ANAESTHESIA FOR CAROTID ENDARTERECTOMY

THESIS

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By

Raouf Ramzy Gadalla

M.B., B.Ch. (Ain Shams)

SUPERVISED BY :

Prof. Dr. Mohamed Hamed Shaker

Prof. of Anaesthesiology

AIN SHAMS UNIVERSITY

Dr. Mohey El-Din Waheed El-Din

Lec. of Anaesthesiology

AIN SHAMS UNIVERSITY

FACULTY OF MEDICINE

AIN SHAMS UNIVERSITY

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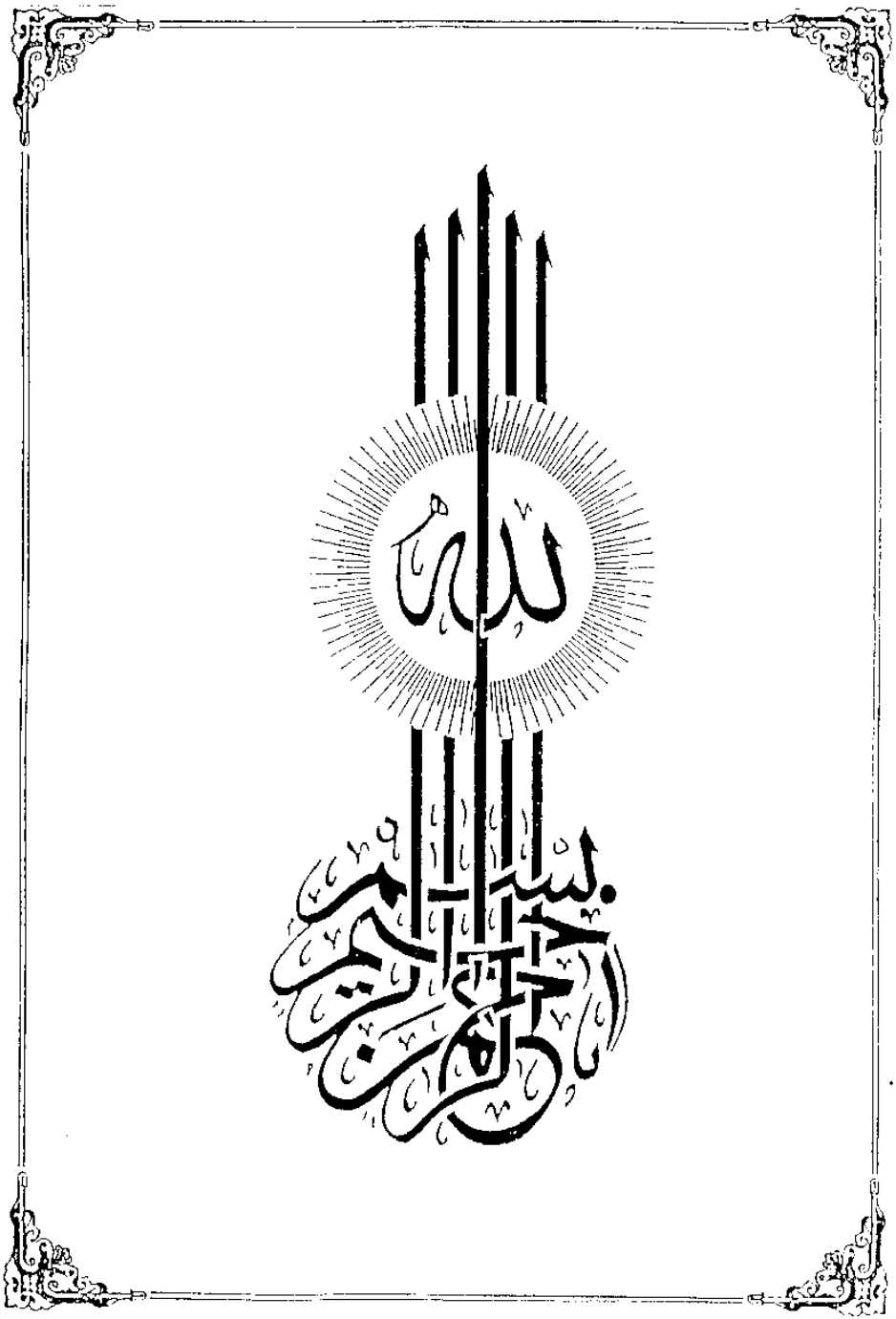

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INTRODUCTION

There is progressive increase of incidence of cerebral infarction as age advances. Sex distribution indicates that in men the rate is approximately 1.5 times as great as in women of the same age (Kuller, 1972). The chance of a recurrent stroke in the first year following initial cerebral infarction is 10 percent and the chance of a recurrent stroke in the first five years is 20 percent (Kuller, 1972).

A comprehensive study carried out by the members of the staff of the Mayo Clinic on Rochester, Minnesota population showed that the incidence of new transient ischaemic attacks amounted to 31 patients per 100,000 population per year for all ages with a rapidly rising incidence associated with advancing age. The incidence of new attacks in the 65 to 74 age group was 200 patients per 100,000 population per year. 36 percent of the patients with transient ischaemic attacks developed a stroke due to cerebral infarction (Matsumoto, 1970).

