### **CHAPTER ONE**

### 1.1-Introduction:

Non-traumatic subarachnoid hemorrhage (SAH) is a neurologic emergency characterized by the extravasation of blood into the space surrounding the central nervous system, which is usually filled with cerebrospinal fluid. The major determinant of nontraumatic SAH is rupturing of an intracra-nial aneurysm (80% of cases), which has a high rate of death and complications. A major cause for a poor outcome among patients with aneurismal SAH is re-bleeding, resulting in a mortality rate of 80%, Aneurismal rebreeding occurs in 4% of patients within 24 hours, 20% within 2 weeks and 50% within 6 months after the initial occurrence, Non-aneurysmal SAH including isolated perimesencephalic subarachnoid hemorrhage (20% of cases) has a good prognosis with some uncommon neurologic Complications, The diagnosis of nontraumatic SAH requires the physician to ascertain its etiology, since the cause of SAH guides its management, When compared to DSA, spiral CT angiography (CTA) is a faster and a more easily applied method. In contrast to another non-invasive imaging method, magnetic resonance angiography (MRA), spiral CTA enables faster acquisition of three dimensional images related to the cerebral vascular anatomy without patient motion, artifacts or artifacts due to flow rate. Another advantage of CTA is its applicability following routine non-enhanced cranial computed tomography (CT) in patients with suspected SAH in emergency conditions. In this study, we aimed to compare the effectiveness of single detector spiral CTA to DSA in diagnosis and evaluation of intracranial aneurysms in cases with acute SAH

#### 1.2 Problem

Non enhanced contrast CT brain fror the patient with subarachnoid hemorrhage lead to miss & delay the diagnosis there for the trail to investigate the patient with contrast is necessary ( CT Angiography of the vessels).

## 1.3 General Objectives:

To evaluate clinical usefulness of CT cerebral angiography in confirmation of Reason of SAH hemorrhage.

## 1.4 Specific Objectives:

To evaluate the usefulness of CTA in SAH.

To evaluate the findings pre and post contrast.

To correlate the findings with patient age and gender.

### **CHAPTER TOW**

### 2.1.1 central nervous system

The human central nervous system (CNS), having been evolved over the last 600 million years, is the most complex living organ in the known universe. It has been extensively investigated over centuries, and a vast body of materials has been gathered in the print form and more recently also in electronic format.

Neuroanatomy is presented in numerous textbooks, print brain atlases and electronic brain atlases. Several textbooks combine text with atlases, and some provide neuroanatomy for various specialties including neurosurgery, neurology, and neuroscience. neuroradiology, The comprehension in neurosurgical, neuroradioneuroanatomy is crucial any neuro-oncological, or neurological procedure. Therefore, CNS anatomy has been intensively studied by generations of neuroanatomists, neurosurgeons, neurologists, neuroradiologists, neurobiologists, and psychologists, among others, including Renaissance artists. This resulted, however, in neuroa natomy discrepan-cies, inconsistencies, and even controversies among various communities in terms of parcellation, demarcation, grouping, terminology, and presentation. Williams et-al (2009)

### 2.1.1.1 Structural (Gross) Neuroanatomy:-

1.parcellation of the brain was presented in 3D followed by sectional neuroanatomy. The stereotactic target structures and functional (Brodmann's) areas also are outlined. Parthenon, Lancaster (2001)

### 2.1.1.2 Brain Parcellation

The CNS consists of the brain and the spinal cord. The brain encases the fluid-filled ventricular system and is parcellated into three main components (Fig. 2.1a): Cerebrum, Cerebellum (the little brain), Brainstem.

The cerebrum comprises: Left and right cerebral hemispheres Interbrain between the cerebrum and the brainstem termed the diencephalon.

Deep gray nuclei the cerebral hemispheres are the largest compartment of the brain and are intercon- nected by white matter fibers (see Sect. 2.4.2). The

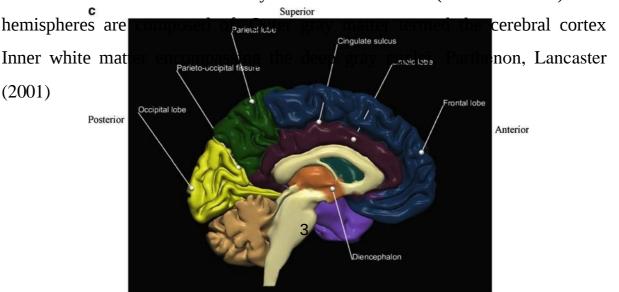


Fig. 2.1 Gross anatomy of the left cerebral hemisphere. Afshar, Watkins, et al Atlas of the Human Brainstem and Cerebellar Nuclei. Raven, New York (1978)

The gray matter contains mainly nerve cell bodies, while the white matter is made up predominantly of nerve fibers (axons). The cerebral cortex is highly convoluted. The folds form gyri that are separated by grooves called sulci or fissures (deep sulci). The cerebral hemispheres are parcellated into five lobes (Fig. 2.1b, c): Frontal lobe, Temporal lobe, Parietal lobe, Occipital lobe and Limbic lobe.

The insula is sometimes classified as the central or insular lobe. The lobes are partly demarcated by the sulci/fissures, Fig. 2.1. The central sulcus separates the frontal lobe anterior from the parietal lobe posterior, Fig. 2.1b. The Sylvian (lateral) fissure demarcates the temporal lobe below from the frontal and parietal lobes above, Fig. 2.1b. The parieto-occipital fissure separates the parietal lobe anterior from the occipital lobe posterior, Fig. 2.1c. The cingulate sulcus separates the frontal lobe above from the limbic lobe below, Fig. 2.1c. The diencephalon contains (Fig. 2.1c):Thalamus, Subthalamus including the subthalamic nucleus Hypothalamus. 7th edn. Lippincott Williams & Wilkins, Baltimore (2008)

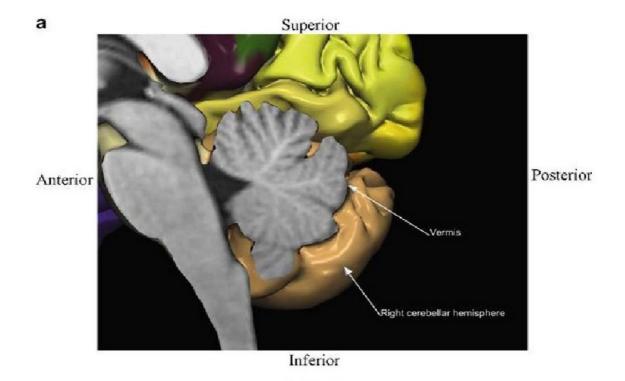
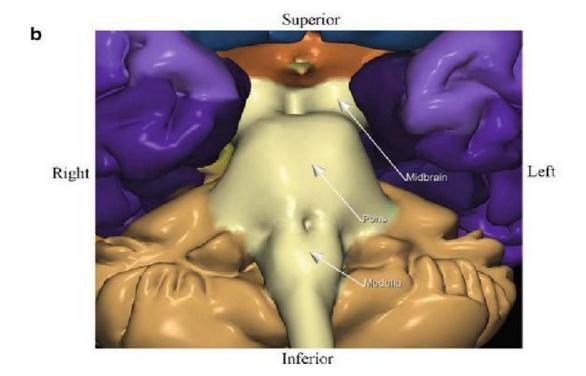


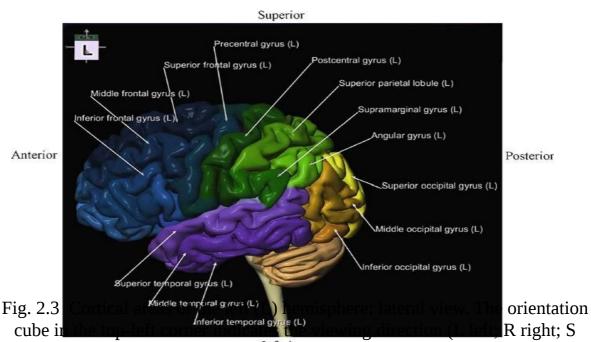
Fig. 2.2 Cerebellum and brainstem, cerebellum (medial view) Afshar, Watkins, et al Atlas of the Human Brainstem and Cerebellar Nuclei. Raven, New York (1978)

Left and right cerebellar hemispheres, vermis which unites them

The brainstem is subdivided into (Fig. 2.2b): Midbrain , Pons, Medulla



(Fig. 2.2b) (b) midbrain, pons, and medulla of the brainstem. Afshar, Watkins, et al Atlas of the Human Brainstem and Cerebellar Nuclei. Raven, New York (1978)



superior (dorsal); I inferior (ventral), A anterior; P posterior). Each gyrus is assigned a unique color. Afshar, Watkins, et al Atlas of the Human Brainstem and Cerebellar Nuclei. Raven, New York (1978)

### 2.1.1.3 Cortical Areas

The cortex has three surfaces: lateral, medial, and inferior (also called basal or

ventral). Moreover, the transitional areas form the frontal, temporal, and occipital poles. Williams & Wilkins, Baltimore (2008)

### 2.1.1.4 Lateral Surface

Four lobes are present on the lateral surface: frontal, temporal, parietal, and occipital, Fig. 2.1b. The lateral surface of the frontal lobe is subdivided by three sulci (superior frontal sulcus, inferior frontal sulcus, and precentral sulcus) into four gyri.

