INTRODUCTION

As outcomes from cardiac surgery have been more carefully studied, it is clear that even subtle neurological damage can produce unacceptable declines in physical and social function. Because the brain is such a complex organ, even small injuries may produce symptomatic, functional losses that would not be detectable or important in other organs. Regional hypoperfusion, edema, microemboli, circulating cytotoxins, or subtle changes in blood glucose, insulin, or calcium may result in changes in cognitive function, ranging from subtle to profound. A small 2-mm infarct may cause a disruption of behavioral patterns. Physiologic and physical function changes can pass unnoticed, be accepted and dismissed, or profoundly compromise the patient's quality of life. Move the lesion half a centimeter and the same volume lesion may result in a catastrophic stroke. Thus, the brain is the most sensitive organ exposed to damage by cardiac surgery and also the organ that, with the heart, is most important to protect***(David et al., 2011)***

Cardiac surgical procedures are life saving procedures for hundreds of thousands of patients every year. However, despite this clinical utility, many patients who undergo this surgery suffer neurological injury as a result. In addition to the morbidity and mortality caused by neurological injury, these

complications are associated with increases in hospital length of stays, costs, and admissions to rehabilitation facilities***(Johnet al., 2012)***

By far, themost feared neurological complication of cardiac surgeries is stroke, with an incidence of between 1% and 6%. However, subtle decreases in neurocognition and impairments in level of consciousness occur frequently in the early postoperative period and can be equally distressing for patients and their families. Impaired consciousness can lead to additional neurological sequelae including encephalopathy, delirium, and depression. It is believed that atherosclerotic emboli from the aorta and hypoperfusion in watershed brain territories are the principal causes of stroke following cardiac surgery. The pathogenesis of cognitive impairment is likely multifactorial and generally depends on whether the impairment occurs early or late after surgery***(Haglet al., 2001).***

Early deficits are likely related to microemboli, hypotension, general anesthesia, and inflammatory state initiated by cardiopulmonary bypass (CPB) while late deficits are likely related to increasing age, preoperative neurocognitive conditions, and vascular disease common for this group of patients ***(Rebecca et al., 2013).***

Understanding of the molecular and biochemical basis of neurologic injury following cardiac surgical procedures is an important stepping stone to the development of effective neuroprotective strategies. Several neuroprotective strategies are currently employed to prevent neurologic injury following cardiac surgery, these focus on prevention of hyperthermia (>37°C) during rewarming as well as the avoidance of rapid rewarming, use of arterial line filters, avoidance of hyperglycemia, minimization of direct cardiac and aortic manipulation, the use of alpha stat pH techniques in CPB, decreased levels of hemodilution, and avoidance of cardiotomy suction to minimize the risk of fat emboli. Although these techniques have resulted in a diminution of stroke and neurocognitive dysfunction, when these events occur, they can result in devastating complications for patients and families ***(Randall et al., 2013).***

The introduction of hypothermic circulatory arrest (HCA) has revolutionized the surgical treatment of this pathology. Because of its important place in the application of current clinical methods of cerebral protection, a thorough understanding of HCA is essential for planning a reliable and reproducible strategy of brain protection. ***(Hagl et al., 2001).***

AIM OF THE WORK

The aim of this work is to provide updated information about central nervous system protection especially brain for patients undergoing cardiac surgery as regards techniques and complication.

ANATOMY AND PHYSIOLOGY OF BRAIN BLOOD SUPPLY

Vascular supply of the brain

The brain, though representing 2% of the total body weight, it receives one fifth of the resting cardiac output. This blood supply is carried by the two internal carotid arteries and the two vertebral arteries that anastmose at the base of the brain to form the circle of Willis.

I-Arterial supply:

A-Anterior circulation:

- It consists of carotid arteries and their branches.
- It supplies anterior portion of the brain.

B-Posterior circulation:

- It consists of vertebrobasilar system.
- It supplies posterior portion of the brain.

(Sheldon, 1981)

The arterial supply to the brain is derived from the two internal carotid arteries (ICAs) and the two vertebral arteries, which unite first then divide again to form the two posterior cerebral arteries (PCAs). These vessels and the two internal

carotid arteries form an anastmotic system known as the circle of Willis at the base of the brain. The main arteries supplying the cerebral hemispheres are the anterior, middle and posterior cerebral artery for each hemisphere. the majority of cerebral aneurysms are of vessels that are part of, or very close to, the circle of Willis.Other important vessels supplying the brainstem and cerebellum are branches from the basilar artery ***(Alan et al., 2007)***.

