stroke patients.

CHAPTER III: METHODOLOGY

III.1. STUDY DESIGN

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

III.2. STUDY AREA AND SETTING

This study was carried out in the outpatient neurology and cardiology services of the Yaoundé Central Hospital (YCH):

The Yaoundé Central Hospital (YCH) is a category two hospital in the city of Yaoundé

Yaoundé (spread over seven hills) is the capital of Cameroon, with a population of more than 2.5 million. It lies in the center region of the nation at an elevation of about 750m above sea level. Most of Yaoundé's economy is centered on the administrative structure of the civil and diplomatic services. Owing to these high-profile central structures, Yaoundé has a higher standard of living and security than the rest of the country.

The Yaoundé Central Hospital is located behind CENAME (National Central of Essential Medicines) and not far from Camp Sic Mesa in the capital city of Yaoundé. It has several specialization units: the Surgery and Specialties Unit, the Home Unit of Anesthesia and Intensive Care Emergency, the Gynecology/Obstetrics Unit, and the Medicine and Specialties Unit. It receives a daily influx of patients from Yaoundé, its environs, and other health facilities. The Neurology unit includes a hospitalization unit, a nurse station,3 consultation offices, a waiting room, and an electroencephalogram room. It receives patients with neurological disorders of various types, of which stroke makes up a good number of the cases received. There is no rehabilitation unit; stroke patients are being followed up by the consulting neurologist on outpatient consultations.

III.3. STUDY PERIOD

This study was carried out over six months, from November 13th, 2023, to May 16th, 2024.

III.4. STUDY POPULATION

III.4.1. Source of population

Our study participants were stroke patients attending the outpatient unit at the neurology and cardiology services of the YCH.

III.4.2. Inclusion criteria

Participants with a confirmed diagnosis of stroke with neuroimaging or a prior neurologist diagnosis of stroke in the case of negative imaging results.

- Patients within the age range of 18 years to 70 years old.
- Patients who gave their consent.
- Patients who were at least 2 weeks post-event but not more than 6 months post-event.

III.4.3. Exclusion criteria

- Patients who opted out in the course of the study.
- Patients who had been diagnosed with primary sleep disorders prior to stroke onset.
- Patients who have an end-stage medical illness and those with a neurological disease or a psychiatric disorder diagnosed by a physician which is known to affect sleep.
- Patients with profound aphasia without a caretaker who can give information.
- People with impaired consciousness.

III.5. SAMPLING OF PARTICIPANTS

III.5.1. Sampling method

An exhaustive consecutive sampling was carried on all patients who met up with our inclusion criteria during the study period.

III.5.2. Sample size estimation

The minimum sample size was calculated using the formula for descriptive studies:

N = Z2. pq / e2

where:

- n is the sample size
- z is the z-score for the desired confidence level
- $\hat{p}$ is the estimated prevalence of stroke patients in Cameroon
- E is the margin of error

Assuming a 95% confidence level, a 5% margin of error, and a prevalence of 7.3% [4] the calculation would be:

n = (1.96^2 * 0.073 * (1 - 0.073)) / (0.05) ^2
≈ 103.43= **104 patients**.

III.6. STUDY PROCEDURE

Prior to recruiting patients, ethical clearance was obtained from the Ethics Committee of the Faculty of Medicine and Biomedical Sciences of the University of Yaoundé I. Furthermore, administrative authorizations were obtained from the Director of the YCH. The aim of the study was explained to participants, and their written consent was taken.

- Sampling was done in a consecutive manner.
- Stroke patients were received at the outpatient service for routine follow-up visits.
- Stroke patients were seen at the in-patient wards. The study was explained to the patients using an information notice, and consent was obtained from those who agreed to participate in the study during their outpatient visits.
- At the outpatient service, stroke patients were given an information notice in French and English. After that, those who gave their consent were asked questions from the questionnaire. Aphasic patients' information was gotten from their caretaker, who knew the most about the patient's history, sleep, and behavior post-stroke.
- The questionnaire form comprised four parts: the first part was to get the sociodemographic characteristics, the second part contained information on the present physical examination, the third part contained standardized sleep study scales, and the last part of the questionnaire included functional evaluation scales.
- Patients underwent a detailed history and clinical examination and were asked specific sleep-related questions. The questions were read in a language the patients were versed in, and their responses were noted.

III.7. DATA COLLECTION

III.7.1. Resources

➢ **Human resources**

- **Principal investigator:** MBANGE LIKOWO GERMAINE, 7th-year General medicine student
- **Supervisors:** Professor Alfred K. Njamnshi, Doctor Leonard Ngarka.
- **Statistician.**

➢ **Materials**

- **For data collection**
  - ✓ Structured questionnaire including score
  - ✓ Pittsburg Sleep Quality Index (PSQI).
  - ✓ STOP BANG questionnaire.
  - ✓ The Epworth Sleepiness Scale (ESS)
  - ✓ Hospital Anxiety and Depression scale (HADS)
  - ✓ Modified Ranking Scale (mRS)
  - ✓ Barthel index
  - ✓ Pens, pencils, erasers,
- **For patient examination**
  - ✓ Data sheet
  - ✓ Reflex harmer
  - ✓ Gloves
  - ✓ Blood pressure machine
  - ✓ A weight balance
  - ✓ Alcoholic solution
- **For data analysis**
  - ✓ Laptop computer with statistical analysis software
  - ✓ A USB flash disk of 2GB
  - ✓ A scientific calculator
- **For research**
  - ✓ Scientific articles
  - ✓ Science textbooks
  - ✓ Past thesis
  - ✓ Internet connection

III.7.2. Data collection procedure

Potential study participants were approached by the primary investigator. Those who could read, were able to read the consent form. Otherwise, they were given a comprehensive explanation of the consent form by the primary investigator in their preferred language (English or French). In all cases, the project's aim was explained to the participants.

Participants were permiited to ask questions before giving their consent. Consented subjects were interviewed by the primary investigator,

- Information on socio-demographic and clinical characteristics was obtained with the help of a structured questionnaire;
- Physical examination, which consisted of evaluating and recording their general state, vital parameters, and full neurologic examination, was conducted, after which,
- Sleep was assessed with the help of standardized tools, which include the Pittsburg Sleep Quality Index (PSQI), Epworth Sleepiness Scale, Berlin questionnaire; thereafter
- Patients' functional status was evaluated with the help of the Modified Ranking scale and Barthel's index

III.8. DEFINITION OF VARIABLES TO BE STUDIED

III.8.1. Sociodemographic variables

- Name
- Age
- Sex
- Marital status
- Social status
- Lifestyle (alcohol, smoking, physical activity)

III.8.2. Clinical parameters

- Past Medical history (hypertension, Diabetes, prior sleep disorder, heart disease)
- Past family history (narcolepsy, diabetes, obstructive sleep apnea).
- Stroke clinical variables
  - ✓ Type (Ischemic, hemorrhagic)
  - ✓ hemisphere (Dominant hemisphere, non-dominant hemisphere)
  - ✓ Lesion (Cortical lesions, subcortical lesions, Brain stem lesion, other lesions
  - ✓ Severity (NIHSS; National Institute of Health Stroke Scale)
  - ✓ Time from symptom onset

III.8.3. Measurements

- The weight of study participants was measured in kilograms using a scale while the height in centimeters was measured using a meter tape. Alternatively, the height was obtained from the patient's ID card. The height was used to calculate the body mass index (BMI). For patients who were unable to stand to get their weight, their last weight from the previous month was used. If that was not available, the patient's waist circumference was measured, and if it was above 102 cm for males and 88 cm for females, it was classified as obesity.
- The neck circumference was measured using a meter tape at the midpoint of the neck between the mid-cervical spine and the mid-anterior neck, to the nearest 0.5cm, just below the laryngeal prominence if palpable. Neck circumference was considered high when it measured more than 40 cm.
- Current smoking and alcohol intake history were ascertained from either the patient or a reliable relative.

III.8.4. Sleep Disorders were evaluated with the help of the following tools

The Pittsburg Sleep Quality Index and Epworth Sleepiness Scale were used in the diagnosis of Insomnia, and Excessive daytime Sleepiness respectively.

The STOP-BANG Questionnaire was used as a screening tool for Obstructive sleep apnea and The Pittsburg Sleep Quality Index as a screening tool for Restless legs syndrome.

- **Pittsburg Sleep Quality Index (PSQI)** [105, 106]

This is a self-rated questionnaire designed to evaluate sleep quality and sleep disturbances over a one-month interval. It contains 24 questions aimed at evaluating seven main areas: sleep latency, sleep duration, sleep quality, habitual sleep efficiency, sleep disturbances, sleep medication use , and daytime medication.

Every area is being graded on a scale of 0- 3, wherein 3 is extreme negativity. Questions rated by the roommate or bed partner are included in clinical purposes and are not scored. Total score range 0-21. A high score indicates poor sleep quality.

A global score above or equal to 5 indicates poor sleep quality.

A global score below or equal to 4 indicates good sleep quality

This tool was been used to evaluate our patients' sleep quality and to diagnose insomnia in these patients. The diagnosis of insomnia was based on DSM-5 criteria. Restless legs syndrome was equally screened with the help of this tool and based on DSM-5 criteria

- Insomnia was present as per DSM-5 criteria if a person complained of either difficulty in initiating sleep or maintaining sleep or early morning awakening, occurring at least 3 times per week associated to daytime dysfunction (fatigue, somnolence, impairment in social activities and behavior
- Restless leg syndrome was confirmed as per DSM-5 criteria if a patient complained of an urge to move the leg, which begins or worsens at rest, occurs, or is worse in the evening, and is partially or totally relieved by moving. The symptom had to occur atleast 3 times a week and caused significant distress or functional impairment.

➢ **Epworth Sleepiness Scale** [97]

This is a subjective measure of a patient's sleepiness and consists of 8 situations by which the client or patient rates his tendency to be sleepy. Each of these situations is scored on 3. A scale of 0 indicates no chance of dozing, and 3 indicates a high chance of dozing, with a total score range of 0 to 24. Interpretation;

- 0-7: Patient is unlikely to be abnormally sleepy.
- 8- 9: Patient has an average amount of daytime sleepiness.
- 10-15: Depending on the situation, the patient may be excessively sleepy and may want to consider seeking medical attention.
- 16-25 Patient is excessively sleepy and should consider seeking medical attention.

EDS was considered as a score ≥ 11. Patients who score [11-14] experience mild excessive daytime sleepiness, while those with an ESS score of [15-17] have moderate excessive daytime sleepiness, and individuals scoring [18-24] on the ESS have severe daytime sleepiness.

➢ **STOP-Bang Questionnaire** [107]

It is a highly specific tool used in screening for the risk of obstructive sleep apnea. It assesses 8 items related to clinical factors of sleep apnea: snoring, tiredness, observed apnea, high BP, BMI, age, neck circumference, and male gender. Each of the 8 items has a dichotomous

grading (yes or no) with a total score range of 0 to 8. This score was simple to use for screening and had a high diagnostic accuracy.

Low risk of OSA: Yes 0-2

Intermediate risk of OSA: Yes 3-4

High risk of OSA: Yes 5-8

- **Hospital Anxiety and Depression scale**[108, 109]

This is a self-reported tool that enables the measurement of depression and anxiety in hospital outpatient clinic settings. It consists of 14 items, each with a scale range of 0-3. There are 7 items for each subscale: Anxiety and Depression. For each subscale, the total score range of the 7 items is 0-21; the total score is the sum of the 14 items.

A score of 0-7 for non-cases, 8-10 for borderline/mild cases, and 11 to 21 for definite cases.