Superior frontal gyrus, Middle frontal gyrus, Inferior frontal gyrus and Precentral gyrus.

The lateral surface of the temporal lobe is subdivided by two sulci (superior temporal sulcus and inferior temporal sulcus) into three gyri Superior temporal gyrus, Middle temporal gyrus Inferior temporal gyrus

The lateral surface of the parietal lobe is subdivided by the intraparietal sulcus into three gyri.

Postcentral gyrus, Superior parietal gyrus (lobule), Inferior parietal gyrus (lobule), Supramarginal gyrus – Angular gyrus.

The lateral surface of the occipital lobe is subdivided by two sulci (superior occipital sulcus and inferior occipital sulcus) into three gyri.

Williams & Wilkins, Baltimore (2008).

### 2.1.1.5 Medial Surface

The frontal, parietal, occipital, and limbic lobes are present on the medial surface, Fig. 2.1c. The limbic lobe contains the gyri located at the inner edge (or limbus) of the hemisphere including. Subcallosal gyrus (areas), Cingulate gyrus, Isthmus (of cingulate gyrus) Parahippocampal gyrus The superior frontal gyrus (separated from the limbic lobe by the cingulate sul- cus, occupies most of the medial surface of the frontal lobe, Fig. 2.4. The parietal lobe includes the precuneus, Fig. 2.4 (separated from the occipital lobe by the parieto-occipital fissure, Fig. 2.1c). The occipital lobe comprises the cuneus and the lingual gyrus, Fig. 2.4.

#### 2.1.2 Inferior Surface

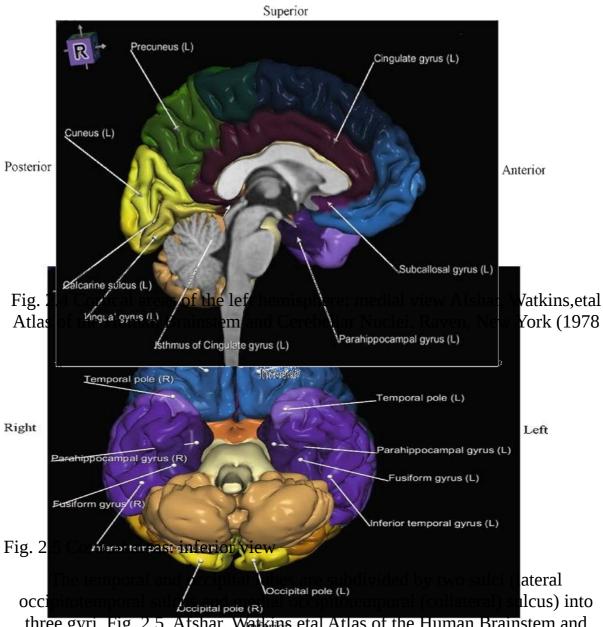
The inferior surface includes the frontal, temporal, and occipital lobes.

The frontal lobe comprises (Fig. 2.5):

Straight gyrus

Orbital gyri parcellated by the approximately H-shape sulcus into the anterior, medial, lateral, and posterior orbital gyri.

Williams & Wilkins, Baltimore (2008)



three gyri, Fig. 2.5. Afshar, Warkins, et al Atlas of the Human Brainstem and Cerebellar Nuclei. Raven, New York (1978

Medial occipitotemporal gyrus whose temporal part constitutes the parahip- pocampal gyrus and the occipital part the lingual gyrus. Inferior temporal gyrus The lingual gyrus is separated from the cuneus by the

Lateral occipitotemporal gyrus (called also the fusiform gyrus) calcarine sulcus (fissure).

## 2.1.2.1 Deep Gray Nuclei

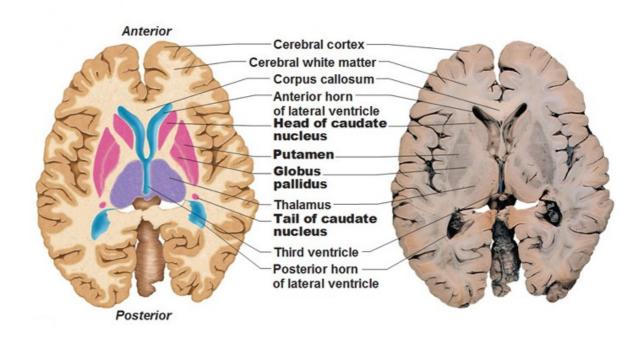
The deep gray nuclei are paired gray matter structures. The main deep gray nuclei (Fig. 2.6):

Basal ganglia (nuclei) include :-

Lentiform nuclei, Caudate nucleus, Putamen, Globus pallidus, Lateral (or outer) segment, Medial (or inner)

Thalamus, Hippocampus

Amygdala (amygdaloid body) The lentiform nuclei and the caudate nucleus form the striatum. Williams & Wilkins, Baltimore (2008)



**Fig**. 2.6 Planar neuroanatomy in axial orientation show deep gray nuclei Afshar, Watkins, et al Atlas of the Human Brainstem and Cerebellar Nuclei. Raven, New York (1978)

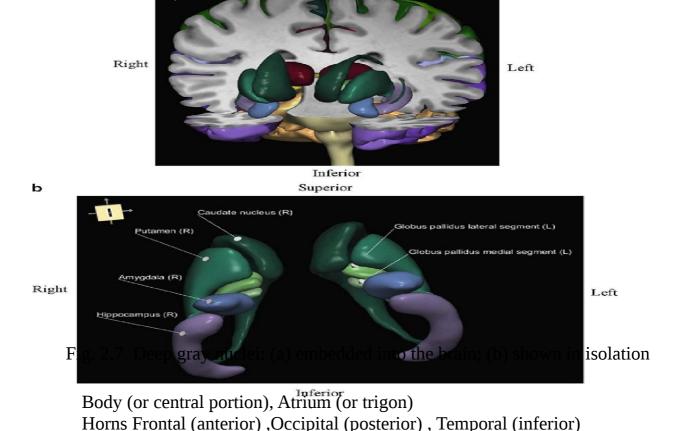
## 2.1.2.2 Ventricular System

The ventricular system contains four interconnected cerebral ventricles (cavities) filled with cerebrospinal fluid (CSF) (Fig. 2.7a):

left and right lateral ventricles, Third ventricle, Fourth ventricle

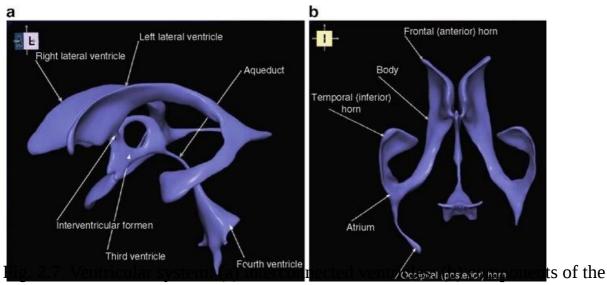
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CSF is secreted mainly in the choroid plexus (a network of vessels) and circulates from the lateral ventricles through the paired interventricular foramina (of Monro) to the third ventricle, and then via the aqueduct to the fourth ventricle, Fig. 2.7a. The lateral ventricles are the largest and each contains Williams & Wilkins, Baltimore (2008)



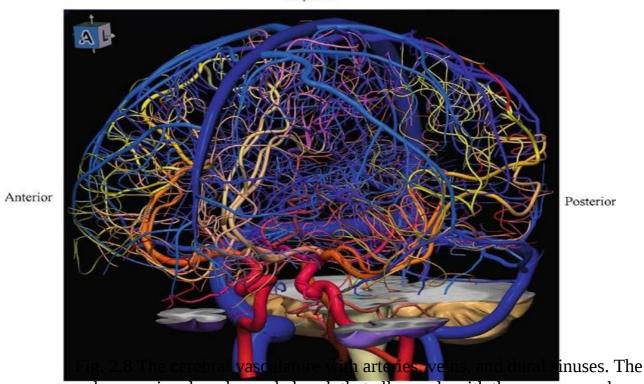
Superior

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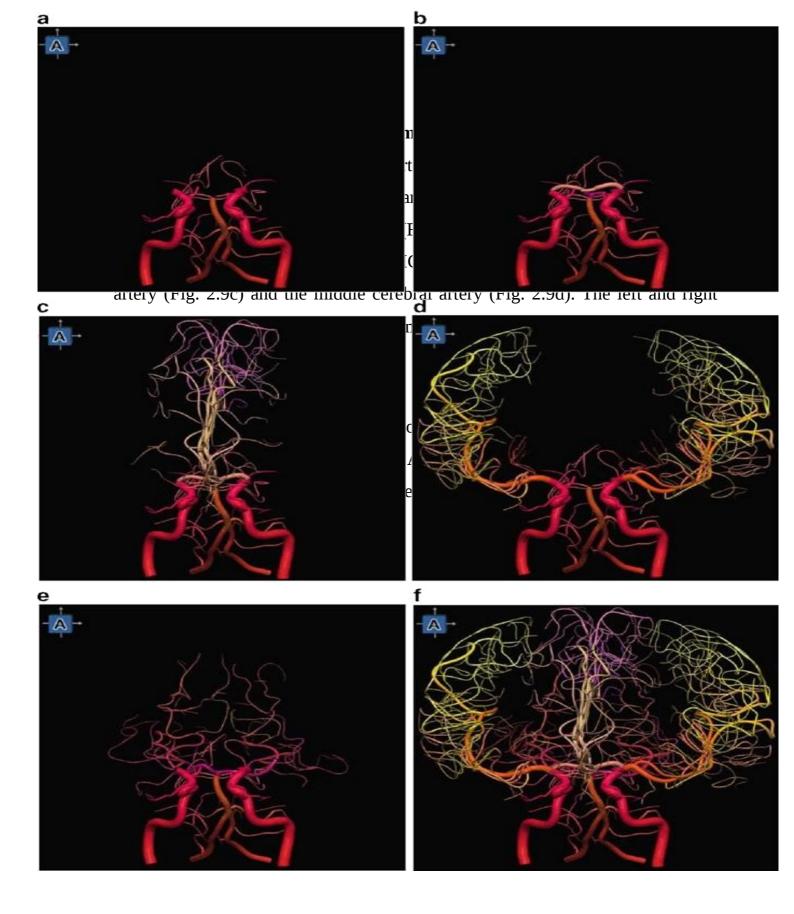


lateral ventricle Afshar, Watkins, et al Atlas of the Human Brainstem and Cerebellar Nuclei. Raven, New York (1978)