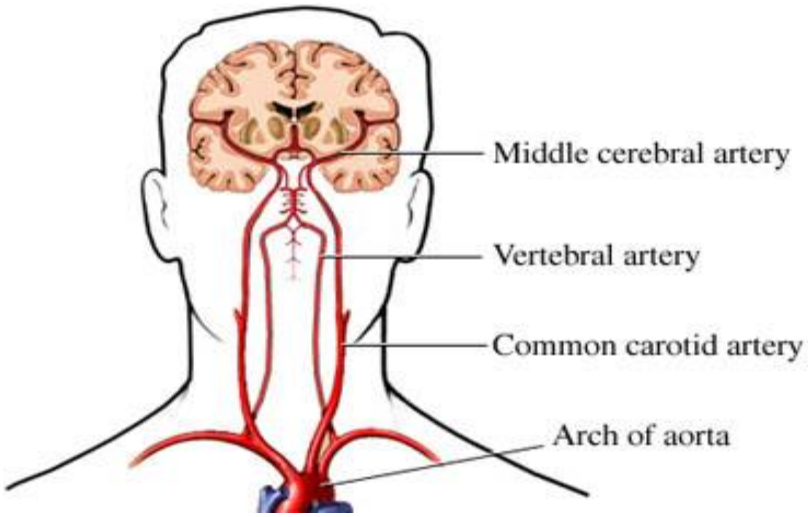

Fig. (1): The blood vessels supplying the brain**.**
(www. medicalook. com, 2007)

1-Carotid arterial system:

a-ICA:

i. Course:

Each ICA ascends along one side of the neck then they pass behind the ear in the temporal lobe and enter the

subarachnoid space. then, they run posteriorly to the medial end of the fissure of Sylvius where they bifurcates into two main branches, the anterior cerebral artery (ACA) and the middle cerebral artery (MCA) ***(Fitz Gerald, 1996).***

ii. Branches:

-The ACA:

It goes above the optic chiasma to the medial surface of the cerebral hemispheres. It arches around the genu of corpus callosum. It supplies blood to the medial cortex, including medial aspect of motor strip and the sensory strip. This means that damage to the anterior cerebral artery can cause sensory and motor impairment in the lower body. The ACA also delivers blood to some parts of the frontal lobe and corpus striatum. so a blockage in this artery can affect cognition and cause motoric problems.***(Fitz Gerald, 1996).***

-The MCA:

This large artery has-tree like branches that bring blood to the entire lateral aspect of each hemisphere. this means that this artey supplies blood to the cortical areas involved in speech, swallowing and language, including the lateral motor strip, lateral sensory strip, Broca`s area,

Wernicke`s area, Heschl`s gyrus, and the angular gyrus. in addition it provides most of the blood supply of corpus striatum.***(Alan et al., 2007)***.

If a patient has a blockage in the middle cerebral artey, it is probable that s/he will have aphasia. s/he will probably have impaired cognition and corticohyposthesia, or numbness, on the opposite side of the body. problems with hearing and the sense of smell may also result from injury to the artery because it supplies the lateral surface of the temporal lobe. The central branches of the MCA are the medial and lateral strial arteries, the strial artries supply the basal ganglia, internal capsule and thalamus ***(Fitz Gerald, 1996).***

Because they are the main blood supply to the internal capsule, they are called by some the arteries of stroke. when something happens to these arteries, the bottleneck of fibers within the internal capsule can be damaged, causing many disabilities. the striata are very thin arteries and pressure within it is high. for this reason, they are considered by many to be more vulnerable to hemorrhages than to blockage, although Fitz Gerald says that occlusion of one of these arteries is the major cause of classical stroke where pyramidal tract damage results in contralateral hemiplagia***(Henry Gray, 2000).***

- Other arteries which arise from the ICAs include:

Anterior communicating artery:

Joins the ACAs of each hemispheres together.

Posterior communicating arteries:

Join the MCAs to the posterior cerebral arterie, which are part of the basilar artery system ***(Fitz Gerald, 1996)***.