- **Barthel index**[110]

We will be using this 10-item index for the Activity of Daily Living Assessment (ADL) assessment. This index permits the evaluation of functional independence and mobility in day-to-day activities. The 10 items include; feeding, bathing, grooming, dressing, bowel control, bladder control, toilet use, chair transfer, ambulation and stair climbing.
Items are graded according to the level of nursing needed by the patient. Persons who perform activities independently, with some assistance, or are dependent are scored 10, 5, and 0, respectively. There are various interpretations of the score, and it is recommended that it should not be used alone as the only functional evaluation scale. One of such interpretations reported by David Goldemun was;

- 0-40 highly dependent
- 45-60 partially dependent
- 65-95 minimally dependent
- 95-100 Independent

For our study, we considered a cut-off of 95, as reported by the American Heart Association [108]. A score ≥ 95 was considered a good outcome, and a score < 95 was considered a poor outcome.

- **Modified Rankin Scale**[111, 112]

A tool used in measuring the degree of disability in patients from stroke or other causes of neurological disability is as follows;

0 = No symptom
1 = No significant disability in spite of some symptoms, able to carry out all activities.
2 = Slight disability, looks after his own affairs without help, but unable to carry out all activities
3 = Moderate disability. Requires some help but is able to walk without assistance.
4 = Moderately severe disability. Unable to carry out personal activities unassisted and walk unassisted
5 = Severe disability; the individual is bedridden and needs nursing care.
6 = Dead

A score of 0- 2 represents a good functional outcome, and 3-6 represents a poor functional.[112]

III.9. DATA MANAGEMENT AND ANALYSIS

III.9.1. Data management

The data collected was cross-checked and entered into epi info. It was stored on a password-protected laptop, and an external hard drive was used for backup. A copy of the work was also be self-emailed.

III.9.2. Data analysis

Data from completed and validated questionnaires was entered and analyzed using Epi info version 7.2.4.0. A confidence interval of 95% was used. For descriptive statistics, quantitative variables (continuous variables) were presented as mean ± standard deviation (SD) when they were normally distributed and as median (25^{th} percentile, 75^{th} percentile) when data was skewed, while categorical variables were presented as frequencies and percentages. Relationships between continuous quantitative variables were established using independent samples *t*-test and analysis of variance, and that between categorical variables was established with chi-square. Factors associated with sleep disturbances were determined using multiple

logistic regression analysis after bivariate analysis. $P < 0.05$ was used to determine statistical significance.

III.10. ETHICAL CONSIDERATION

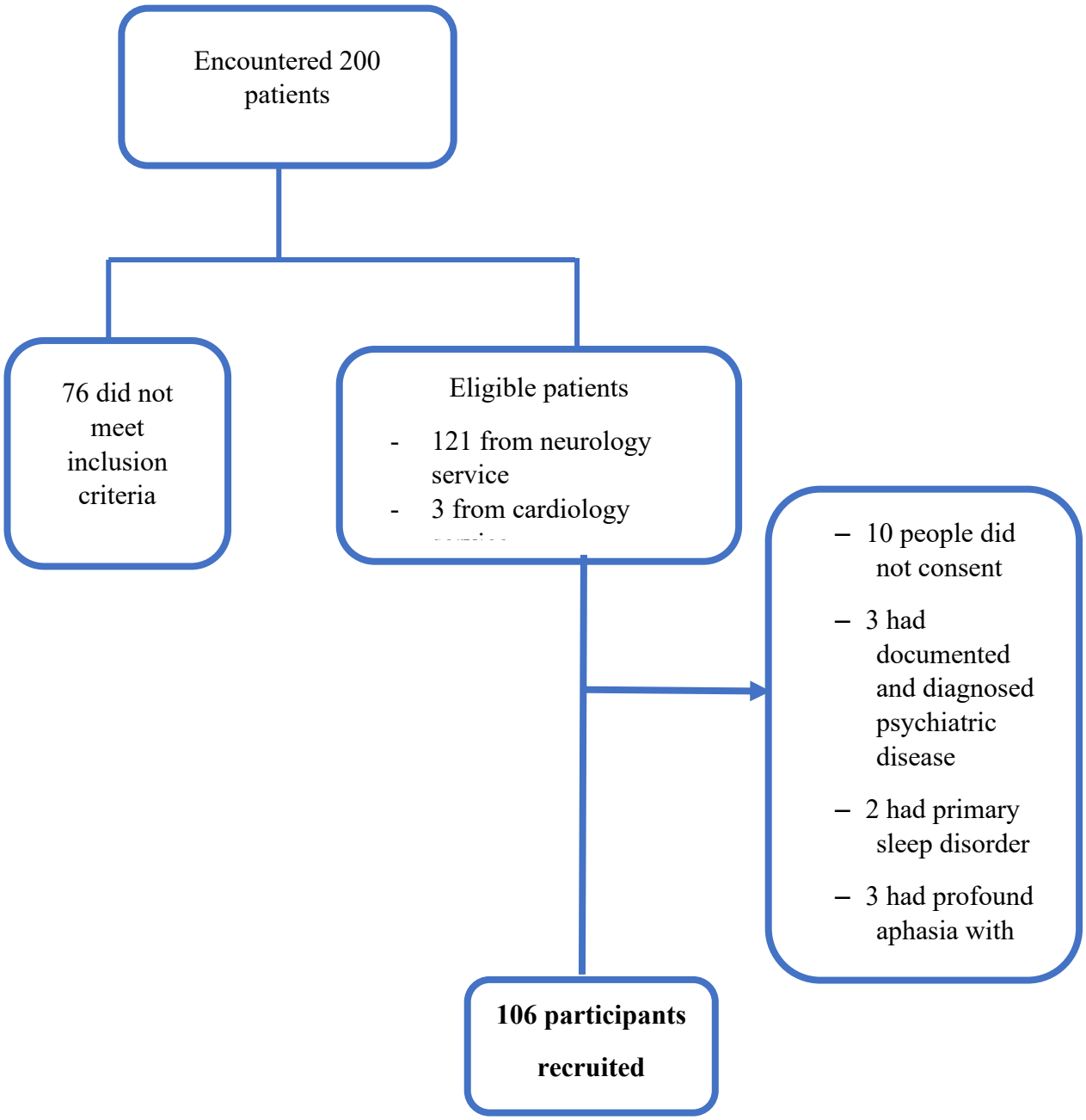
Ethical principles for medical research involving human subjects, as outlined in the World Medical Association Declaration of Helsinki, were upheld. Ethical clearance was acquired from the Ethical Committee of the Faculty of Medicine and Biomedical Sciences to carry out this study. Administrative authorization was also obtained from the Director of the Yaoundé Central Hospital. All selected participants were informed of the objectives of our study. For each participant recruited, we ensured anonymity and confidentiality. The informed consent of every person recruited was obtained. We carried out our study in strict compliance with the fundamental principles of medical research:

- **The principle of Beneficence**: According to information from existing data, poor sleep deters recovery outcomes in stroke patients; creating awareness of sleep disorders in stroke patients could lead to improved treatment protocols and monitoring.
- **The principle of non-maleficence**: No invasive procedures were performed. Patients who presented with sleep disorders were managed (medications) based on established evidence showing safety and efficacy by the neurologist.
- **Principle of autonomy**. Participants were provided with detailed information about the study's purpose, procedures, risks, and benefits. They were allowed to ask questions and informed that participation was entirely voluntary and that they could withdraw from the study without any negative consequence.
- **Confidentiality**: The participants' data were handled with strict confidentiality. All information was anonymized and kept solely by research personnel.
- **Justice**: the subject was free to participate or not in the study and did not suffer any prejudice in the event of refusal.

CHAPTER IV: RESULTS

ENROLLMENT FLOW CHART

Out of 200 patients assessed for eligibility, we excluded 94 individuals for the following reasons: 76 patients whose stroke duration did not fall within the two-week to six-month window, 10 who did not consent to participate, 3 with profound aphasia and no reliable informant, 2 with diagnosed psychiatric disorders, and 2 with diagnosed primary sleep disorders prior to their stroke event. We retained **106** participants whose data were included in our analysis.



IV.1. GENERAL CHARACTERISTICS OF THE STUDY POPULATION

IV.1.1. Socio Demographic profile of the study population

The ages of our participants ranged from 28 to 98 years, with a mean value of 60.89±13.08 years. The 50–85 years age group was the most represented. Most of our study participants were females (52.83%), with a female-to-male ratio of 1.12. Among the study participants, 42.5% had completed secondary education. Most of our study population were married (61.32%). As seen in **Table V.**

Table V: Sociodemographic profile of our study population

Sociodemographic variables	Frequency(N=106)	Percentage (%)
Age (years)		
≥50	86	81.13
<50	20	18.87
Sex		
Male	50	47.17
Female	56	52.83
Level of education		
None	15	14.15
Primary	31	29.25
Secondary	45	42.45
University	15	14.15
Marital status		
Married	65	61.32
Single	15	14.15
Divorced	4	3.77
Widowed	22	20.75

IV.1.2. CLINICAL PROFILES OF OUR STUDY PARTICIPANTS

IV.1.2.1. Risk factors and Comorbidities of the study population

The BMIs of our participants ranged from 18.20 kg/m^2 to 44.29 kg/m^2, with a mean value of 28.18±5.80 kg/m^2. Overall, 39.62% of our participants had a BMI of more than 30 kg/m^2. A recent smoking history was recorded in 9.43% of our participants, while 66.04% had a history of alcohol intake. Most of our study participants (94.34%) were hypertensive. Patients with diabetes mellitus accounted for 23.58% of our study population (**Table VI)**

Table VI: Risk factors and comorbidities of our study participants

Risk factors/comorbidities	Frequency	Percentage (%)
Alcohol consumption	70	66.04
Smoking	10	9.43
Obesity	42	39.62
Diabetes mellitus	25	23.58
Prior stroke	13	12.26
Cardiopathy	20	18.87
Hypertension	100	94.34
Epilepsy	3	2.83

IV.1.2.2. Clinical characteristics and paraclinical characteristics of stroke in the study population

The duration of stroke in our study population ranged from two weeks to six months, with a median duration of 28 [20–55] days. More than half of our study participants (51.89%) were seen less than a month after the onset of stroke symptoms.

Ischemic stroke (73.58%) was the most prevalent type of stroke in this study. The National Institute of Health Stroke Scale (NIHSS), which was used to measure stroke severity, had a median value of 3 [1–6]. The majority of our study population (45.28%) had a minor stroke.

In our study, 46.23% of participants had lesions in the left hemisphere. The majority of patients (56.60%) experienced a subcortical stroke.

The clinical characteristics of our study participants are presented in Tables VII and VIII.

Table VII: Clinical variables of stroke amongst our study population.

Clinical variables	Frequency	Percentage (%)
Duration of stroke symptoms		
<1 month	55	51.89
1–3 months	43	40.57
3–6 months	8	7.55
Stroke Severity (NIHSS)		
No stroke symptom (0)	19	17.92
Minor stroke (1–4)	48	45.28
Moderate stroke (5–15)	38	35.83
Moderately severe stroke (16–20)	1	0.94

Table VIII:CT-scan characteristics of our study population

Variables	Frequency	Percentage (%)
Type of stroke		
Ischemic	77	73.58
Hemorrhagic	28	26.42
Hemisphere		
Right	49	46.23
Left	48	45.28
Both	7	6.60
None	2	1.89
Stroke location		
Cortical	28	26.42
Subcortical	73	68.87
Brainstem	2	1.89
Cerebellum	1	0.94
No evident stroke lesion	2	1.89

IV.1.2.3. Functional status and psychological assessment characteristics of our study population

The Modified Rankin Scale (mRS) and Barthel index (BI) were used to assess functional disabilities of participants. The mRS ranged from 0 to 5, with a median value of 2 [1–5]. The Barthel index scores of the study participants ranged from 10 to 100, with a median value of 80 [40–95]. Among our participants 42.06% had poor functional outcome according to the mRS.