#### Superior



vessels are uniquely color-coded such that all vessels with the same name have the same color. Diamond,., Fusco, et al: Structure of the Human Brain. A Photographics Atlas, 3rd edn. Oxford University Press, New York (1989))



**Fig. 2.9** The cerebral arteries: (a) blood supply to the brain by the internal carotid artery (ICA) anteriorly, and the vertebral artery (VA) and the basilar artery (BA) posteriorly; (b) ICA and VA connected by the circle of Willis; (c) anterior cerebral artery along with the ICA, VA, and BA; (d) middle cerebral artery along with the ICA, VA, and BA; (e) posterior cerebral

artery along with the ICA, VA, and BA; (f) complete arterial system. Diamond, Fusco, etal: Structure of the Human Brain. A Photographics Atlas, 3rd edn. Oxford University Press, New York (1989)

#### **2.1.2.6 Middle Ce**

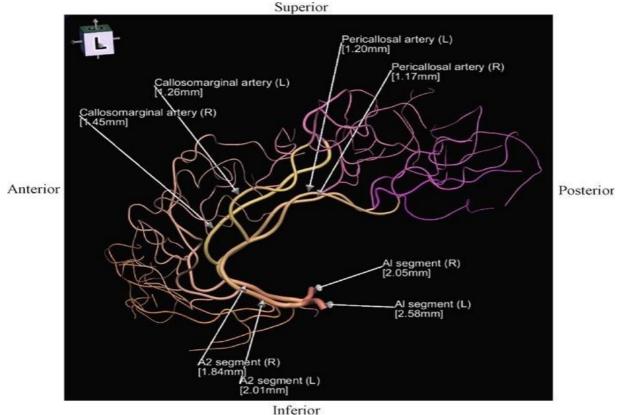
### rebral Artery

The middle cerebral artery is subdivided into four segments (Fig. 2.19a):

M1 segment (sphenoid part), M2 segment (insular part), M3 segment (opercular part), M4 segment (terminal part) Its main branches for the left hemisphere are shown in Fig. 2.19b. Williams & Wilkins, Baltimore (2008)

## 2.1.2.7 Posterior Cerebral Artery

The posterior cerebral artery is parcellated into four segments (Fig. 2.20):
P1 segment (precommunicating part), P2 segment (postcommunicating part)
P3 segment (lateral occipital artery), P4 segment (medial occipital artery)



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Fig. 2.10 show cerebral arteries Diamond,., Fusco, etal: Structure of the Human Brain. A Photographics Atlas, 3rd edn. Oxford University Press, New York (1989))

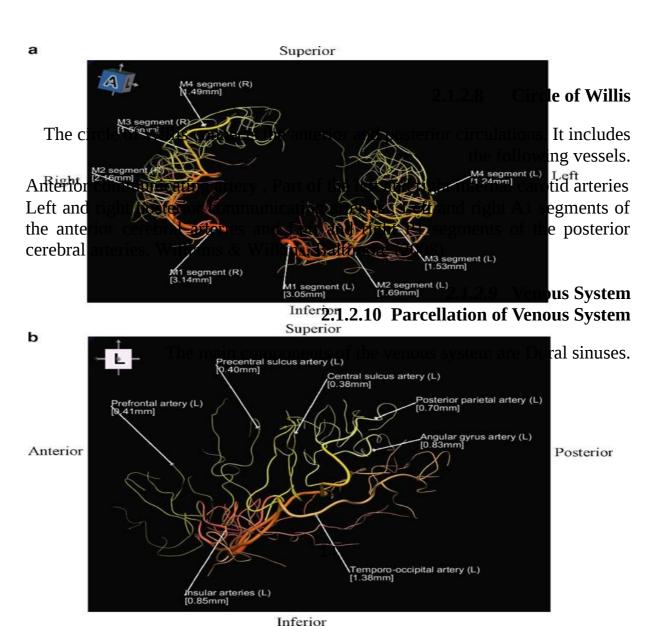


Fig. 2.11 Middle cerebral artery: (a) M1, M2, M3, and M4 segments; (b) main branches of the left hemisphere. Diamond,., Fusco, etal: Structure of the Human Brain. A Photographics Atlas, 3rd edn. Oxford University Press, New York (1989)

### 2.1.2.11 Cerebral Veins

The main superficial cerebral veins are: Frontopolar veins , Prefrontal veins , Frontal veins , Parietal veins , Occipital veins . Williams & Wilkins, Baltimore (2008)

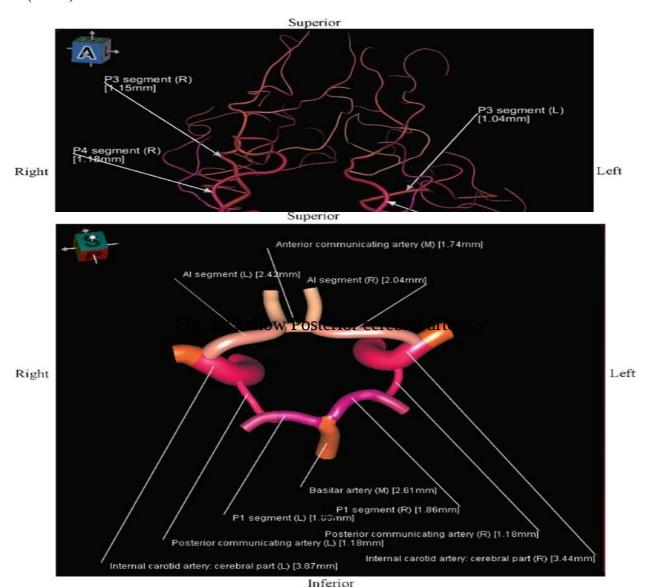


Fig. 2.13 show The circle of Willis Diamond, Fusco, etal: Structure of the Human Brain. A Photographics Atlas, 3rd edn. Oxford University Press, NY1989

2.1.2.12 Dural sinuses

The main dural sinuses are:

Superior sagittal sinus , Inferior sagittal sinus , Straight sinus , Left and right transverse sinuses and Left and right sigmoid sinuses . Williams & Wilkins, Baltimore (2008)

### 2.1.2.13 Cerebral Veins

The main superficial cerebral veins are:

Frontopolar veins, Prefrontal veins, Frontal veins, Parietal veins, Occipital veins. Williams & Wilkins, Baltimore (2008)

#### 2.1.2.14 Vascular Variants

The human cerebrovasculature is highly variable and vascular variants have been extensively studied. Variations exist in terms of origin, location, shape, size, course, branching patterns as well as surrounding vessels and structures. The knowledge of cerebrovascular variants is central in diagnosis, treatment, and medical education. Williams & Wilkins, Baltimore (2008)

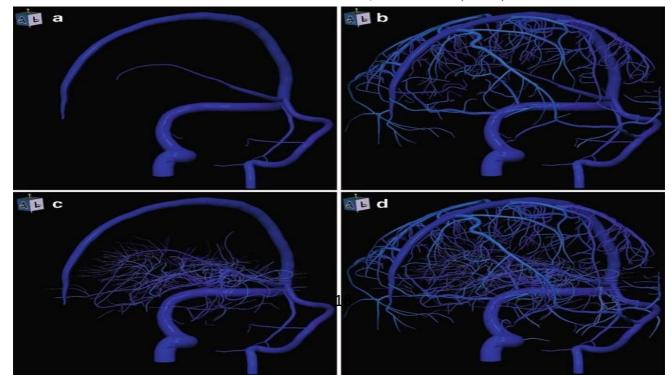


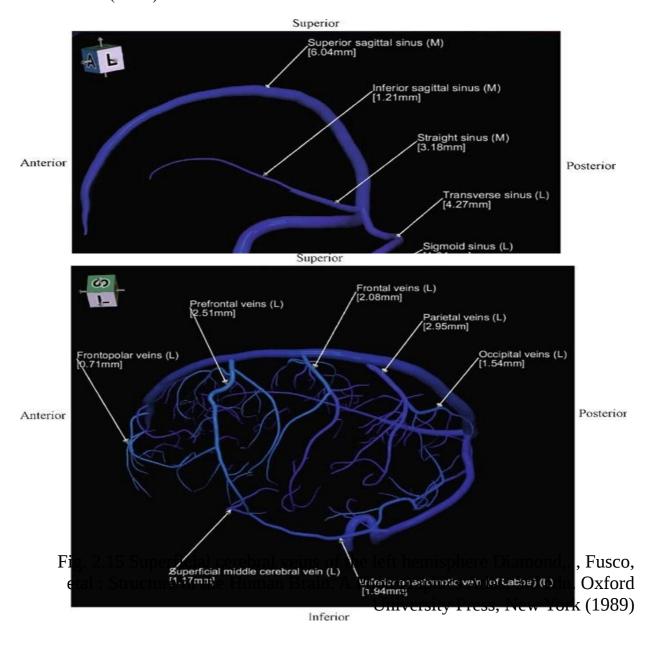
Fig. 2.14 Parcellation of the venous system: (a) dural sinuses (DS); (b) superficial veins with the DS; (c) deep veins with the DS; (d) complete venous system. Diamond,., Fusco, et al: Structure of the Human Brain. A Photographics Atlas, 3rd edn. Oxford University Press, New York (1989)