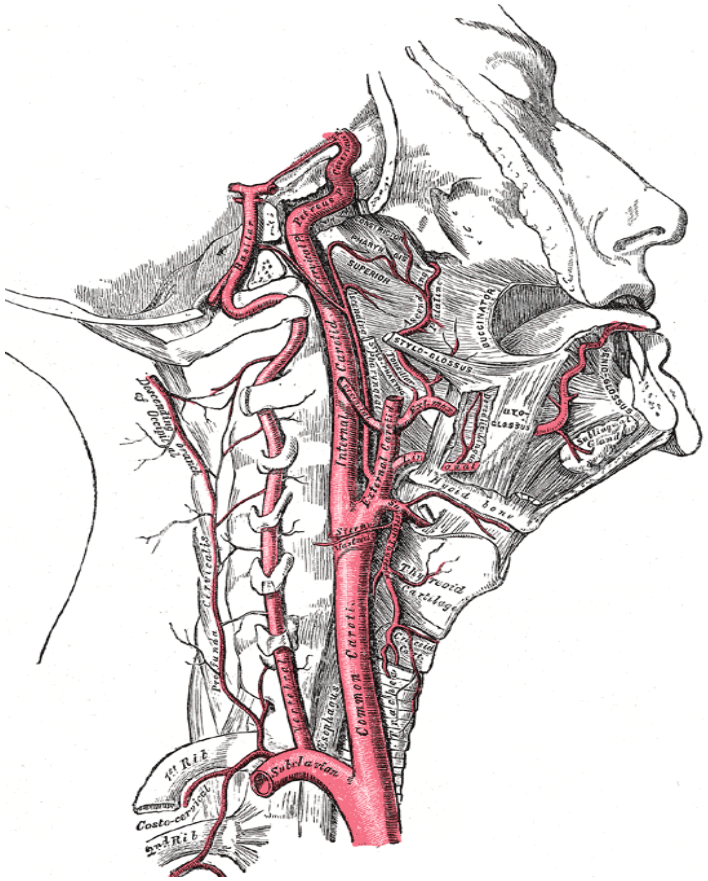

Fig. (2): The internal carotid and vertebral arteries
Right side ***(Henry, 2000)***

2- Vertebrobasilar system:

The vertebral arteries are branches of the subclavian arteries and pass through foramina in the transverse processes of the upper six cervical vertebrae. They join together anterior to the brainstem to form the single basilar artery.Both of the vertebral arteries ascend through the spinal column and enter the brain through the foramen magnum. Once in the brain, they continue to ascend, travelling beside the brainstem. ***(Alan et al., 2007)***.

a. The basilar artery:

At the lower border of pons the two vertebral arteries join together to form the basilar artery or vertebro-basilar artey.

The vertebral arteries and the basilar are straight arteries and therefore not as subject to blockage due to the build up of cholesterol as are the internal carotids***(Hirsch et al., 2004).***

b. Branches of the basilar artery:

i- The posterior inferior cerebellar artery (PICA): not only supply the cerebellum but also take blood to the lateral medulla, respectively.

ii- The anterior inferior cerebellar artery (AICA):gives blood supply to sides of the pons and the cerebellum. it has a branch, the labyrinthine artey, that supplies the inner ear.

iii- The superior cerebellar artery (SCA):gives blood supply to sides of the pons and the cerebellum.

iv- Twelve pontine arteries: they supply the medial pons ***(Fitz Gerald, 1996)***.

v- ***Posterior cerebral artery(PCA):***The basilar artery ends by dividing into the two PCAs, they encircle the midbrain close to the occulomotor nerve at the level of tentorium cerebelli and supply the inferior part of the temporal lobe and the occipital lobe ***(Marinkovic et al., 1987)***.

Many small perforating arteries arise from the PCA to supply the midbrain, the thalamus, hypothalamus and geniculate bodies ***(Fitz Gerald, 1996)***.

II-Venous drainage:

Venous blood drains into the cerebral venous sinuses, whose walls are formed from the dura matter. These sinuses join and empty into the internal jugular veins ***(Alan et al., 2007).***

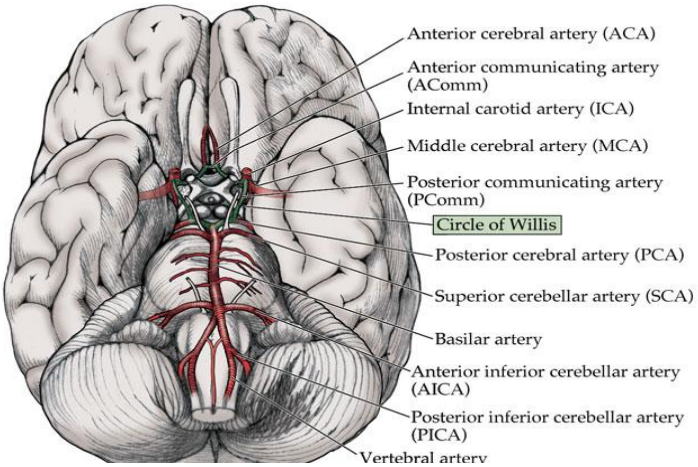

Fig. (3): Major arteries of the brain and formation of circle of Willis ***(Blumenfeld, 2002).***