The anxiety HADs score of our participants ranged from 0 to 16, with a median value of 3[2–4], while the depression HADs score of our study participants ranged from 0 to 17, with a median value of 4 [1–8]. We recorded that 9.43% were cases of depression and 1.89% cases of anxiety (i.e., HADs score: ≥ 11), as presented in Table IX.

Table IX:: Psychological and functional assessment characteristics of our study population

Variables	Frequency	Percentage (%)
Degree of disability (Modified Rankin Scale)		
(mRS) > 2	52	49.06
(mRS) ≤ 2	54	50.94
Activities of Daily Living Assessment		
Barthel index ≥ 95	40	37.74
Barthel index < 95	66	62.26
HADS depression score		
Normal score (<8)	79	74.53
Borderline (8–10)	17	16.04
Presence of depression (≥ 11)	10	9.43
HADS anxiety score		
Normal score (<8)	101	95.28
Borderline (8–10)	3	2.83%
Psychological	2	1.89

Regarding medications, most of our study participants (94.34%) were taking antihypertensives. We recorded that 42.45% were using antidepressants, and 30.19% were on sedatives, as shown in Table **X.**

Table X: Medications consumed by our study population

Medications	Frequency	Percentage (%)
Antihypertensives	100	94.34
Statins	69	65.09
Antiplatelets agents	68	64.15
Anti-depressive medications	45	42.25
Sedatives(benzodiazepines)	32	30.19
Oral anti-diabetic	14	13.21
Insulin	6	5.66
Anti-epileptics	6	5.66
Oral anti-coagulants	3	2.83
Anti-vitamin k agents	2	1.89
Physiotherapy	58	54.72

IV.2. PREVALENCE OF SLEEP DISORDERS IN OUR STUDY POPULATION

The prevalence of sleep disorders in our study population was **52.83%.**

The most common sleep disorder in our study population was excessive daytime sleepiness (28.30%). Figure 8 below tells us more.

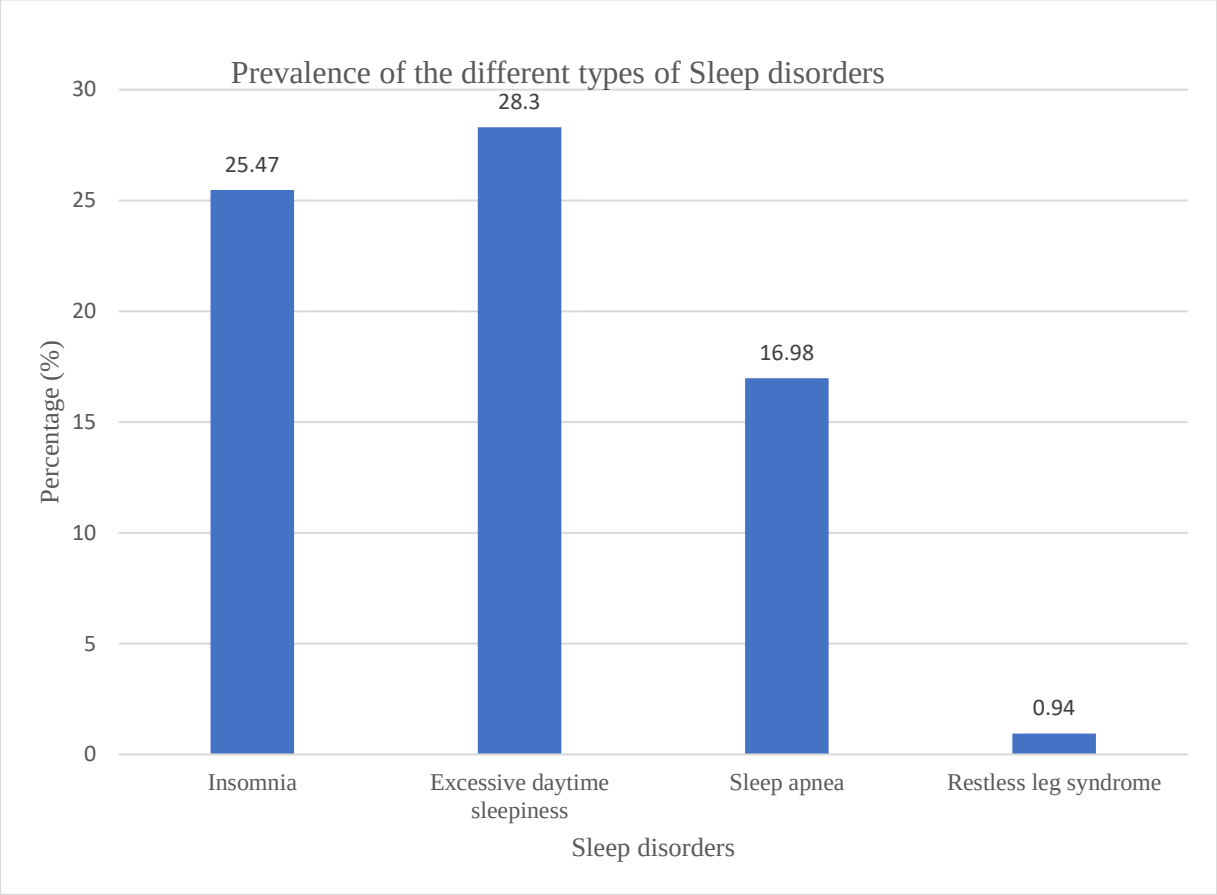


Figure 8: Prevalence of the different types of sleep disorders in our study (N= 106 participants)

IV.3. TYPES OF SLEEP DISORDERS IN OUR STUDY POPULATION

All of our participants reported no confirmed diagnosis of a sleep disorder prior to their stroke event. Out of the hundred and six participants we included, 30.19% had sleep problems reported by the primary care physician and were placed on medication (benzodiazepines). After the assessment of sleep disorders was done using standardized sleep scales, poor sleep quality, insomnia, excessive daytime sleepiness, high risk of obstructive sleep apnea, and restless leg syndrome were identified in the study population

IV.3.1. Insomnia

- **Prevalence of insomnia**

Insomnia, as diagnosed according to (DSM-5) criteria, was present in 27 patients (25.47%), with 12.26% presenting with symptoms less than a month after their stroke event. Tables **XI** and **XII** tell us more.

Table XI: Prevalence of insomnia in our study population

Insomnia	Frequency	Percentage (%)
Insomnia present	27	**25.47**
Insomnia absent	79	74.53

Table XII: Time of screening from stroke event

Duration of stroke symptoms	Frequency	Percentage (%)
<1 month	13	48.15
1-3 months	13	48.15
> 3months	1	3.70

Out of the 27(25.47%) patients with insomnia, 19 (76%) were not on sleep medications (benzodiazepines)

Also, out of the 30.19% of our participants who were placed on benzodiazepines by the physician for insomnia symptoms after their stroke event, 75% were on didn't present with insomnia at screening.

➢ **Types of Insomnia according to DSM-5 criteria in our study population**

Sleep maintenance insomnia was the most frequent type of insomnia in the study population

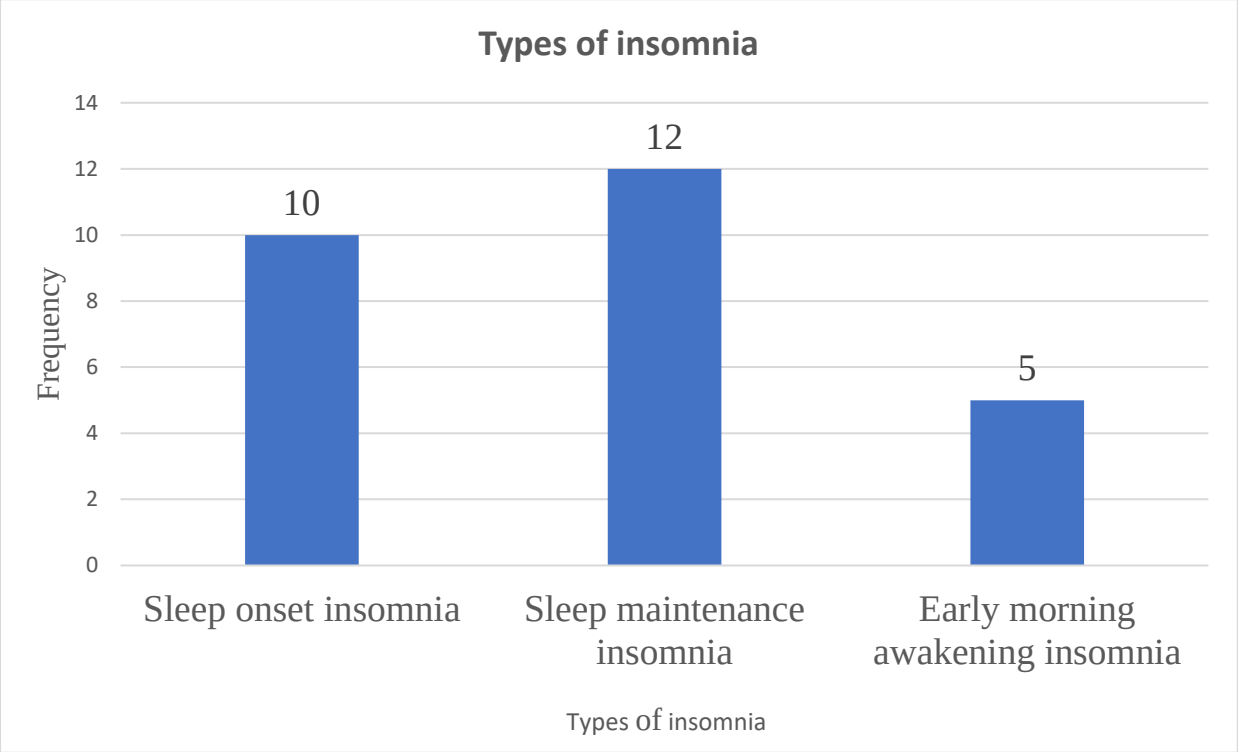


Figure 9: Types of Insomnia

IV.3.2. Obstructive Sleep Apnea Risk

The STOP-BANG questionnaire was used to screen for OSA suspicion. It ranged from 1 to 7 with a median value of 3[2-4]. Table XIII below shows how it classifies OSA.