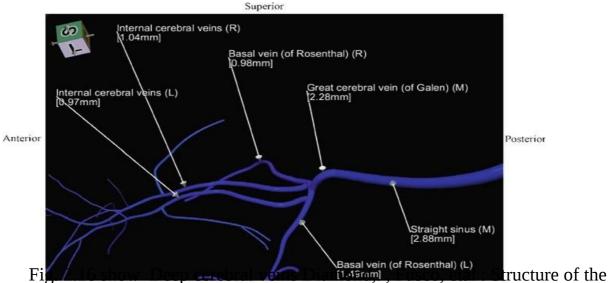
## 2.1.2.15Connectional Neuroanatomy

Three types of white matter connections (or tracts, fibers, bundles, fiber pathways, fascicles) are distinguished in the cerebral hemispheres.

Commissural tracts, Association tracts, Projection tracts In addition, three cerebellar paired peduncles:

Middle peduncle ,Superior peduncle, Inferior peduncle. Williams & Wilkins, Baltimore (2008)





Human Brain. A Photographics Atlas, 3rd edn. Oxford University Press, New York (1989))

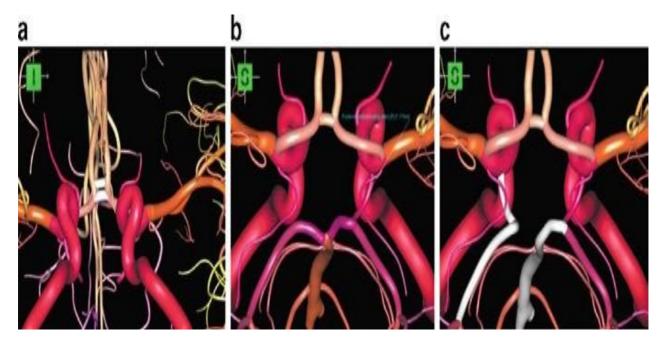


Fig. 2.17 Vascular variants of the circle of Willis: (a) double anterior communicating artery; (b) absent left posterior communicating artery; (c) absent left P1 segment (the variants are in *white*) Diamond,. , Fusco, etal : Structure of the Human Brain. A Photographics Atlas, 3rd edn. Oxford University Press, New York (1989))

## 2.2 physiology of the brain:-

Thr brain can be divide into six parts in terms of physiological functions:

Cerebrum, Hypothalamus, Midbrain, Cerebellum, Pons and Medulla oblongata. Rodney Rhoades, David R. Bell (2009)

2.2.1. Cerebrum

This is the most developed area of brain in the human species and is considered to be the center of the highest functions. The major functions include: awareness of sensory perception; voluntary control of movement (regulation of skeletal muscle movement); language; personality traits; sophisticated mental activities such as thinking, memory, decision making, predictive ability, creativity and self-consciousness. We will examine 4 lobes of the cerebrum. Rodney Rhoades, David R. Bell - 2009

### 2.2.2The Frontal Lobe

Concerned with higher intellectual functions and is involved in the many behavioral aspects of humans. It inhibits certain primitive behaviors. The Primary motor cortex controls the movement of the rest of the body while the premotor cortex just adjacent to it is concerned with the initiation, activation, and performance of the actual movement. Rodney Rhoades, David R. Bell 2009

#### 2.2.3The Parietal Lobe

This lobe is primarily concerned with the interpretation and integration of sensory inputs. The Somatosensory cortex is associated with reception and perception of touch, vibration, and position sense of the body. Rodney Rhoades, David R. Bell ( 2009)

## 2.2.4The Temporal Lobe

The temporal lobe contains the auditory cortex - for the reception and interpretation of sound information, and the olfactory cortex - for the sense of smell. It also houses the language cortex in the dominant hemisphere (usually the left hemisphere) and participates in recognition and interpretation of language. Rodney Rhoades, et-al (2009)

## 2.2.5 The Occipital Lobe

This lobe contains the primary visual cortex for visual information interpretation. degenerative conditions in specific regions can cause problems in fine motor control. Parkinson's disease is characterize by slow jerky movements; tremors of the face and hands; muscle rigidity; and great difficulty initiating voluntary movements. In Parkinson's disease, an overactive region acts like a stuck brake, continuously inhibiting the motor cortex. The disease results from the degeneration of a region called the substantia nigra, in particular dompaminergic neurons (those using the neurotransmitter dopamine) in this region. Huntington's disease involves an over stimulation of motor activities, such that limbs jerk uncontrollably. Syamal K,et-al (2008)

### 2.2.6The Limbic system

Is a group of structures on the medial aspect of each hemisphere and diencephalon and is more a functional system than an anatomical one. The limbic system is the "emotional brain", participating in the creation of emotional states such as fear, anger, pleasure, affection, arousal, etc. and processing vivid memories associated with those states. For example, the amygdala is central for processing fear and stimulates a sympathetic response. The amygdala enables us to recognize menacing facial expressions in others and to detect the precise gaze of someone who is looking at us. Syamal K,et-al (2008)

### 2.2.7Cerebral Lateralization

Although anatomically the two hemispheres of the cerebrum look very similar, functionally the two sides are different. Thus, the term lateralization is used to denote that each lobe has developed special functions that are not shared by other lobes. In general:

#### Left side:

Language, logic, analytical, sequential, verbal tasks, (holistic information processing). "Thinkers"

## Right side:

Spatial perception, artistic and musical endeavors (fragmentary information processing). "Creators"

## 2.2.8 Epithalamus, Thalamus and Hypothalamus

The epithalamus contains the pineal gland, a hormone secreting endocrine structure. Under the influence of the hypothalamus, the pineal gland secretes the hormone melatonin, which prepares the body for the night-time stage of the sleep/wake cycle. The thalamus makes up about 80% of the diencephalon and is the main relay center for the various sensory and motor functions The Hypothalamus controls and regulates many important functions of the body, including:

Control of the Autonomic Nervous System - adjusts, coordinates, and integrates the A.N.S. centers in the brain that regulate heart rate, blood pressure, bronchiole diameter, sweat glands, G.I. tract activity, etc. It does this via the Parasympathetic and Sympathetic divisions of the A.N.S.

Control of Emotional Responses - in association with the limbic system, it forms part of the emotional brain. Regions involved in fear, pleasure, rage and sex drive are located in the hypothalamus. Regulation of Body Temperature - the body's thermostat and set point is located in the hypothalamus. There are also 2 centers in the hypothalamus that respond to changes in the set point.

Heat-losing center: activation of this center causes sweating and cutaneous vasodilation. Heat-promoting center: activation of this center causes shivering and cutaneous vasoconstriction. Regulation of Hunger and Thirst Sensations - hypothalamus contains the feeding and thirst centers.

Feeding center: this center is always active and stimulates hunger which is 'fed' by eating. Satiety center: stimulated when satisfied, this inhibits the always hungry feeding center. Thirst center: osmoreceptors detect changes in osmotic pressure of blood, ECF, stimulate thirst.

Control of the Endocrine System - controls the release of pituitary hormones. Controls the anterior pituitary gland, when the hypothalamus releases hormones, it can stimulate or inhibit the release of other hormones form the pituitary (6 hormones). Also, it makes the 2 hormones (oxytocin and antidiuretic hormone (ADH)) that are stored in the posterior pituitary and released when signaled. All of these hormones regulate many other organs in the body. Rodney Rhoades,et-al (2009)

#### 2.2.9 Midbrain

Portions receive visual input auditory input from the medulla oblongata and are involved in cranial reflexes, e.g., when you turn your head if you thought you heard your name called out. Rodney Rhoades,et-al (2009)

## 2.2.10 The Cerebellum has two primary functions:

Controls postural reflexes of muscles in body - i.e., it coordinates rapid, automatic adjustments to maintain equilibrium, e.g. regaining your balance when you start to fall.