Circle of Willis

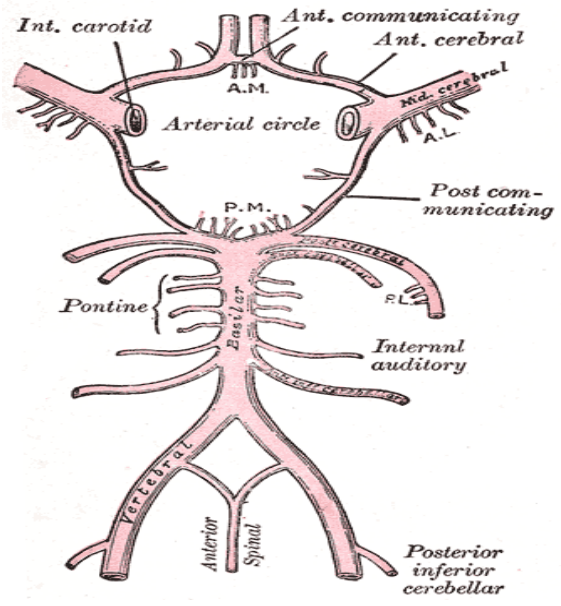

Fig. (4): The arterial circulation at the base of the brain. A. L. Antero-lateral. A. M. Antero-medial. P. L. Posterolateral. P. M. Posteromedial ganglionic branches ***(Henry, 2000).***

The circle of Willis or the circulus arteriosus is the main arterial anastmotic trunk of the brain.Anastmosis occurs when blood vessels bring blood to one spot from which it is then redistibuted.The circle of Willis is a point where the blood carried by the two ICAs and the basilar system comes together and then redistributed by the anterior, middle, and posterior cerebral arteries.The ACAs of the two hemispheres are joined together by the anterior communicating artery, the middle cerebral arteries are linked to the posterior cerebral arteries by the posterior communicating arteries.***(Bhatnagar and Andy, 1995).***

This anastmosis or communication between arteries makes collateral circulation which is defined as "the flow of blood through alternate route" possible. this is a safety mechanism, allowing brain areas to continue receiving adequate blood supply even if there is a blockage somewhere in an arterial system. the blood streams of the internal carotid system and basilar system meet in the posterior communicating arteries. If there are no problems in either system, the pressure of the streams will be equal and they will not mix. however, if there is a blockage in one of them, blood will flow from the intact artery to the damaged one, preventing a cerebral vascular accident ***(Love and Webb, 1995).***

As long as the circle of Willis can maintain blood pressure at fifty percent of normal, no infarction or death of tissue will occur in an area where a blockage exists. if collateral is good, no permanent effects may result from a blockage***(Fitz Gerald, 1996).***

Sometimes, an adjustment time is required before collateral circulation can reach a level that supports normal functioning; the communicating arteries will enlarge as blood flow through them increases. In such cases, a transient ischemic attack may occur, meaning that parts of the brain are temporarily deprived of oxygen. Some people lack one of the communicating arteries that form the circle of Willis, in this case if a blockage develops, collateral circulation will be impeded and the collateral blood supply will be compromised, causing brain damage to occur***(Alan et al., 2007)***.

There are some watershed areas in the brain located at the ends of the vascular systems. problems with blood supply are particularly likely to occur here, especially in those who have hardening of the arteries ***(Fitz Gerald, 1996)***.

Cerebral blood flow

The normal cerebral blood flow (CBF) is 45-50 ml/ 100gm/ min, ranging from 20ml /100gm/ min in white matter to 70 ml/ gm/ min in grey matter.***(Hirsch and Lawrence, 2004).***

There are two essential facts to understand about CBF:

- Firstly, in normal circumstances when the flow falls to less than 18-20 ml /100gm /min, physiological electrical function of the cell begins to fail.***(Henry Gray, 2000)***

- Secondly, an increase or decrease in CBF will cause an increase or decrease in cerebral arterial blood volume because of arterial dilatation or constriction. thus in a brain which is decompensated as a result of major intracranial pathology, increases or decreases in CBF will in turn lead to a significant rise or fall in intracranial pressure (ICP) ***(Chan et al., 1992)***.