Table XIII: Obstructive Sleep apnea risk distribution of our study population

Obstructive sleep apnea risk	Frequency	Percentage (%)
High Risk	18	**16.98**
Intermediate Risk	58	54.72
Low Risk	30	28.30
Total	106	100.00

IV.3.2. Excessive Daytime Sleepiness

The Epworth sleepiness scale (ESS) score which was used to measure excessive daytime sleepiness, ranged from 1 to 24, with a median ESS score of 8 [5–11]. Excessive daytime sleepiness (EDS) was present in 28.3% of our participants, as seen in Figure 10 below

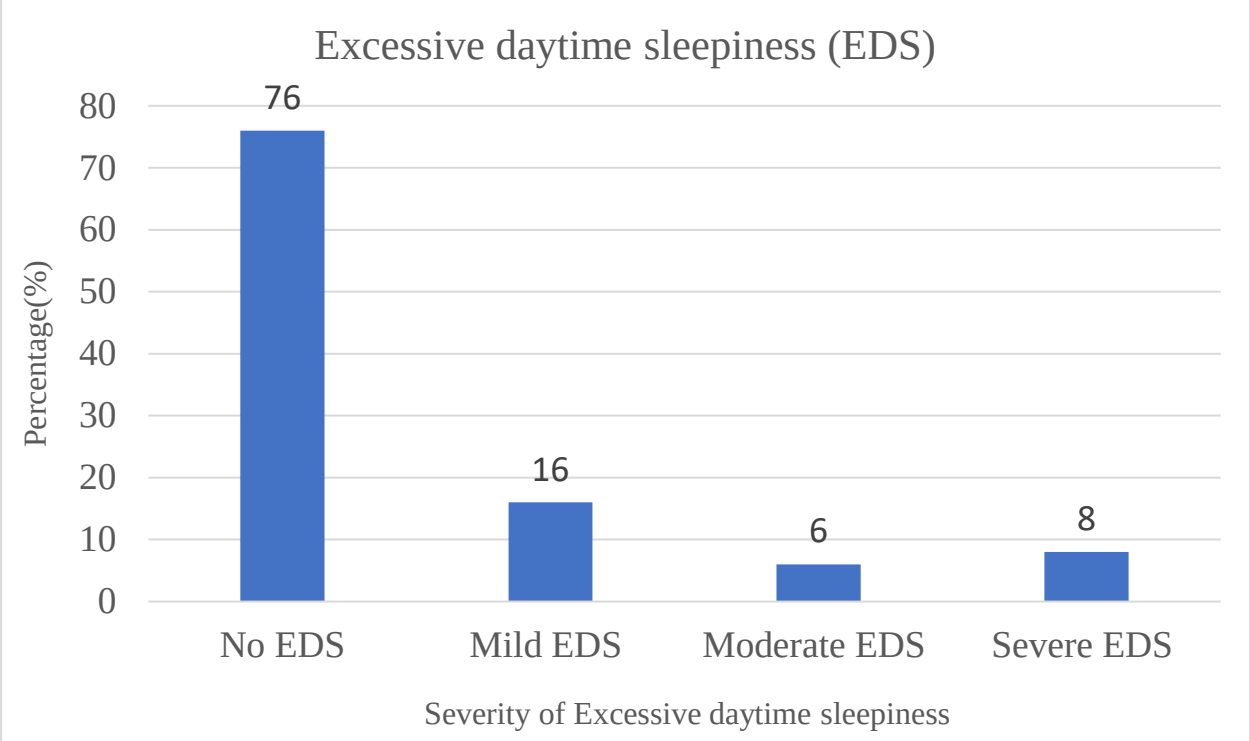


Figure 10: Excessive daytime sleepiness

IV.3.4. Restless leg syndrome

One of our participants (0.94%) reported signs of restless leg syndrome in accordance with the DSM-5 criteria.

IV.5.5. Sleep Quality

The Global PSQI score ranged from 0 to 16 with a median value of 6[4-8]. Most of our study participants (67.92%) had poor sleep quality; a global PSQI ≥ 5. The sleep quality of our study population is presented in Table **XIV**.

Table XIV: Sleep quality of our study population

Sleep Quality	Frequency	Percentage (%)
Good sleep quality (PSQI < 5)	34	32.08
Poor sleep quality PSQI ≥ 5	72	**67.92**

IV.4. FACTORS ASSOCIATED WITH THE OCCURRENCE OF SLEEP DISORDERS IN STROKE PATIENTS

Univariate analysis and multiple logistic regression were performed on the sociodemographic and clinical variables of our study population, with the presence of sleep disorders generally and with each of the several sleep disorders, that is Insomnia, Excessive Daytime Sleepiness, Sleep apnea risk, and poor sleep quality

1. Insomnia

There was no significant association between sociodemographic and clinical factors and the diagnosis of Insomnia in our study population as seen in Tables XV and XVI, below.

Table XV: Sociodemographic and comorbidities associated with insomnia from univariate analysis

Variables	Insomnia		OR [95%CI]	p-value
	Yes (%)	No (%)		
Age, years (>50/≤50)	24(27.91)	62(72.09)	2.19[0.59-8.17]	0.18
Sex (M/F)	10(20)	40(80)	0.57[0.23-1.41]	0.16
Married (yes/no)	14(21.54)	51(78.46)	0.59[0.24-1.43]	0.17
Cardiopathy	7(36.84)	12(63.16)	1.93[0.66-5.54]	0.17
Duration of stroke diagnosis				
Less than 1 month	13(23.64)	42(76.36)	0.82[0.34-1.96]	0.41
Between 1-3 months	13(30.23)	30(69.77)	1.51[0.63–3.66]	0.24
Between 3–6 months	1(12.50)	7(87.50)	0.40[0.05–3.38]	0.35
Stroke location				
Cortical stroke	6(21.43)	22(78.57)	0.74[0.26–2.08]	0.38
Subcortical stroke	17(28.33)	43(71.67	1.42[0.58–3.49]	0.29
Both cortical and subcortical stroke	3(23.08)	10(76.92)	0.86[0.22–3.40]	0.57
mRS score (>2/≤2)	14(26.92)	38(73.08)	1.16[0.48–2.79]	0.45
Barthel's index (≥95/<95)	10(25)	30(75)	0.96[0.39–2.37]	0.56

Table XVI: Multivariate analysis of factors associated with insomnia

Variables	Adjusted Od[95%CI]	p-value
Age, years (≥50)	2.26[0.59–8.71]	0.24
Sex, (male)	0.66[0.23–1.88]	0.43
Married	0.72[0.25–2.09]	0.54
Cardiopathy	1.83[0.62–5.41]	0.27

2. Excessive daytime sleepiness

There was a significant association between age, moderate stroke, poor functional status (mRs > 2), a Barthel index score and excessive daytime sleepiness. Diabetes was found to be independently associated to EDS on multivariate analysis. Tables XVII, XVIII, presents these factors

Table XVII: Sociodemographic and clinical factors associated with EDS in study population on univariate analysis

Variables	EDS		OR [95%CI]	p-value
	Yes (%)	No (%)		
Age, years (>50/≤50)	28(32.56)	58(67.44)	4.34[0.94–20.04]	**0.03**
Sex(M/F)	18(36)	32(64)	2.06[0.87–4.88]	0.07
Obesity	11(26.19)	31(73.81)	0.84[0.35–2.01]	0.44
Diabetes	4(16)	21(84)	0.40[0.12–1.29]	0.09
Stroke severity				
No stroke symptoms	3(15.79)	16(84.21)	0.42[0.11–1.55]	0.15
Minor stroke symptoms	10(20.83)	38(79.17)	0.5[0.21–1.21]	0.09
Moderate stroke	16(42.11)	22(57.89)	2.81[1.17–6.71]	**0.02**
Sedative (benzodiazepines)	10(31.25)	22(68.75)	1.23[0.50–3.04]	0.41
Anticonvulsants	2(33.33)	4(66.67)	1.29[0.22–7.42]	0.55
Depression	7(70)	3(30)	7.41[1.77–30.99]	**0.005**
mRS scores (>2/mRS≤2)	21(40.38)	31(59.62)	3.39[1.37–8.37]	**0.006**
Barthel index (≥95/<95)	6(15)	34(85)	0.31[0.11–0.84]	**0.01**

Table XVIII:multivariate analysis of factors associated with EDS in our study population

Variables	Adjusted Odds Ratio[95%CI]	p-Value
Age, years (>50)	5.36[1.00–28.76]	0.05
Sex (male)	1.81[0.65–5.05]	0.26
Smoking	4.92[0.51–47.34]	0.17
Diabetes	**4.65[1.04–20.69]**	**0.04**
Antidepressants	2.67[0.96–7.40]	0.06
Depression	4.3765[0.74–26.00]	0.10
mRS score (>2/≤2)	2.54[0.41–15.86]	0.32
Barthel Index (≥95/<94)	0.98[0.18–5.22]	0.98

3. Obstructive Sleep Apnea Risk

On univariate analysis, sex, mRs scores and Barthel index score were significantly associated to high risk for OSA. The independent factors identified following multivariate analysis were male sex, obesity, prior stroke and poor functional outcome (mRs) scores. These factors are presented in tables XIX and XX

Table XIX: Factors associated with the high risk for OSA on univariate analysis

Variables	STOP-BANG score ≥ 5		OR [95%CI]	p-value
	Yes (%)	No (%)		
Age, years (>50/≤50)	17(19.77)	69(80.23)	4.68[0.59–37.46]	0.1
Sex (M/F)	14(28.00)	36(72)	5.06[1.54–16.61]	**0.004**
Alcohol consumption	15(21.43)	55(78.57)	3[0.81–11.15]	0.07
Obese	10(23.81)	32(76.19)	2.19[0.78–6.10]	0.11
Cardiopathy	6(31.58)	13(68.42)	2.85[0.91–8.93]	0.07
Prior stroke(yes/no)	4(30.77)	9(69.23)	2.51[0.68–9.27]	0.15
mRS score (>2/≤2)	14(26.92)	38(73.08)	4.6[1.40-15.12]	**0.01**
Barthel index score (≥95/<95)	3(7.50)	37(92.50)	0.28[0.07-1.02]	**0.04=**

Table XX: Multivariate analysis of factors associated with high for Obstructive Sleep Apnea

Variables	Adjusted Odds Ratio[95%CI]	p–Value
Age, years (≥50)	9.27[0.42–206.29]	0.16
Sex, (male)	**15.42[2.82–84.35]**	**0.002**
Cardiopathy	2.49[0.52–12.01]	0.26
Prior Stroke(No/yes)	**0.10[0.02–0.69]**	**0.02**
mRS score (>2)	**16.96[1.54–186.59]**	**0.02**
Barthel index (≥95)	1.02[0.10–10.43]	0.99
Alcohol consumption	0.26[0.04–1.55]	0.14
Obesity	**6.42[1.39–29.62]**	**0.02**

4. Sleep quality

On univariate analysis, there were associations between age, sex, depression, and mRS score > 2 with poor sleep quality in our study population. Conversely, not all of them were statistically significant. The only factor found to be significantly associated with poor sleep quality (PSQI ≥ 5) from univariate analysis was the use of sedatives. The factors independently associated with poor sleep quality on multivariate analysis were older age and the use of sedatives. These factors are present in Tables XXI and XXII below

Table XXI: Factors associated with poor sleep quality (PSQI ≥5) on univariate analysis

Variables	Poor sleep quality		OR [95%CI]	p-value
	Yes (%)	No (%)		
Age, years (>50/≤50)	62(72.09)	24(27.09)	2.58[0.10–7.0]	**0.05**
Sex (male)	38(76)	12(24)	2.05[0.88–4.76]	0.07
Hemisphere				
Left hemisphere	31(64.58)	17(35.42)	0.76[0.33–1.71]	0.32
Right hemisphere	37(77.08)	11(22.92)	2.21[0.94–5.19]	**0.05**
Both hemispheres	4(57.14)	3(42.86)	0.61[0.13–2.88]	0.4
Antidepressants	28(62.22)	17(37.78)	0.64[0.28–1.45]	0.19
Sedative use	27(84.38)	5(15.63)	3.48[1.21–10.07]	**0.01**
Depression	9(90)	1(10)	4.71[0.57–38.83]	0.11
mRS score (>2/≤2)	39(75)	13(25)	1.91[0.83-4.39]	0.09

Table XXII: Multivariate analysis of factors associated with poor sleep quality.