Produces skilled movements - involved in implementing routines for fine tuned movements. Controlled at the conscious and subconscious level, refines learned routines (e.g. driving, skating, playing an instrument) until the action becomes routine. This then reduces the need for conscious attention to the task. The cerebellum gets incoming information from proprioceptors, a type of sensory receptor found in movable joints, tendons and muscle tissue. Using the information from proprioceptors in the body, the cerebellum can determine the relative position of various body parts and compares motor commands and intended movements with the actual position of the body part (legs, arms). In this way, it can perform any adjustments needed to changes the direction or make the movement (action) smooth and coordinated .Syamal K,et-al (2008)

#### 2.2.12Pons

Plays a role in the regulation of the respiratory system. Contains two 'pontine' respiratory centers: 1) the pneumotaxic center and 2) the apneustic center. These two centers will be discussed later in the respiratory system. The pons is not responsible for the rhythm of breathing (the medulla oblongata is) but controls the changes in depth of breathing and the fine tuning of the rhythm of breathing set by the medulla oblongata. The pons also prevents over inflation of the lungs. Rodney Rhoades, et-al (2009)

### 2.2.13 Medulla Oblongata

The medulla oblongata is the last division of the brain. It becomes continuous with the spinal cord. It houses some very important visceral or vital centers, The cardiac center adjusts the force and rate of the heartbeat.

The vasomotor center regulates the diameter of blood vessels and therefore systemic blood pressure (constriction increases and dilation decrease blood pressure) and the respiratory center – for control of the basic rhythm and rate of breathing. Additional centers regulate sneezing, coughing, hiccupping, swallowing and vomiting. Rodney Rhoades,et-al (2009)

## 2.3 Pathology of the brain

### 2.3.1Cerebral Edema

Excess fluid (increased volume) within or around the brain parenchyma. Owl Club Review Sheets (2013)

### 2.3.2Raised ICP and Herniation

Mean ICP of CSF > 200 mmH2O with patient recumbent, occurring when expansion of the brain parenchyma exceeds compression of veins and CSF . Owl Club Review Sheets (2013)

### 2.3.3 Types of Herniation

# 2.3.3.1 Subfalcine Herniation Cingulate Gyrus

Unilateral expansion of the cerebral hemisphere displaces the cingulated gyrus under the falx cerebri, compressing pericollosal arteries (arteries of corpus callosum) and anterior cerebral circulation. Owl Club Review Sheets (2013)

## 2.3.3.2Transtentorial Herniation Uncal

Medial Aspect of Temporal lobe goes through the tentorium cerebella Compression of the 3rd CN ipsilateral pupil dilation and eye paralysis Compression of the posterior cerebral artery infarct of visual cortex Compression of the contralateral peduncle ipsilateral hemiparesis (relative to the herniation); called Kernohan's Notch Hemorrhage in midbrain and pons may result (Duret's Hemorrhage) Owl Club Review Sheets (2013)

### 2.3.3.3 Tonsilar Herniation Cerebellum

Fatal herniation of cerebellum through the foramen magnum Compresses brainstem, leading to death Owl Club Review Sheets (2013)

### 2.3.4 Hydrocephalus

Accumulation of excessive CSF within the ventricular system. Owl Club Review Sheets (2013)

## 2.3.5 MALFORMATIONS AND DEVELOPMENTAL DISEASES:

### 2.3.5.1 Neural Tube Defects:

Failure of the neural tube portion to close or a closed region reopening, the most common CNS malformations. Owl Club Review Sheets (2013)

### 2.3.6Type and Morphology:

### 2.3.6.1 Anencephaly:

Incompatible with life, occurring around day 28 gestation . Owl Club Review Sheets (2013)

### 2.3.6.2 Encephalocele:

Protrusion of brain through a defect in the skull. Owl Club Review Sheets (2013)

## 2.3.6.3 Spina Bifida

Most common neural tube defect; failure of closure of caudal aspect usually occurring in the lumbarsacral region. Owl Club Review Sheets (2013)

#### 2.3.7Posterior Fossa:

### 2.3.7.1Arnold-Chiari Malformation:

☐ Small posterior fossa misshapen cerebellum vermis of cerebellum extending through foramen magnum (Herniation). Owl Club Review Sheets (2013)

## 2.3.7.2 Dandy-Walker Malformation:

☐ Enlarged posterior fossa + absent cerebellar vermis + midline cyst Owl Club Review Sheets (2013)

#### 2.3.8 PERINATAL INJURY:

### 2.3.8.1 Intraparenchymal Hemorrhage

- -Germinal Matrix is present only in the fetal and neonatal brain around the ventricles
- -Hypoxia/Ischemia causes bleeding in this region
- Divided into 4 grades depending on involvement of ventric

Grade 1: Germinal Matrix Only, Grade 2: Germinal Matrix Ventricles without Hydrocephalus / Dilation, Grade 3: Germinal Matrix Ventricles with Hydrocephalus, Grade 4: Germinal Matrix Ventricles Parenchyma Owl Club Review Sheets (2013)

### 2.3.9 Periventricular Leukomalacia:

Infarcts occurring in white matter near to the ventricles, especially in premature babies Owl Club Review Sheets (2013)

### 2.3.10 Parenchymal Injuries:

#### **2.3.10.1 Concussion**

Clinical syndrome of altered mental status following a change in the momentum of the head (abrupt stop against a brick wall, for example) Owl Club Review Sheets (2013)

## 2.3.11 Direct Parenchymal Injury:

#### 2.3.11.1Contusion or Laceration

Contusion is the transfer of kinetic energy resulting in a "brain bruise Laceration is the penetration of an object into the tissue

### 2.3.12 Diffuse Axonal Injury:

Associated with Angular Acceleration even in the absence of impact (like in a car accident, where the patient doesn't actually strike anything, but dies) Owl Club Review Sheets (2013)

## 2.3.13Traumatic Vascular Injury:

## **2.3.13.1 Epidural**

Dura is closely affixed to skull, representing a potential space between

Associated with the middle meningeal artery and temporal trauma

Smooth linear contour of hematoma that compresses brain "Lens" or "Ellipicatal" shape on CT scan Usually a clinically lucid interval just prior to rapid progression to death. Owl Club Review Sheets (2013)

### 2.3.13.2Subdural

Between the dura and the arachnoid exists a real space Associated with bridging veins and dural sinuses coursing through Brain can move but the vessels are fixed; with trauma, brain shears the vessels and the patient bleeds Superior sagital sinus of the elderly and demented are at highest risk Hematoma hugs the brain matter, but does not enter subarachnoid space (isn't between the sulci), called a crescent shaped hematoma. Owl Club Review Sheets (2013)

## 2.3.14 Hypoxia, Ischemia, Infarction

Infarction from Obstruction to Flow (Focal Cerebral Ischemia)

Thrombotic or Embolic event that occludes the lumen to blood flow, depriving a particular region of tissue, supplied by that artery, of O2. Owl Club Review Sheets (2013)

## 2.3.15 Intracranial Hemorrhage:

Bleeding into the cerebral tissue from cerebral vasculature within the tissue. This is bleeding inside the brain.

## 2.3.15.1 Subarachnoid Hemorrhage, Ruptured Saccular Aneurysm:

Bleeding into and around the brain parenchyma (between pia and arachnoid layers) from cerebral vasculature.

### 2.3.16 Vascular Malformations:

### 2.3.16.1Arteriovenous Malformation

Arteries connected to veins without an intervening capillary bed. Owl Club Review Sheets (2013)

## 2.3.16.2 Cavernous Hemangioma

Occur most commonly in the cerebellum, pons, subcortex Distended, loosely organized, low-flow vasculature with thin collaginized walls devoid of intervening nervous tissue. Owl Club Review Sheets (2013)

## 2.3.17 Hypertensive Vascular Disease

## 2.3.17.1Hypertensive Cerebral Hemorrhage

Bleeding into and around the brain parenchyma (between pia and arachnoid layers) from cerebral vasculature. Owl Club Review Sheets (2013)

## 2.3.17.2 Lacunar Infarcts

HTN affects the blood vessels that supply the basal ganglia and white matter developing arteriolar sclerosis that may become occluded (just like regular vessels from the CV block . Owl Club Review Sheets (2013)

## **2.3.17.3** Hypertensive Encephalopathy

HTN causes dementia, loss of function, basically "screwy-brain"

☐ Cerebral dysfunction, headache, confusion, vomiting, coma ☐ Rapid intervention required as this will not resolve. Owl Club Review Sheets (2013)

## 2.3.18 Acute Meningitis:

## 2.3.18.1 Acute Pyogenic Meningitis Bacterial

Inflammation of the meninges brought about by bacterial infection. Owl Club Review Sheets (2013)

## 2.3.18.2 Acute Aseptic Meningitis Viral

A misnomer, "aseptic" is a clinical term for meningeal signs without the ability to demonstrate causative organisms. Owl Club Review Sheets (2013)

## **2.3.19 Acute Focal Supparative Infections:**

#### 2.3.19.1 Brain Abscess

Discrete lesions with central liquifactive necrosis surrounded by a fibrous capsule found within brain parenchyma. Owl Club Review Sheets (2013)

## 2.3.19.2 Subdural Empyema

Emergent collection of pus between dura and arachnoid. Owl Club Review Sheets (2013)

### 2.3.19.3 Epidural Abscess

Slow growing infection between dura and skull associated with osteomyelitis from another source (sinusitis or surgery). Owl Club Review Sheets (2013)

### 2.3.20 Chronic Bacterial Meningocephalitis

#### 2.3.20.1 Tuberculosis

Found at the base of the brain may cause a mass effect from the tuberculoma may cause arachnoid fibrosis leading to hydrocephalus. Owl Club Review Sheets (2013)

### 2.3.21 DEMYELINATING DISEASES:

## 2.3.21.1 Multiple Sclerosis (Autoimmune)

An autoimmune demyelinating disorder characterized by distinct neurologic deficits separated by time, caused by white matter lesions separated in space. Owl Club Review Sheets (2013)