Variables	Adjusted Odds Ratio[95%CI]	p-value
Age, years (≥50/<50)	**4.01[1.06–15.23]**	**0.04**
Sex (M/F)	1.76[0.65–4.79]	0.27
Right hemisphere	2.66[0.95–7.46]	0.06
Cortical And Subcortical stroke	0.33[0.07–1.54]	0.16
Antidepressants use	0.47[0.18–1.26]	0.12
Sedatives use	**7.47[1.87–29.83]**	**0.004**
Depression	4.21[0.41–43.71]	0.23
mRS score (>2 / ≤ 2)	1[0.25–4.10]	0.10
Barthel index (≥95 / <95)	0.87[0.20–3.81]	0.87
Obesity	1.02[0.37–2.84]	0.97

5. Sleep Disorders

Age, mRs scores and Barthel index score had a significant association with the presence of at least one of any of the sleep disorders on univariate analysis. The factors independently associated with sleep disorders in stroke patients were older age and poor functional outcome (mRS scores >2). (Tables XXIII and XXIV)

Table XXIII: Factors associated with sleep disorders on univariate analysis

Variables	**Sleep disorder**		**OR [95%CI]**	**p-value**
	Yes (%)	**No (%)**		
Age (years)	50(58.14)	36(41.86)	3.24[1.14–9.24]	**0.02**
Sex	31(62)	19(38)	2.02[0.93–4.44]	0.06
Stroke severity				
No stroke symptom	8(42.11)	11(57.89)	0.59[0.22–1.61]	0.22
Minor stroke	21(43.75)	27(56.25)	0.51[0.24–1.11]	0.07
Moderate stroke	26(68.42)	12(31.58)	2.74[1.19–6.32	**0.01**
Antidepressant use	25(55.56)	20(44.44)	1.21[0.56–2.62]	0.39
Sedative use	18(56.25)	14(43.75)	1.22[0.53–2.80]	0.4
Depression	7(70)	3(30)	2.24[0.55–9.17]	0.21
mRs (>2/≤2)	36(69.23)	16(30.77)	3.83[1.71–8.58]	**0.001**
Barthel index (≥95/<95)	16(40)	24(60)	0.43[0.19–0.97]	**0.03**

Table XXIV: Multivariate logistic regression analysis for factors independently associated with sleep disorders.

Variable	Odds Ratio	P-Value
Age>50	**3.76[1.13-12.55]**	**0.03**
Barthel index≥95	1.26[0.31-5.17]	0.75
Sex(male)	2.11[0.87-5.14]	0.10
Left hemisphere (True/False)	0.401[0.08-2.20]	0.30
Right hemisphere	0.75[0.14-4.03]	0.74
mRS (>2)	**5.36[1.07-26.73]**	**0.04**
Moderate stroke	0.55[0.08-3.56]	0.53
Minor stroke	0.49[0.13-1.86]	0.30
Type of stroke (Hemorrhagic stroke)	0.72[0.26-2.03]	0.53

IV.5. IMPACT OF SLEEP DISORDER ON FUNCTIONAL STATUS

The functional status of our participants was evaluated with the mRS score and Barthel index. An mRS score of > 2 indicates poor functional outcome. Upon multivariate analysis, worse mRs scores were significantly associated with sleep disorders in general with a **p-value of 0.04** and precisely associated to high risk of having obstructive sleep apnea with a **p-value of 0.02.** This is presented in Table XXIV and Table XX respectively.

CHAPTER V : DISCUSSION

There is an increasing interest in sleep-related studies in stroke patients. This is owing to the high prevalence of sleep disorders recorded and the negative impact of poor sleep on rehabilitation. The worldwide increase in the number of stroke cases has led to a higher number of individuals facing disabilities[2], resulting in a significant economic burden, as documented in Cameroon[20, 21]. Therefore, it is crucial to address factors that adversely affect the functional status of stroke patients. In this study, we aimed to describe the epidemiological and clinical aspects of sleep disorders in stroke patients at least two weeks after the stroke event but no more than six months after the event at the Yaoundé Central Hospital. Specifically, we aimed to determine the prevalence of sleep disorders in stroke patients, identify the different types of sleep disorders in this population, pinpoint factors associated with sleep disorders in stroke patients, and evaluate the influence of sleep disorders on patients' functional status.

V.1. Sociodemographic data

The mean age of our study participants was 60.89±13.08 years. This was similar to the mean age (61.4±11 years) reported in Nigeria by Iwuozo et al. in 2023 [13], who studied the prevalence of sleep disturbances and associated factors among stroke survivors. This similarity could be explained by the fact that the incidence of stroke doubles every 10 years after the age of 55 years [113].

In our study, we observed a female-to-male ratio of 1.12. Our findings are consistent with a study by Nutakki et al. in Zambia [114], who studied the sex-specific differences in stroke epidemiology and found a higher prevalence of stroke among female patients. Although males are generally known to be at a higher risk for stroke, one possible reason for females being at a greater risk could be their longer life expectancy compared to males [113, 115, 116]. In addition, most of the women in our study were obese, and obesity has been reported to increase the risk of stroke [117].

V.2. Prevalence of sleep disorders in stroke patients

We found the prevalence of sleep disorders in stroke patients to be 52.83%. Our findings are similar to the 61.6% prevalence of sleep disturbances in stroke patients, reported in a study in Canada by Jeffers et al. in 2023 [118]. These findings are consistent with the recorded range of sleep disorders from various studies, which range from 20% to 78% [11, 13–15]. Stroke lesions have been found to disrupt neural pathways that lead to disruption in sleep regulatory areas in the brain. For example, injury to the brainstem or thalamus might affect the reticular activating system, which is essential for maintaining alertness. Furthermore, strokes affecting

the hypothalamus can change the release of neurotransmitters and chemicals that govern sleep, such as melatonin and orexin[119]. In addition, there is a robust inflammatory process that results in an attempt to repair and limit damage. Inflammatory molecules like IL-1b and TNF-a affect the balance of circadian rhythm and neurotransmitters involved in sleep regulation. These molecules have been TNF-a increases NREM sleep at the expense of REM sleep, which can cause daytime sleepiness. Furthermore, TNF-a can affect CLOCK-BMAL1 activation, which suggests that circadian rhythms are altered [120]. Furthermore, a study in Nigeria by Iwuozo et al. in 2023 had a prevalence of 33.6% of sleep disturbances in stroke patients. A possible explanation to this could be that we used more screening instruments to evaluate for sleep disorders in our study population as compared to them. The high prevalence obtained in our study underscores the significant burden of sleep disorders in stroke patients. Sleep disorders alter and fragment sleep [121], thereby hindering neuroplasticity and recovery, impeding rehabilitation efforts, and prolonging hospital stays. These adverse effects highlight the paramount importance of early identification and intervention.

Excessive Daytime Sleepiness had a prevalence of 28.3%. This prevalence was similar to that recorded by Iwuozo et al. of 20.9 % [12]. These results are consistent with the prevalence documented in many studies, which range from 1.1 to 27 % [17]. A variety of mechanisms are attributed to the occurrence of hypersomnia, also referred to as excessive daytime sleepiness, in stroke patients. A possible explanation for this is the high prevalence of hypertension found in both studies. EDS has been found to be frequent in hypertensive patients by Mbatchou et al. in Cameroon [17]. Hypertension causes increased sympathetic nervous system activity, which can lead to disturbances in sleep architecture, including reduced slow-wave sleep and increased nocturnal awakenings, consequentially increasing daytime sleepiness [122]. Hypersomnia has been associated with high chances of nursing home visits and poor functional outcomes. These could be explained by the reduced participation in therapy and behavioral changes leading to increased dependencies in patients brought about by excessive sleep [123]. This highlights the importance of identifying and managing this condition in stroke patients.

Insomnia had a prevalence of 25.47%, which was similar to Tayade et al. in 2023 in India [124], who found a prevalence of 20% of people with insomnia. This could be due to similarities in study design and sample sizes. This high prevalence could be explained by the fact insomnia has been connected to disruptions to subcortical areas like the paramedian

thalamus and brain where sleep spindles are generated. The majority of our participants had lesions in the subcortex (56.66%). Also, studies have reported insomnia to be linked to other factors like unfamiliar environments and being hospitalized [119]. It is important to note that post-stroke insomnia can exacerbate anxiety, impact daytime energy, concentration, and memory, and ultimately hinder rehabilitation and performance [18].

The prevalence of high-risk for obstructive sleep apnea in our study population was 16.98%. This was similar to the findings in Ghana by Sarfo et al. in 2017 [125], where 18.5% of stroke survivors had a high risk for obstructive sleep apnea. This could be explained by the similarities in the study setting and instruments used. Also, a possible explanation for this is that our patients were obese and that obesity, being a coexisting risk factor for stroke, is also a significant risk factor for OSA. Obesity can cause airway obstruction by increased fat deposition around the muscles of the neck. The prevalence of OSA has been shown to differ across studies due to differences in screening instruments. Sleep apnea has been attributed to an increased risk of stroke reoccurrence. Hence, early detection and management is needed.

We found a prevalence of 0.94% for restless leg syndrome, which differs from the 2.7% reported by Iwuozo et al. in 2023. This difference could be attributed to the fact that we used the PSQI and DSM-5 criteria for screening, which were not utilized by Iwuozo et al. According to Schlesinger et al. in Israel, RLS was significantly more prevalent in acute stroke/TIA patients compared to the control (non-stroke patients) [126]. Hence, there is more need for screening among stroke patients. The prevalence of stroke-related RLS has been linked to geographical location, with a prevalence between 1% and 3% in most Asian populations and a prevalence of 5% to 13% in North America and Europe [127].

We found that the prevalence of poor sleep quality (Global PSQI score > 5) in our study population was 67.92%. In a study carried out in Egypt by Mansour et al. in 2020, 61.3% had poor sleep [15]. Our results are also similar to those of a global meta-analysis, which reported a pooled prevalence of 66% of poor sleep quality among stroke patients. This study also reports that all sleep disorders are associated with poor sleep quality, which lowers patients' quality of life and impedes stroke recovery[128].

V.3. Associated factors

In our study, univariate analysis revealed a significant association between depression and ESS score and PSQI score, with 9.43% of our cohorts being depressed. This is different from that obtained by Iwuozo et al. in Nigeria on univariate analysis, which was 21.8%, and could be because a good number of our study participants were on anti-depressants. However, our prevalence falls within the range reported by previous studies which have estimated the prevalence of sleep disorders to range between 5% to 63%, with a global projected prevalence of around 33% [129]. Depression after stroke is common, as most of the survivors suffer from emotional trauma related to post-stroke outcomes like physical independence[130].

Age and gender were found to be significant factors associated with sleep disorders in our study population. This result is consistent with that of Iwuozo et al. in Nigeria and also with a meta-analysis by Hasan et al. in 2021. These studies significantly revealed that age and gender influenced the prevalence of sleep disorders in stroke patients. A possible explanation for this could be that increasing age has been connected with greater fragmented sleep, in which the patient's sleep is interrupted numerous times during the night, resulting in excessive daytime weariness and sleepiness [131].On the other hand, Iddagoda et al. in Australia found no significant link between sleep disorders in stroke survivors and demographics such as age or gender [132].

We had a significant association between the male sex and high-risk of Obstructive Sleep Apnea. Our study is similar to that of Dharmakulaseelan et al. in 2023, who reported male sex as an independent predictor of increasing Obstructive Sleep Apnea severity, but this association doesn’t mean that sex is an independent predictor of specific symptoms of OSA such as daytime sleepiness, snoring, tiredness, and observed apneas [133]. The association of the male sex to OSA could be due to the link between the male sex and OSA as they are both cardiovascular risk factors of stroke.

In our study, we observed that diabetes was an independent predisposing factor for excessive daytime sleepiness (EDS) in our study population after carrying out a multivariate analysis. Our findings are consistent with those obtained by Šiarnik et al. in 2018 in Slovakia [134], where diabetes mellitus was significantly associated with measures of daytime sleepiness. This similarity in results could be attributed to similar study designs and instruments. Furthermore, the symptoms of diabetes, such as nocturia, polyuria, and diabetic neuropathy, may disrupt night sleep and contribute to excessive daytime sleepiness [135]. Additionally, diabetes has

been linked to various chronic conditions, including obstructive sleep apnea, cardiovascular problems, and hypertension, which have all been shown by studies to negatively impact sleep [17, 119]. It is possible that patients may not raise their sleep concerns during healthcare appointments due to more immediate health issues taking precedence. Hence, sleep education is a needed part of management in these patient groups.

Obesity and prior stroke are factors found to be independently associated on multivariate analysis with a high risk of Obstructive Sleep Apnea risk. Obesity being associated with a high risk of OSA is consistent with the literature. Obesity causes narrowing of the upper respiratory airways due to fatty tissues, this leads to recurrent episodes of apnea/hypopnea due to either a collapse or partial collapse of the airway. This hypoxia then turns to create overproduction of reactive oxygen species from oxidative stress. These reactive then cause endothelial wall damage resulting in atherosclerosis. Hence OSA in stroke patients is vital in preventing future vascular diseases which may be a recurrent stroke. On the other hand, Sarfo et al.in 2017 [125] did not have obesity as an associated factor of OSA in stroke patients, which could be explained by the low prevalence of obese patients in their study sample as compared to ours. Prior stroke was found to be associated with high-risk OSA. This is consistent with the literature, which demonstrates OSA as a risk factor for having more strokes. Obstructive sleep apnea results in atherosclerosis, as explained above, and can predispose patients to stroke recurrence. In our study population, patients who had a prior stroke scored high on the STOP-BANG score hence the need for frequent screening of OSA in our study population.

Poor sleep quality was found to be associated with the use of hypnotics. About 30.19% of our participants were placed on sleep medications after complaining of insomnia symptoms in the very acute phase of stroke and they were still on sedatives at the time of screening. Taking sleep medications is part of the PSQI scale score hence the reason for the strong association to hypnotics. A study carried out in Cameroon by Anita et al. in 2018 reported the consumption of hypnotic medications to be associated with poor sleep quality among medical students [136]

V.4. Impact of Sleep disorders on the functional status of our study population

Worse modified ranking score (mRs) outcomes were recorded in our study to be associated with sleep disorders and, specifically, a high risk of obstructive sleep apnea. Sleep has been shown to be important for neuroplasticity and memory consolidation, which are key aspects needed for brain recovery and relearning lost functional abilities [121]

Sarfo et al. in Ghana reported that patients who were at high risk of OSA were less able to perform activities of daily living [125] which was similar to ours. Also, a cross-sectional study by Tayade et al. in 2023 equally found worse mRS outcomes associated with sleep disorders[124].

Poststroke functional status is one of the main predictors of functional outcomes after rehabilitation, it is believed that impaired sleep disorders may adversely affect the success of rehabilitation programs by worsening functional status [19, 106, 132].

Study Limitations

There are some limitations to our study. Our study used questionnaires to screen for sleep disorders. The diagnosis of sleep disorders such as obstructive sleep apnea and restless legs syndrome was not confirmed using overnight polysomnography due to its high cost, which made this limitation insurmountable. However, the questionnaires that have been proven valid are easy to use, less costly, and more readily available for screening, making them a fair substitute. Additionally, our study was a cross-sectional study, which limits its ability to determine cause and effect in the observed associations. Nonetheless, our study's insights provide a foundation on which longitudinal studies can build.

CONCLUSIONS

In summary, at the end of our study on sleep disorders among stroke survivors at the Yaoundé Central Hospital, we can conclude that:

1. The prevalence of sleep disorders was 52.83%, with the most common being excessive daytime sleepiness (28.3%).
2. The sleep disorders reported in our study were excessive daytime sleepiness, insomnia, obstructive sleep apnea risk, and restless leg syndrome. We also found that a majority of our patients had poor sleep quality.
3. The factors associated with sleep disorders in stroke survivors are age, sex, obesity, diabetes, prior stroke, and use of sedatives.
4. Sleep disorders and poor sleep quality are associated with poor functional status, which might further impair rehabilitation outcomes.

RECOMMENDATIONS

To researchers

- To carry out longitudinal studies in order to establish causality-effect between the factors found to be associated with sleep disorders in our study.

To the Ministry of Public Health

- To raise awareness among physiotherapists about the diagnosis of sleep disorders and the importance of directing patients to the appropriate services, through talks and scientific meetings.

To health care professionals attending to patients with

- To routinely investigate symptoms of sleep disorders in stroke patients during follow-up visits at outpatient consultations.
- To encourage the use of sleep questionnaires, which are easy are less expensive to screen for and confirm sleep disorders in stroke patients.
- To educate patients on symptoms of sleep disorders and on the importance of sleep on their recovery and overall health.

To patients and the general public

- To frequently self-evaluate their sleep and promptly seek medical attention when faced with symptoms of poor sleep.

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APPENDICES

APPENDIX 1A: INFORMATION SHEET

- **Topic:** Sleep disorders among stroke survivors at the Yaoundé Central Hospital: Prevalence, Associated Factors and Impact on Functional Status.
- **Principal investigator**: MBANGE LIKOWO GERMAINE, a final year general medicine student at the Faculty of Medicine and Biomedical Sciences, University of Yaoundé I.

Telephone number: 671969415; Email address: mbangegermaine@gmail.com.

- **Supervisors:** PROFESSOR ALFRED K. NJAMNSHI, Professor of Neurology and Clinical Neurophysiology, and DOCTOR LEONARD NGARKA, Senior Lecturer in Neurology.
- **Study objectives:** Our study aims to describe the epidemiological and clinical aspects of sleep disorders among stroke patients.
- **Study period:** November 13, 2023, to May 16th, 2024.
- **Study Risk:** Our study does not present any risk to our patients.
- **Benefits of the study:** The study will help raise awareness among neurologists and health professionals on the occurrence of sleep disorders among stroke survivors which will aid in better management and follow-up of stroke patients.
- **Study procedure:** The study entails first obtaining your consent to participate in the study, after which you will be given a paper with questions to answer, or you will be verbally asked the questions and expected to answer verbally. Thereafter, certain information will be obtained from your medical booklet concerning your stroke episode. After which we will conduct a physical examination on you.
- **Ethical considerations and confidentiality**: All data obtained from the study will be used for the sole purpose of research and will be treated with utmost confidentiality. The questionnaires are anonymous in order to respect patients' private information. You can refuse to participate in this study or opt-out at any time. Also, in order to carry out this study, ethical clearance has been obtained from the Ethical Committee of the Faculty of Medicine and Biomedical Sciences, Univerity of Yaounde I, and authorization from the administration of the Yaoundé Central Hospital.

APPENDIX 1B: FICHE D'INFORMATION

• **Sujet** : Troubles du sommeil chez les survivants d'un AVC à l'Hôpital Central de Yaoundé : Prévalence, facteurs associés et impact sur le statut fonctionnel.

• **Investigateur principal** : MBANGE LIKOWO GERMAINE, étudiante en dernière année de médecine générale à la Faculté de Médecine et des Sciences Biomédicales, Université de Yaoundé I.

Numéro de téléphone : 671969415 ; Adresse e-mail : mbangegermaine@gmail.com.

• **Superviseurs** : PROFESSEUR ALFRED K. NJAMNSHI, Professeur de Neurologie et de Neurophysiologie Clinique, et DOCTEUR LEONARD NGARKA, Maître Assistant en Neurologie.

• **Objectifs de l'étude** : Notre étude vise à décrire les aspects épidémiologiques et cliniques des troubles du sommeil chez les patients victimes d'un AVC.

• **Période de l'étude** : Du 13 novembre 2023 au 16 mai 2024.

• **Risque de l'étude** : Notre étude ne présente aucun risque pour nos patients.

• **Bénéfices de l'étude** : L'étude contribuera à sensibiliser les neurologues et les professionnels de santé à la survenue de troubles du sommeil chez les survivants d'un AVC, ce qui aidera à une meilleure prise en charge et un meilleur suivi des patients victimes d'un AVC.

• **Procédure de l'étude** : L'étude consiste d'abord à obtenir votre consentement pour participer à l'étude, après qu'un questionnaire vous sera remis à remplir, ou les questions vous seront posées verbalement et vous devrez y répondre verbalement. Par la suite, certaines informations seront obtenues de votre carnet médical concernant votre épisode d'AVC. Ensuite, nous procéderons à un examen physique.

• **Considérations éthiques et confidentialité** : Toutes les données obtenues dans le cadre de l'étude seront utilisées uniquement à des fins de recherche et seront traitées avec la plus grande confidentialité. Les questionnaires sont anonymes afin de respecter la confidentialité des informations des patients. Vous pouvez refuser de participer à cette étude ou vous retirer à tout moment. Afin de mener à bien cette étude, une autorisation éthique a été obtenue auprès du Comité d'éthique de la Faculté de Médecine et des Sciences Biomédicales de l'Université de Yaoundé I, ainsi qu'une autorisation de l'administration de l'Hôpital Central de Yaoundé."

APPENDIX 2A: PARTICIPATION CONSENT FORM

Titled: "Sleep disorders among stroke survivors at the Yaoundé Central Hospital: Prevalence, Associated Factors and Impact on Functional Status"

I,

undersigned……

Declare to have been informed and fully briefed by the Final Year General Medicine Student, MBANGE LIKOWO GERMAINE, and have accepted to participate in the study in view of her M.D thesis. She clearly stated I was free to accept or deny the proposal. I have received and understood the information on the aim of the study, the possible constraints, and the risks.

I accept that all data collected about 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 at any time if the principal investigator sees it necessary or if I wish, without any prejudice. At any time, I can ask for supplementary information or make any corrections to my data

I hereby accept to participate in the study under the set conditions.

Read and approved: YES NO

At Yaoundé, Date……. /……. /……..

Investigator's signature Volunteer's signature

APPENDIX 2B : FICHE DE CONSENTEMENT ECLAIRÉE

Je soussignée, ..……...……

Avoir été invité à participer au travail de recherche intitulé “Troubles **du sommeil chez les patients victimes d'un accident vasculaire cérébral à l’Hôpital Central de Yaoundé: prévalence, facteurs associés et impact sur l’état fonctionnel.**” dont l’investigateur principal s’appelle MBANGE LIKOWO GERMAINE, étudiante en fin d’année de Médecine Générale à la Faculté de Médecine et des Sciences Biomédicales de l’Université de Yaoundé 1(FMSB-UY1).

J’ai bien compris la notice information qui m’a été remise concernant cette étude. J’ai bien compris le but et les objectifs de cette étude. J’ai reçu toutes les réponses aux questions que j’ai posées. Les risques et bénéfices m’ont été présentés et expliqués. J’ai bien compris que je suis libre d’accepter ou de refuser d’y participer. Mon consentement ne décharge pas les investigateurs de la recherche de leurs responsabilités, je conserve tous mes droits garantis par la loi. J’accepte librement de participer à cette étude dans les conditions précisées dans la notice de l’information, c’est-à-dire répondre aux questions de l’enquête.