### 2.3.21.2 Guillan-Barre Syndrome (Autoimmune)

Ascending Paralysis begins in the lower limbs and distal extremities (toes and fingers first) that finishes with death from paralysis of the diaphragm. Owl Club Review Sheets (2013)

#### **2.3.22 DEGENERATIVE DISEASES:**

### 2.3.22.1Alzheimer's

A progressive degenerative disease of the cerebral cortex caused by accumulation of abnormal proteins, demonstrable as plaques and tangles. Owl Club Review Sheets (2013)

### 2.3.22.2 Parkinsonism

Caused by damage to the nigrostriatal dopamanergic system Characterized by diminished facial expression, stooped posture, slow movements, cog-wheel rigidity, pill-rolling tremor, and festinating gait (progressively shortened, accelerated steps, aka "Shuffling"). Owl Club Review Sheets (2013)

### **2.3.23 TUMORS:**

## 2.3.23.1 Glioblastoma Multiforme (GBM)

Most common CNS tumor and is ring enhancing has rows of anaplastic cells lined up around a region of central necrosis, called pseudopalasading necrosis. Owl Club Review Sheets (2013)

## 2.3.23.2 Pilocytic Astrocytoma

Benign astrocytic tumor of children and young adults characteristic cystic lesion connected to a mural nodule seen on MRI. Owl Club Review Sheets (2013)

## 2.3.23.3 Meningiomas

Derived from meningothelial cells of the arachnoid Tumors occur in adulthood, men more often than women. Owl Club Review Sheets (2013)

### 2.3.23.4 Medulloblastoma

Derived from primordial neuroglial precursors, so is a poorly differentiated tumor typically develop in children, usually in the cerebellum.

## 2.3.23.5 CNS Lymphoma

High grade B-cell non-hodgkins lymphoma, commonly infected with Epstein Barr Virus Occurs in immunocompromised such as AIDS. Owl Club Review Sheets (2013)

#### 2.3.24 Generalities:

## **2.3.24.1** Epilepsy

is a disease of the brain in a patient who has had at least one seizure and a cerebral defect predisposing them for others. Owl Club Review Sheets (2013)

## 2.3.24.2 Seizure

is a clinical neurological event characterized by a change in behavior, sensation, or cognition associated with hypersynchronous cerebral discharge. Owl Club Review Sheets (2013)

**2.3.24.3** Status Epilepticus is one long seizure or multiple seizures back to back without regainin, Owl Club Review Sheets (2013)

## 2.4 Pervious study:

This study was done in The University of Michigan Neurosurgery Intensive Care Unit (Neuro ICU) 80% of SAH cases are caused by a ruptured brain aneurysm. An aneurysm is a weak area in the wall of brain artery that bulges out like a balloon, usually in the shape of a berry or a blister. The bulge may stretch and cause the vessel's wall to get thinner and thinner until it breaks. This is called a rupture. An injury, infection or an inherited tendency may start an aneurysm that grows silently over time. There are two types of aneurysms: a saccular aneurysm and a fusiform aneurysm. The differing shapes may affect treatment choices. Scientists suspect that up to 15 million Americans (about five out of every 100) may have brain aneurysms. About 30,000 people per year experience an aneurysm rupture. Other conditions in which blood vessels in the brain become strained also increase the risk for SAH. These include: High blood pressure, A strong blow to the head from an accident or fall Rare, genetic conditions, Arteriovenous malformation (in 5% of cases).

This study was done by the Society for Academic Emergency Medicine that prevalence in ED headache patients was conservatively estimated at 15%. Representative studies reported CT sensitivity for SAH to be 91% (95% confidence interval [CI] = 82% to 97%) and sensitivity of CTA for aneurysm to be 97.9% (95% CI = 88.9% to 99.9%). Based on these data, the posttest probability of excluding an eurysmal SAH after an egative CT/CTA was 99.43% (95% CI = 98.86% to 99.81%). Robert et-al (2009)

This study is done by the Society for Academic Emergency Medicine A total of 131 patients were approached, 116 were enrolled, and 106 completed the study. In six of 116 patients (5.1%), aneurysm was found on CTA with normal CT and positive findings on LP; three had a positive CTA with normal CT and LP findings (one of which had a negative cerebral angiogram), and there was one false-positive CTA. Follow-up of all 131 patients showed no previously

undiagnosed intracranial pathology. In this patient population, 4.3% (5/116) were ultimately found to have an SAH and/or aneurysm. Conclusions: In this pilot study, CTA was found to be useful in the detection of cerebral aneurysms and may be useful in the diagnosis of aneurysmal SAH. A larger multicenter study would be useful to confirm these results. Shaun D et-al (2006).

This study is done by Department of Neurosurgery, Sheri- Kashmir Institute of Medical Sciences (SKIMS), India they found 60 ruptured aneurysms in total of 75 patients on which 3D-CTA was done. 58 aneurysms found on 3D-CTA were confirmed on intra-operative findings during surgery and were surgically managed. In the study, CTA have a sensitivity of 96.7% and specificity of 75.0% for detecting aneurysms. Conclusion: Majority of patients with intracranial aneurysms could. Javeed I Zargar et-al 2014

This study is done by AJNR Am J Neuroradiol 31:696–705Apr 2010 that One hundred ninety-three patients with SAH and negative findings on CTA who underwent subsequent DSA were identified. The distribution of blood on unenhanced CT was the following: in 93 patients, diffuse aneurysmal pattern in 50, no blood on CT (xanthochromic LP) in 32, and peripheral sulcal distribution in 18. All patients with PMH had negative findings on DSA. One patient with noblood CT had vasculitis on DSA. Six of 18(33%)patients with peripheral blood had vasculitis on DSA. Three of these were also diagnosed by CTA. All except 1 patient with diffuse aneurysmal blood had negative findings on DSA. One patient was diagnosed with an aneurysm on DSA (1/50, 0.5%). Repeat delayed DSA performed in 28 of these patients revealed a small aneurysm in 4 (14%). Five patients had a complication of DSA (2.6%); 1 was a clinical stroke (0.5%). T. Andersson et-al (2010)

### **CHAPTER THREE**

## 3.1 Study design, area and duration

This study was a descriptive, study designed to assess the ability of CTA to diagnose SAH in adult Emergency Department patients presenting with a chief complaint of headache conc.

erning for SAH. The study was collected from radiology department of ROYAL CARE INTERNATIONAL HOSPITAL, AL ZAYTONA SPECIALIST HOSPITAL AND ROYAL SCAN CENTER. The study was carried out in the (Khartoum- Sudan). The study was carried out with in 9 month from march 2014 to February 2015.

#### 3.1.1 Machine used

The machin used is multi slice CT Scan 64 slice (Toshiba).

## 3.1.2 Machine principle.

Technique and 3d subtraction MIP +MPR

### 3.1.3Accessories Instrumentations used

Automatic injector

Multislice CT scan

Contrast media (OMNIPAQUE) 70-80ML

Flow rate 4-4.5 ML/SEC

## 3.2.1 Study Population

The study include Sudanese group of patient with subarachnoid hemorrhage.

#### 3.2.2 Inclusion criteria

Study includes 50 patients visiting the emergency department with headache, neck stiffness diagnosing as subarachnoid hemorrhage.

#### 3.2.3 Exclusion criteria

Exclusion criteria Patient whom have traumatic subarachnoid hemorrhage and normal patient.

#### 3.2.4 Variables

The data of patients obtained from work sheet is used to collect data on 10 variables (appendix1).these variables were divided in to main categories data of

the patient include: age, gender, clinical findings, findings pre contrast, findings post contrast, final diagnosis, site, size, and CT NO In HU unit.

#### 3.2.5 Data collection

Data collection according to work sheet (appendix) include all above variables data .

### 3.2.6 Data analysis

Data analysis by using spss vertion 11 .using significant test like T test, Frequencies and regression and also correlation between age and prevalence and gender .

### 3.2.7 Methods

50 patients were diagnosed as SAH received a spiral scan with 120 kVp and, 300 MAS and contrast media of 70-80 ml. Patients. The technique used patient supine head first CT combines the use of x-rays with computerized analysis of the images with 5mm slice thickness. Beams of x-rays are passed from a rotating device through the patient head from several different angles to obtain projection images, which then are assembled by computer into a three-dimensional picture of the head vessels. Contrast Material Injection Short scan times require short contrast material injection. The injection protocols used to deliver an appropriate amount of iodine, injection rates of 4–5 mL/sec and highly concentrated contrast medium (iodine, 350–370 mmol/mL). The utility of the contrast material bolus can be increased if a saline bolus is appended. Flushing of the veins reduces streak artifacts due to beam hardening.

## 3.2.8 Image Postprocessing Techniques

The image processing techniques used is multiplanar reformation (MPR) and maximum intensity projection (MIP), as well as surface and volume rendering. Because bone and calcifications are seen as a particular problem in CT angiography, a variety of different approaches have been advocated to cope with this problem. Editing, volume cropping, manipulation with transfer functions, and segmentation are common but time-consuming techniques, not convenient in the emergency setting. Visualization with interactive MPR, sliding thin-slab MIP, or standardized volume rendering presets in combination with clip planes is more appropriate. Sophisticated operations like volume rendering with 2D transfer functions or bone subtraction are emerging techniques that enhance the visualization of vascular disease with minimal user interaction.