Je donne mon accord pour que les données collectées pour cette étude soient utilisées dans les études ultérieures.

Fait le ……/……/……

APPENDIX 3: QUESTIONNAIRE

Subject: Sleep disorders among stroke survivors at the Yaoundé Central Hospital: prevalence, associated factors and their impact on functional outcomes of stroke.

INTERVIEWER: MBANGE LIKOWO GERMAINE

PATIENT CODE: _____

Date: _____

SECTION A: SOCIODEMOGRAPHIC AND CLINICAL DATA

1. Sociodemographic and clinical data

Q/N0	VARIABLE	POSSIBLE ANSWERS	ANSWER	EXACT ANSWER
1	Date of birth			----------
2	Sex	1 - Male 2- Feminine	1. ◯ 2. ◯	
3	Address			----------
4	Level of education	1 – Not in school 2 - Primary 3 - Secondary 4 – University	1. ◯ 2. ◯ 3. ◯ 4. ◯	
5	Marital status	1- Married 2- Single 3- Divorced 4- Widow / widower	1. ◯ 2. ◯ 3. ◯ 4. ◯	
6	Occupation	1- Student 2- Unemployed 3- Civil servant 4- Private sector 5- Business 6- Household 7- Farmer 8- Retirees	1. ◯ 2. ◯ 3. ◯ 4. ◯ 5. ◯ 6. ◯ 7. ◯ 8. ◯	
7	Religion	1- Christian 2- Muslim 3- Atheist 4- Other 5- None	1. ◯ 2. ◯ 3. ◯ 4. ◯ 5. ◯	

2. HISTORY

Q/N0	VARIABLE	POSSIBLE ANSWERS	ANSWER	EXACT ANSWER
1	Hypertension	1- Yes 2- No	1. ◯ 2. ◯ 3. ◯	
2	Diabetes	1- Yes 2- No 3- Unknown	1. ◯ 2. ◯ 3. ◯	
3	Dyslipidemia	1- Yes 2- No 3- Unknown	1. ◯ 2. ◯ 3. ◯	
4	Sickle cell disease	1- Yes 2- No 3- Unknown	1. ◯ 2. ◯ 3. ◯	
5	Epilepsy	1- Yes 2- No 3- Unknown	1. ◯ 2. ◯ 3. ◯	
6	Coronary disease	1- Yes 2- No 3- Unknown	1. ◯ 2. ◯ 3. ◯	
7	Atrial fibrillation	1- Yes 2- No 3- Unknown	1. ◯ 2. ◯ 3. ◯	
8	Chronic kidney disease	1- Yes 2- No 3- Unknown	1. ◯ 2. ◯ 3. ◯	
9	Prior Stroke	1- Yes 2- No	1. ◯ 2. ◯	
10	Sleep disorder	1- Yes 2- No	1. ◯ 2. ◯	If Yes, what disorder?
11	HIV	1- Yes 2- No 3- Unknown	1. ◯ 2. ◯ 3. ◯	
12	Alcoholism	1- Yes 2- No 3- Unknown	1. ◯ 2. ◯ 3. ◯	

13	Smoking	1- Yes 2- No 3- Unknown	1. ◯ 2. ◯ 3. ◯	If Yes, how many packs of years _____

3. Clinical characteristics

Q/N0	VARIABLE	POSSIBLE ANSWERS	ANSWER	EXACT ANSWER
1	Symptom start date			----------
2	Blood pressure	1. Grade 1 2. Grade 2 3. Grade 3	1. ◯ 2. ◯ 3. ◯	----------
3	Heart rate (beats per minute) (bpm)			----------
4	Respiratory rate (cycles per minute) (cpm)			----------
5	Weight (kilograms-kg)			----------
6	Height (meters- m)			----------
7	Temperature (degrees celsius)	1- >38. 5 2- < 38.5	1. ◯ 2. ◯	
8	Body mass index (kg/m2)			----------
9	Glasgow Coma Scale			----------
10	Cognitive function	1- Altered 2- Preserved	1. ◯ 2. ◯	
	if the answer above is altered, what function is altered,	1- Aphasia 2- Amnesia 3- Agnosia 4- Apraxia	1. ◯ 2. ◯ 3. ◯ 4. ◯	----------
11	Epilepsy	1- Yes 2- No	1. ◯ 2. ◯	
12	Cranial nerve function	1- Altered 2- Preserved	1. ◯ 2. ◯	

	if the above answer is impaired, which nerve is impaired			----------
	Clinical manifestations nerve palsy			----------
13	Muscle strength of the right upper limb			----------
14	Muscle strength of the right lower limb			----------
15	Muscle strength of the left upper limb			----------
16	Muscle strength of the left lower limb			----------
17	Reflexes of the right upper limb	1- Hyperreflexia 2- Normal 3- hyporeflexia	1. ◯ 2. ◯ 3. ◯	
18	Reflexes of the left upper limb	1- Hyperreflexia 2- Normal 3- hyporeflexia	1. ◯ 2. ◯ 3. ◯	
19	Reflexes of the right lower limb	1- Hyperreflexia 2- Normal 3- hyporeflexia	1. ◯ 2. ◯ 3. ◯	
20	Reflexes of the left lower limb	1- Hyperreflexia 2- Normal 3- hyporeflexia	1. ◯ 2. ◯ 3. ◯	
21	Right Babinski reflex	1- Positive 2- Negative 3- Indifferent	1. ◯ 2. ◯ 3. ◯	
22	Left Babinski reflex	1- Positive 2- Negative 3- Indifferent	1. ◯ 2. ◯ 3. ◯	

23	Current NIHSS score	1- Mild stroke (1-4) 2- Moderate stroke (5-15) 3- Moderate to severe stroke (16-20) 4- Very serious stroke (exclude them	1. ◯ 2. ◯ 3. ◯ 4. ◯	
24	Laterality	1- Left 2- Right 3- Bilateral	1. ◯ 2. ◯ 3. ◯	

4. Para-clinical characteristics

Q/N0	VARIABLE	POSSIBLE ANSWERS	ANSWER	EXACT ANSWER
1	Type of stroke	1- Ischemic 2- Hemorrhagic	1. ◯ 2. ◯	
2	Location	1- Supratentorial 2- Infratentorial	1. ◯ 2. ◯	
3	**1. Supratentorial**			
	• Hemisphere	1- Left 2- Right 3- Bilateral	1. ◯ 2. ◯ 3. ◯	
	• Cortical	1- Yes 2- No	1. ◯ 2. ◯	
	If yes,	1- Anterior cortex (ant. and middle cerebral artery and its branches) 2- Posterior cortex (post. Cerebral artery and its branches) 3- Frontal lobe 4- Parietal lobe 5- Temporal lobe 6- Occipital Lobe	1. ◯ 2. ◯ 3. ◯ 4. ◯ 5. ◯ 6. ◯	
	• subcortical	1- Yes 2- No	1. ◯ 2. ◯	

	If yes	1- Internal capsule 2- Thalamus	1. ◯ 2. ◯	
4	**2. Infratentorial**			
	If the lesion is infratentorial, what structure?	1- Brainstem 2- Cerebellum	1. ◯ 2. ◯	
5	HIV serology	1- Positive 2- Negative	1. ◯ 2. ◯	
6	Fasting blood sugar or Glycated hemoglobin	1- High 2- Low 3- Normal	1. ◯ 2. ◯ 3. ◯	
7	LDL level (N = < 100mg/dl or 1g/l)			----------
8	HDL level (N = > 40mg/dl or >0.4g/l for males and > 55mg/dl or >0.5g/l for females.)			----------
9	TG level (N = < 150 mg/dl or < 1.5g/l.)			----------
10	Complications	1- Infection 2- Worsening level of consciousness 3- Others	1. ◯ 2. ◯ 3. ◯	
	Other Complications			----------
11	Urea (N = 6 – 24mg/dl gold			----------
12	Creatinine (N = 0.7 to 1.3 mg/dl or			----------
13	AST (N= 10-40 units/L)			----------
14	ALT (N= 10 – 55 units/L)			----------
15	Stroke etiology	1- Large vessel Atherosclerosis 2- Cardioembolism 3- Small vessel disease 4- Undetermined etiology	1. ◯ 2. ◯ 3. ◯ 4. ◯ 5. ◯	If other etiologies are determined, which ones? ----------

		5- Other determined etiology		
16	Electrolyte abnormality	1) Yes 2) No 3) Not documented	1. ◯ 2. ◯ 3. ◯	If yes, what anomaly?

5. Ongoing medications

Q/N0	VARIABLE	POSSIBLE ANSWERS	ANSWER
1	Antihypertensive treatment	1- Yes 2- No	1◯ 2.◯
2	If yes, what is the therapeutic class?	1- ICA 2- Diuretic 3- Beta-blockers 4- IEC/ARA2	
3	Insulin therapy	1- Yes 2- No	1◯ 2.◯
4	LMWH at preventive dose	1- Yes 2- No	1◯ 2.◯
5	Treatment with AVK	1- Yes 2- No	1◯ 2◯
6	Antiepileptic treatment	1- Yes 2- No	1◯ 2◯
7	If ischemic stroke, antiplatelet treatment	1- Yes 2- No	
8	Statins	1- Yes 2- No	1◯ 2◯
9	Osmotic Laxative	1- Yes 2- No	1◯ 2◯
10	Antidepressant treatment	1- Yes 2- No	1◯ 2◯
11	Hypnotic or sedative	1- Yes 2- No	1◯ 2◯
12	Other medication		----------
13	Re-education	1- Yes 2- No	1◯ 2◯
14	If yes,	1- Speech therapy 2- Physiotherapy 3- Number of sessions	1◯ 2◯ 3. ◯ 1◯

15	The person carrying out rehabilitation	1- Qualified 2- Member of the family 3- Others	1◯ 2◯ 3. ◯

SECTION B. SLEEP DISORDERS SCREENING

6. Pittsburgh Sleep Quality Index (PSQI)

Instructions: The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. **Please answer all questions.**

1. During the past month, what time have you usually gone to bed at night? _____
2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night?____
3. During the past month, what time have you usually gotten up in the morning? _____
4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spent in bed.) _____

5. During the past month, how often have you had trouble sleeping because you…	Not during the past month	Less than once a week	Once or twice a week	Three or more times a week
a. Cannot get to sleep within 30 minutes				
b. Wake up in the middle of the night or early morning				
c. Have to get up to use the bathroom				
d. Cannot breathe comfortably				
e. Cough or snore loudly				
f. Feel too cold				
g. Feel too hot				
h. Have bad dreams				
i. Have pain				
j. Other reason(s), please describe:				
6. During the past month, how often have you taken medicine to help you sleep (prescribed or "over the counter")?				
7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?				

	No problem at all	Only a very slight problem	Somewhat of a problem	A very big problem
8. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?				
	Very good	Fairly good	Fairly bad	Very bad
9. During the past month, how would you rate your sleep quality overall?				
	No bed partner or room mate	Partner/room mate in other room	Partner in same room but not same bed	Partner in same bed
10. Do you have a bed partner or room mate?				
	Not during the past month	Less than once a week	Once or twice a week	Three or more times a week
If you have a room mate or bed partner, ask him/her how often in the past month you have had:				
a. Loud snoring				
b. Long pauses between breaths while asleep				
c. Legs twitching or jerking while you sleep				
d. Episodes of disorientation or confusion during sleep				
e. Other restlessness while you sleep, please describe:				

7. Epworth Sleepiness scale

How likely are you to doze off or fall asleep in the following situations, not just feeling tired? Use the scale to choose the most appropriate number for each situation.

Do you find yourself dozing or falling asleep and not just feeling tired in the following situations:

0 = Would never doze / fall asleep 1 = Slight chance of dozing / fall asleep 2 = Moderate chance of dozing / fall asleep 3 = High chance of dozing / fall asleep

	0	1	2	3
Sitting reading				
Watching TV				
Sitting, inactive, in a public place (cinema, theater, meeting)				
As a passenger in a car driving non-stop for an hour				
Lying down in the afternoon to rest when circumstances permit				
Sitting talking to someone				
Sitting calmly after a meal without alcohol 0				
In a car immobilized for a few minutes in a jam				

8. **STOP-BANG Sleep Apnea Questionnaire**

STOP		
Do you **S**NORE loudly (louder than talking or loud enough to be heard through closed doors)?	Yes	No
Do you often feel **T**IRED, fatigued, or sleepy during daytime?	Yes	No
Has anyone **O**BSERVED you stop breathing during your sleep?	Yes	No
Do you have or are you being treated for high blood **P**RESSURE?	Yes	No

BANG		
BMI more than 35kg/m2?	Yes	No
AGE over 50 years old?	Yes	No
NECK circumference > 16 inches (40cm)?	Yes	No
GENDER: Male?	Yes	No

TOTAL SCORE		

High risk of OSA: Yes 5 – 8

Intermediate risk of OSA: Yes 3 - 4

Low risk of OSA: Yes 0 - 2

Section C: Hospital Anxiety and Depression Scale (HADS)

Check the box next to the answer that best describes how you felt over the past week. Don't be too long in your answers: it's better that they are immediate.

Depression	Anxiety		Depression	Anxiety	
		I feel tense or "upset":			**I feel like I am slowed down:**
	3	Most of the time	3		Almost all the time
	2	Most of the time	2		Very often
	1	From time to time, occasionally	1		Sometimes
	0	Not at all	0		Not at all
		I still enjoy the things I used to enjoy:			**I get a sort of frightened feeling like 'butterflies' in my stomach:**
0		Definitely as much		0	Not at all
1		Not quite so much		1	Occasionally
2		Only a little		2	Quite often
3		Not at all		3	Very often
		I feel a kind of dread as if something horrible is about to happen:			**I no longer care about my appearance:**
	3	Most certainly and very badly.	3		Definitely
	2	Yes, but not too bad	2		I don't take as much care as I should
	1	A little, but that doesn't worry me.	1		I may not take quite as much care
	0	Not at all	0		I take just as much care as ever
		I can laugh and see the funny side of things:			**I feel restless as I have to be on the move:**
0		As much as I always could		3	Very much indeed
1		Not quite so much now		2	Quite a lot
2		Definitely not so much now		1	Not very much
3		Not at all		0	Not at all

		Worrying thoughts cross my mind:			**I look forward to things:**
	3	A great deal of the time	0		As much as I ever did
	2	A lot of the time	1		Less than before
	1	From time to time, but not too often	2		Certainly, less than in the past
	0	Only occasionally	3		Not at all
		I feel cheerful:			**I get sudden feelings of panic:**
3		Not at all		3	Very often indeed
2		Not often		2	Quite Often
1		Sometimes		1	Not very often
0		Most of the time		0	Not at all
		I can sit comfortably and feel relaxed:			**I can enjoy a good book or a radio or television show:**
	0	Definitely	0		Often
	1	Usually,	1		Sometimes
	2	Not often	2		Not often
	3	No way	3		Very seldom

Please check that you have answered all questions

The scores: Total score: Depression (D)_____ Anxiety (A)_____

Scoring:

Total score: Depression (D) _____ Anxiety (A) _____

0-7 = Normal

8-10 = Borderline abnormal (borderline case)

11-21 = Abnormal (case)

Section D: Functional status evaluation

1. Barthel's index

1. Feeding	• Unable • Needs help cutting, spreading butter, etc., or needs a modified diet • Independent	**0** **5** **10**		
2. Bathing	• Dependent • Independent (or in the shower)	**0** **5**		
2. Rectal continence	• Incontinent (or needs to be given enemas) • Occasional accident • Continent	**0** **5** **10**		
2. Urinary continence	• Incontinent, or catheterized and unable to manage alone) • Occasional accident • Continent	**0** **5** **10**		
3. Mobility (on level surfaces)	• Immobile or < 50 yards • Wheel chair independent, including corners, > 50 meters • Walks with the assistance of a person (verbal or physical) > 50 yards • Independent (but can use any aid, e.g. a stick) > 50 yards	**0** **5** **10** **15**		
4. Stairs	• Unable • Needs help (verbal, physical, carrying assistance) • Independent	**0** **5** **10**		
5. Clothing	• Dependent • Needs help but can do about half unaided • Independent (including buttons, zips,laces, etc.)	**0** **5** **10**		
6. Toilet use	• Dependent • Needs a little help, but can do something alone • Independent (on and off, dressing, wiping)	**0** **5** **10**		
7. Personal care	• Needs help with personal care • Face/hair/teeth/independent shaving (instruments provided)	**0** **5**		
8. Transfer (Bed to chair	• Unable, no sitting balance • Major help (one or two people, physical), can sit • Minor assistance (verbal or physical) • Independent	**0** **5** **10** **15**		
		Total: /100		

2. **Modified Rankin scale**.

Rankin level	Description
0	No symptoms
1	No significant disability despite symptoms; able to perform all usual tasks and activities
2	Slight disability: Able to perform all daily activities without assistance, but unable to carry out previous activities.
3	Moderate disability. Requires some help, but able to walk without assistance*.
4	Moderately severe disability: unable to walk without assistance and unable to care for one's own bodily needs without assistance.
5	Severe disability: bedridden, incontinent and requiring constant attention and nursing care.
6	Death

Stroke severity.(National Institutes of Health Stroke Scale)

1a Consciousness Overall responsiveness	Vigilant, reacts strongly Non-vigilant, drowsy, reacts or responds after minor stimulation Reaction adapted only after intense or painful stimulation Stereotypical (neurovegetative) response or total unresponsiveness	0 1 2 3
1b Consciousness Orientation in time: age, month	Correctly answers 2 questions: his age and the month of the current year do not Answer only one of the 2 questions correctly Does not answer any of the 2 questions correctly, aphasic	0 1 2
1c ConsciousnessExecution of simple orders	Executes 2 commands well: close-open your eyes, close-open one hand Correctly executes only one order out of the 2 Does not execute either order (choose the non-paretic hand)	0 1 2
2 Oculomotor skills(horizontal only)	Normal voluntary movements and oculo-cephalic reflex Reducible gaze deviation Forced deviation or complete paralysis (despite oculo-cephalic maneuvers)	0 1 2
3 Visual field (4 quadrants tested by counting fingers)	No visual field disturbance Visual field asymmetry Complete hemianopia	0 1 2

	Lack of vision and/or lack of blinking when threatened			3
4 Facial paralysis(show teeth, raise eyebrows, close eyes)	Normal and symmetrical mobility Minor paralysis (obliterated nasolabial fold, asymmetrical smile) Partial paralysis (affecting the lower part of the face) Complete facial paralysis (upper and lower facial)			0 1 2 3
		G	D	T
5 Motor skills MS * 5G left arm (L) 5D right arm (D) 5T = 5G + 5D	Normal = holding the limb (90° or 45°) for 10 seconds Hold possible (90° or 45°) but < 10 seconds Movement against gravity but no support possible Limited movement (no movement against gravity), limb falls No movement possible	0 1 2 3 4	0 1 2 3 4	
6 Motor skills MI * 6G left thigh 6D right thigh 6T = 6G + 6D	Normal = limb held (30°) for 5 seconds Hold possible (30°) but < 5 seconds Movement against gravity but no support possible Very limited movement (no movement against gravity) No movement possible	0 1 2 3 4	0 1 2 3 4	
7 Ataxia * (finger-nose and heel-knee tests)	No ataxia (or motor deficit already taken into account) Ataxia present for one limb Ataxia for two limbs			0 1 2
8 Sensitivity (face, trunk, arm, leg stroke side)	Normal sensitivity Discreet deficit: poorly perceived sting (on the side of the motor impairment) Severe to total deficit: sting not felt (on the side of the motor impairment)			0 1 2
9 Language	Normal Discreet to moderate aphasia: communication difficult but possible Severe aphasia: fragmentary expression, very limited communication Mutism, global aphasia or coma			0 1 2 3
10 Dysarthria Joint	Normal joint Discreet to moderate dysarthria: little interference with comprehension Severe dysarthria: unintelligible speech (aphasia excluded)			0 1 2

11 ExtinctionSearch for negligence	No negligence (neither visual, nor auditory, nor tactile, nor spatial) Neglect of a modality (visual or auditory or tactile or spatial) Neglect Severe: complete hemibody, several modalities	0 1 2
Total score (0 to 42)	0=normal 42=maximum severity	

APPENDIX 4: 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°1080 /UY1/FMSB/VDRC/DAASR/CSD

CLAIRANCE ÉTHIQUE 10 JUIN 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 : MBANGE LIKOWO GERMAINE **Matricule: 17M089**

Travaillant sous la direction de :

- Pr NJAMNSHI Alfred KONGNYU
- Dr NGARKA Leonard

Concernant le projet de recherche intitulé : **Sleep disorders in stroke patients at the Yaoundé Central Hospital : prevalence, associated factors and impact on functional status**

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

APPENDIX 5: HOSPITAL ADMINISTRATIVE AUTHORIZATION

REPUBLIQUE DU CAMEROUN
Paix-Travail-Patrie

MINISTERE DE LA SANTE PUBLIQUE

SECRETARIAT GENERAL

DIRECTION DE L'HOPITAL CENTRAL DE YAOUNDE

UNITE ADMINISTRATIVE ET FINANCIERE

REPUBLIC OF CAMEROON
Peace-Work-Fatherland

MINISTRY OF PUBLIC HEALTH

SECRETARIAT GENERAL

DIRECTORATE OF CENTRAL HOSPITAL

ADMINISTRATIVE AND FINANCIAL UNIT

N°2023/714/AR/MINSANTE/SG/DHCY/UAF

Yaoundé, le 28 NOV 2023

AUTORISATION DE RECHERCHE

Je soussigné, **Professeur Pierre Joseph FOUDA**, Directeur de l'Hôpital Central de Yaoundé, accorde une autorisation de recherche, sous la direction du *Pr NJAMNSHI Alfred Kongnyu* et la codirection du Dr NGARKA Léonard à **Mme MBANGE LIKOWO Germaine**, étudiante en Médecine Générale, niveau 7 à la Faculté de Médecine et des Sciences Biomédicales de l'Université de Yaoundé I, sur le thème : « **Sleep disorders in stroke patients in two hospitals in Yaounde : prevalence, types, associates factors and impact on functional outcome** ».

L'intéressée est tenue au strict respect du règlement intérieur de l'Hôpital Central de Yaoundé et s'engage à déposer un exemplaire de ladite thèse à la Direction dudit hôpital après correction.

En foi de quoi, la présente autorisation lui est délivrée pour servir et valoir ce que de droit. /-

REPUBLIQUE DU CAMEROUN * HOPITAL CENTRAL DE YAOUNDE * MINISTERE DE LA SANTE PUBLIQUE
Le Directeur

Le Directeur,

Pr. Pierre Joseph Fouda.

DETAILED TABLE OF CONTENT

THANK YOU