### **CHAPTER FOUR**

4 Result

From a total 50 patients with different age and gender all the patient are diagnosed as SAH fig No (4). there ware 35 males 70% fig NO(2) and 15 females 30% fig NO(2) in the study they found 46% fig No (3) of patient complain of headache and 50% fig NO(3) complain of headache + neck stiffness. After contrast administration 56% fig No (5) of patient diagnosed as aneurismal dilatation, 8% fig No (5) AVM, 14% AVM + aneurysm and 22% fig No (5) of patient no AVM or aneurysm. The second findings According to site they found that the right side 56% fig No (7) and the left side is 44%. Fig No (7) The percentage in the male 52% is greater than the female 10% in the final diagnosis (rupture aneurism) and the p-value equal 0.004.

Table No 4.1: **show AGE Distribution** 

AGE				
	Frequency	Percentage		
6-14 y	2	4		
15-24 y	1	2		
25-34 y	10	20		
35-44 y	7	14		
45-54 y	10	20		
55+ y	20	40		
Total	50	100.0		

Figure No 4.1 : show AGE show AGE Distribution

**Table No 4.2: show Gender Distribution** 

Gender			
	Frequency	Percent	
Male	35	70.0	
Female	15	30.0	
Total	50	100.0	

Figure No 4.3: show Gender distribution

**Table No 4.4 show clinical findings frequency and Percentage** 

clinical findings				
	Frequency	Percent		
Headache	23	46		

Headache+ neck stiffness	25	50
Headache+neck stiffness +coil	2	4
Total	50	100.0

Figure No 4.5 : show clinical findings of the patients

Table No 4.6 :show CT Findings pre contrast

findings pre contrast				
	Frequency	Percent		
Subarachnoid hemorrhage	50	100		
Total	50	100.0		

Figure No 4.7 show CT findings pre contrast

**Table No 4.8 show CT Findings post contrast** 

findings post contrast							
	Frequency	Percent					
Aneurismal dilatation	28	56.0					
AVM	4	8.0					
AVM + aneurysm	7	14.0					
No AVM or aneurysm	11	22.0					
Total	50	100.0					

Figure No 4.9 : show CT findings post contrast

Table No 4.10 **CT finding post contrast** 

finding post contrast						
Frequency Percent						
AVM	4	8.0				

Rupture aneurysm	31	62.0
Rupture aneurysm+ AVM	4	8.0
SAH left side	6	12.0
SAH rigtht side	5	10.0
Total	50	100.0

Figure No 4.12:CT finding post contrast

Table No 4.13: show the site (RT.LT) of the (SAH)

site (RT.LT)		
	Frequency	Percent
Right	28	56.0
Left	22	44.0
Total	50	100.0

Figure No 4.14: show the site (RT.LT)

Table 4.15Crosstab between final diagnosis and gender:

			Gender		Total
			Male	Female	101d1
	AVM	Count	0	4	4
	AVIVI	% of Total	.0%	8.0%	8.0%
	Rupture	Count	26	5	31
	aneurysm	% of Total	52.0%	10.0%	62.0%
Final	rupture	Count	2	2	4
diagnosis	aneurysm+ AVM	% of Total	4.0%	4.0%	8.0%
	SAH left	Count	5	1	6
	side	% of Total	10.0%	2.0%	12.0%
	SAH rigtht	Count	2	3	5
	side	% of Total	4.0%	6.0%	10.0%
Total		Count	35	15	50
TOldi		% of Total	70.0%	30.0%	100.0%

**P**-value=0.004

Table 4.16Crosstab Crosstab between final diagnosis and clinical findings:

			Clinical	Clinical findings			
			Headac he	Headache + neck stiffness	Headache + neck stiffness +coil	Total	
	AVM	Count	3	1	0	4	
	AVIVI	% of Total	6.0%	2.0%	.0%	8.0%	
	Rupture	Count	15	15	1	31	
	aneurysm	% of Total	30.0%	30.0%	2.0%	62.0%	
Final	rupture	Count	0	3	1	4	
diagnosis	aneurysm+ AVM	% of Total	.0%	6.0%	2.0%	8.0%	
	SAH left	Count	4	2	0	6	
	side	% of Total	8.0%	4.0%	.0%	12.0%	
	SAH rigtht	Count	1	4	0	5	
	side	% of Total	2.0%	8.0%	.0%	10.0%	

Total	Count	23	25	2	50
TOtal	% of Total	46.0%	50.0%	4.0%	100.0%

P-value=0.190 Table 4.17Crosstab Crosstab between final diagnosis and finding pre contrast:

			finding pre contras t	Total
			SAH	
	AVM	Count	4	4
	AVIVI	% of Total	8.0%	8.0%
	Rupture	Count	31	31
	aneurysm	% of Total	62.0%	62.0%
final	rupture	Count	4	4
diagnosis	aneurysm+ AVM	% of Total	8.0%	8.0%
	SAH left	Count	6	6
	side	% of Total	12.0%	12.0%
	SAH rigtht	Count	5	5
	side	% of Total	10.0%	10.0%
Tatal		Count	50	50
Total		% of Total	100.0%	100.0%

Table 4.18 Crosstab Crosstab between final diagnosis and finding post contrast:

			finding pos	Total			
			aneurysm		AVM +	no AVM	
			al	AVM	aneurys	or	
			dialatatio	7 1 1 1 1	m	aneurys	
			n		111	m	
final	AVM	Count	0	4	0	0	4
diagnosis	AVIVI	% of Total	.0%	8.0%	.0%	.0%	8.0%
	Rupture	Count	27	0	4	0	31
	aneurysm	% of Total	54.0%	.0%	8.0%	.0%	62.0%
	rupture	Count	1	0	3	0	4
	aneurysm+ AVM	% of Total	2.0%	.0%	6.0%	.0%	8.0%
	SAH left	Count	0	0	0	6	6

	side	% of Total	.0%	.0%	.0%	12.0%	12.0%
	SAH rigtht	Count	0	0	0	5	5
	side	% of Total	.0%	.0%	.0%	10.0%	10.0%
Terral		Count	28	4	7	11	50
Total		% of Total	56.0%	8.0%	14.0%	22.0%	100.0%

Table 4.19 Crosstab Crosstab between final diagnosis and finding association::

			Assoc	ciation						Total
			brain atrop hy	brain atroph y + IVH	classif ied coroid plexus	dialat ed ventri cles	IVH	mid line shift	unco munic ating hydro cephl us	
	AVM	Coun t	1	0	0	1	0	0	2	4
	AVIVI	% of Total	2.0%	.0%	.0%	2.0%	.0%	.0%	4.0%	8.0%
	Rupt ure	Coun t	10	2	2	7	8	2	0	31
	aneur ysm	% of Total	20.0 %	4.0%	4.0%	14.0%	16.0%	4.0%	.0%	62.0%
	ruptu re	Coun t	2	0	0	0	2	0	0	4
final diagnosis	aneur ysm+ AVM	% of Total	4.0%	.0%	.0%	.0%	4.0%	.0%	.0%	8.0%
	SAH left	Coun t	3	1	0	1	1	0	0	6
	side	% of Total	6.0%	2.0%	.0%	2.0%	2.0%	.0%	.0%	12.0%
	rigtht -	Coun t	2	1	0	0	2	0	0	5
		% of Total	4.0%	2.0%	.0%	.0%	4.0%	.0%	.0%	10.0%
Total		Coun t	18	4	2	9	13	2	2	50
Total		% of Total	36.0 %	8.0%	4.0%	18.0%	26.0%	4.0%	4.0%	100.0

Table 4.20 Crosstab Crosstab between final diagnosis and site:

			site (RT.	LT)	Total
			right	left	Total
	AVM	Count	3	1	4
	AVIVI	% of Total	6.0%	2.0%	8.0%
	Rupture	Count	19	12	31
	aneurysm	% of Total	38.0%	24.0%	62.0%
final	rupture	Count	1	3	4
diagnosis	aneurysm+ AVM	% of Total	2.0%	6.0%	8.0%
	SAH left	Count	0	6	6
	side	% of Total	.0%	12.0%	12.0%
	SAH rigtht	Count	5	0	5
	side	% of Total	10.0%	.0%	10.0%
Total		Count	28	22	50
10ldI		% of Total	56.0%	44.0%	100.0%

**P**-value=0.007

Table 4.21 Crosstab AGE2 \* final diagnosis Crosstabulation

			final diagnosis					Total
			AVM	Rupture aneurys m	rupture aneurysm + AVM	SAH left side	SAH rigtht side	
AGE	6-14	Coun t	2	0	0	0	0	2
		% of Total	4.0%	.0%	.0%	.0%	.0%	4.0%
	15-24	Coun t	1	0	0	0	0	1
		% of Total	2.0%	.0%	.0%	.0%	.0%	2.0%
	25-34	Coun t	0	8	2	0	0	10
		% of Total	.0%	16.0%	4.0%	.0%	.0%	20.0%
	35-44	Coun t	0	4	0	3	0	7
		% of Total	.0%	8.0%	.0%	6.0%	.0%	14.0%
	45-54	Coun t	1	6	0	1	2	10
		% of Total	2.0%	12.0%	.0%	2.0%	4.0%	20.0%
	55+	Coun t	0	13	2	2	3	20
		% of Total	.0%	26.0%	4.0%	4.0%	6.0%	40.0%
Total		Coun t	4	31	4	6	5	50
		% of Total	8.0%	62.0%	8.0%	12.0%	10.0%	100.0%

**P**-value=0.000

**Table 4.22 Crosstab Correlations between age and gender** 

			final diagnosi s	
		Correlation Coefficient	038	
	Gender	Sig. (2-tailed)	.792	
Spearman's		N	50	
rho		Correlation Coefficient	.291(*)	
	AGE	Sig. (2-tailed)	.041	
		N	50	

<sup>\*</sup> Correlation is significant at the 0.05 level (2-tailed). P-value=0.000

## **CHAPTER FIVE**

#### **5.1 DISCUSSION:**

The added value of CTA in the work-up of patients presenting with SAH. When CTA does not show an aneurysm, DSA will be carried out, and when CTA does detect an aneurysm, DSA is often required to decide whether or not endovascular treatment is possible. Therefore, only a CTA capable of providing sufficient information to both detect an aneurysm, AVM and allow for a reliable decision to be made about treatment. CTA is able to correctly guide treatment planning in the majority of cases, thus limiting the need for additional DSA. Few studies have described the use of MRA in assessing feasibility of endovascular treatment, and none make a direct comparison with CTA.

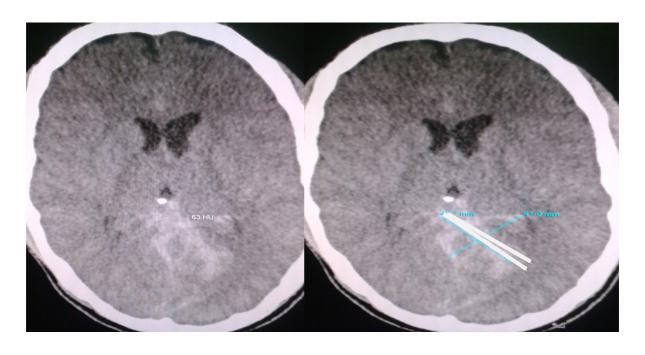
#### 5.2 Conclusion

The performance of CTA is very important in assessing and evaluation of the patient with SAH to detect aneurysms and AVM. Therefore CTA remains the modality of choice in patients presenting with SAH, it showed that the ultimate decision on whether an aneurysm can be coiled is very subjective. The information provided by non-invasive imaging techniques has an important function in the therapeutic decision process in patients presenting with SAH.

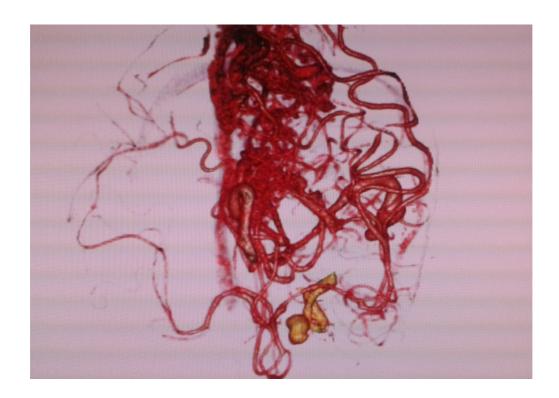
### **5.3 RECOMMENDATIONS:**

Patients with suspected subarachnoid hemorrhage should have a non-contrast CT scan as soon as possible after hospital arrival to confirm the diagnosis. Patients with subarachnoid hemorrhage should undergo vascular imaging of the brain. High-quality CT angiography may be preferable to catheter angiography as an initial investigation, Patients with subarachnoid hemorrhage should have an urgent consultation with a neurosurgeon.

# **Appendixes**



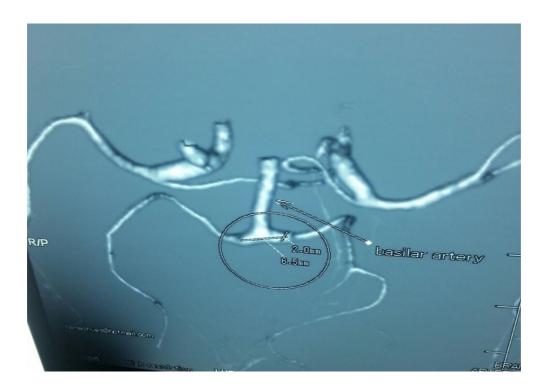
CT brain show the CT NO size & site IN patient with SAH



3D RECONSTRUCTION SHOW AVM + ANEURYSIM DIALATATION



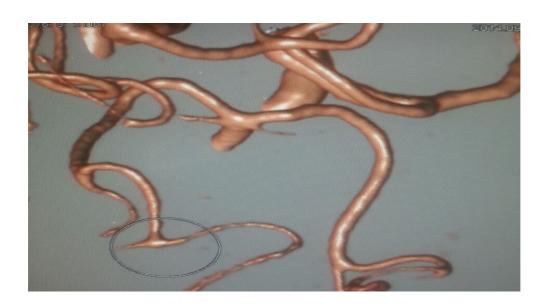
Image show AVM



3D RECONSTRUCTIONA SHAW CERICLE OF WILLS



3D RECONSTRUCTION SHOW THE COIL AFTER RUPTURE ANEURYSM



3D RECONSTRUCTION SHOW ANEURYSMAL DIALATATION

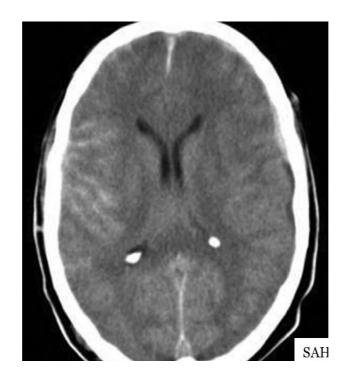


3D RECONSTRUCTION SHOW ANEURYSMAL DIALATATION



CT BRAIN SHOW SAH

CT BRAIN SHOW SAH + IVH



CT BRAIN SHOW SAH + BRAIN ATROPHY



3D ROCNSTRUCTION SHOW ANEURYSIM DIALATATION

# References

- . 1-Connolly, E.S., Jr., et al., Guidelines for the management of aneurysmal subarachnoid hemorrhage a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke, 2012. 2-Agid, R., et al., Acute subarachnoid hemorrhage: using 64-slice multidetector CT angiography to triage" patients' treatment. Neuroradiology, 2006.
- 3-Chen, W., et al., Application of multislice computed tomographic angiography in diagnosis and treatment of intracranial aneurysms. Clin Neurol Neurosurg, 2010.
- 4-Hoh, B.L., et al., Results of a prospective protocol of computed tomographic angiography in place of catheter angiography as the only diagnostic and pretreatment planning study for cerebra aneurysms by a combined neurovascular team. Neurosurgery, 2004..
- 5-Li, Q., et al., Evaluation of 64-section CT angiography for detection and treatment planning of intracranial aneurysms by using DSA and surgical findings. Radiology, 2009.
- 6-Papke, K., et al., Intracranial aneurysms: role of multidetector CT angiography in diagnosis and. endovascular therapy planning. Radiology, 2007.
- 7-Westerlaan, H.E., et al., Multislice CT angiography in the selection of patients with ruptured intracranial aneurysms suitable for clipping or coiling. Neuroradiology, 2007.
- 8-Papke, K. and F. Brassel, Modern cross-sectional imaging in the diagnosis and follow-up intracranial aneurysms. Eur Radiol, 2006.
- 9-W.H.v., et al., Diagnostic performance of contrast enhanced magnetic resonance angiography in detecting intracranial aneurysms in patients presenting with subarachnoid haemorrhage. EJMINT, 2012. Oct) Original article.
- 10- Blumenfeld, H.: Neuroanatomy Through Clinical Cases. Sinauer Associates, Sunderland (2002)
- 11- Borden, N.M.: 3D Angiographic Atlas of Neurovascular Anatomy and Pathology. Cambridge University Press, Cambridge (2007)
- 12-Carpenter, M.B., Sutin, J.: Human Neuroanatomy. Williams and Wilkins,

Baltimore (1983)

13-Grand, W., Hopkins, L.N.: Vasculature of the Brain and Cranial Base: Variations in Clinical Anatomy. Thieme, Stuttgart (1999)

14-Gray, H., Bannister, L.H., Berry, M.M., et al.: Gray's Anatomy: The Anatomical Basis of Medicine and Surgery, 38th edn. Churchill Livingstone, Oxford (1995)

15-Harnsberger, H.R., Osborn, A.G., Ross, J., et al.: Diagnostic and Surgical Imaging Anatomy: Brain, Head and Neck, Spine. Amirsys, Salt Lake City (2006) 16-Hendelman, W.J.: Atlas of Functional Neuroanatomy. CRC Press LLC, Boca Raton (2000) 10. Huber, P.: Cerebral Angiography, 2nd edn. Thieme, Stuttgart (1982)

- 17-Kretschmann, H.J., Weinrich, W.: Neurofunctional Systems. 3D Reconstructions with Correlated Neuroimaging. Thieme, Stuttgart (1998)
- 18 Kretschmann, H.J., Weinrich, W.: Cranial Neuroimaging and Clinical Neuroanatomy, 3rd edn. Thieme, Stuttgart (2004)
- 19-Lasjaunias, P., Berenstein, A., ter Brugge, K.G.: Surgical Neuroangiography: Clinical