During the past thirty years, carotid endarterectomy for management of cerebro-vascular insufficiency has assumed an increasingly important role in the definitive management of those patients whose lesions are located in the extra-cranial vasculature (Thompson, 1982).

The most common extra-cranial arterial lesions which cause symptoms of transient cerebral ischaemia are internal carotid artery stenosis, vertebrobasilar insufficiency and lesions of the first part of the subclavian artery associated with subclavian steal syndrome (Moore, 1984).

The first reported successful carotid reconstruction was achieved in 1954 (Eastcott, Pickering and Rob, 1954) and since that time endarterectomy of a stenosed segment of the carotid artery has become a standard and successful method of treatment. Much knowledge has been gained, many new techniques have been developed and much progess has been made (Thompson, 1982).

The objectives of the operation are to remove the atheromatous plaque without leaving loose intima distally, to leave a smooth arterial wall and to achieve a blood-tight closure (Smellie, 1972).

The unique feature of carotid surgery is that, this procedure is done in patients suffering from cerebral ischaemia and requires, temporarily, the risk of increasing that ischaemia by clamping of the common,

internal and external carotid arteries during the actual removal of the plaque and during reconstruction (Fitch, 1976).

As with every operation, it is important that the benefits from surgery exceed the risks inherent in the procedure. If carotid endarterectomy is to be performed on patients with extracranial occlusive cerebro vascular disease who have not suffered a stroke, then every effort must be made to prevent ischaemic brain damage during the period of operation (Fitch, 1976).

These efforts entail two categories of major considerations. Firstly, monitoring cerebral oxygen delivery so as to recognize ischaemic episodes. Secondly, utilizing techniques which might protect (or at least not aggravate) the brain during periods of reduced cerebral blood flow (Michenfelder, 1984).

Complications following carotid endarterectomy have been reduced. Reports have attributed this reduction to better patient selection, improved surgical technique (Engell, 1973) and application of improved techniques for identifying patients who will require shunts under general or regional anaesthesia (Hays et al, 1972).

PHYSIOLOGICAL ANATOMY OF CEREBRAL CIRCULATION

As prevention of neuronal damage is essential in the anaesthetic management of all patients, awareness of the physiological aspects of cerebral circulation and metabolism is essential in patients with underlying central nervous system pathology especially when the operative procedure involves the brain or its vascular supply.

Neurons perform the primary function of the brain and the spinal cord which is transmission of nerve impulses. This electrical function is intimately related both to perfusion and to cerebral metabolism, which involves aerobic oxidation of glucose (Astrup, 1977).

Because of inadequate primary substrate stores (oxygen and glucose), neurons of the brain are highly dependent upon vascular perfusion for minute-to-minute delivery of these substrates. Oxygen delivery, if inadequate for O_2 demand causes neuronal hypoxia which can produce cell damage. Because neurons regenerate poorly, damage to these cells can be irreversible (Messick et al, 1985).

The arterial supply to the brain is derived from the internal carotid and vertebral arteries on each

side. Two-thirds of the supply come from the carotid vessels. The left common carotid artery arises directly from the aorta whereas the right is a branch of innominate artery. The vertebral arteries arise from the subclavian artery on their respective sides. The common carotid artery divides into external and internal carotid arteries. At its point of division the vessel shows a dilation, termed the carotid sinus, which usually involves and may be restricted to, the proximal part of internal carotid artery. In this situation the tunica media is thinner than elsewhere and the tunica adrentita which is relatively thicker contains a large number of sensory nerve endings derived from the glosso-pharyngeal nerve. The structure of the walls of the sinus enables it to react readily to changes in the arterial blood pressure and brings about appropriate modifications reflexly. Owing to its situation on the main artery of supply to the brain, its function as a pressor-receptor enables it to exercise control over intra cranial pressure (Davies and Davies, 1962).

The internal carotid artery supplies the eye through the ophthalmic artery and then divides into the anterior

and middle cerebral arteries which supply the anterior two-thirds of the cerebral hemisphere. The vertebral artery arises from the first part of the corresponding subclavian artery. It enters the skull through the foramen mangum and at the lower border of the pons joins the vessel of the opposite side to form the basilar artery, whose branches supply the brain stem and cerebellum, then divides into two posterior cerebral arteries supplying the posterior one-third of the cerebral hemisphere (Davies and Davies, 1962). The carotid arterial systems are inter-connected by the anterior communicating artery and are linked to the vertebrobasillar system by the posterior communicating arteries so forming the circle of Willis at the base of the brain. Fifty percent of the population have no posterior communicating artery (Alpers et al, 1959).

Kramer (1912) using a methylene blue technique, first suggested that the streams of blood passing to the circle of Willis do not normally mix but are distributed to sharply demarcated areas of the brain on the same side, and his work with cerebral angiography has confirmed this. However, flow across the

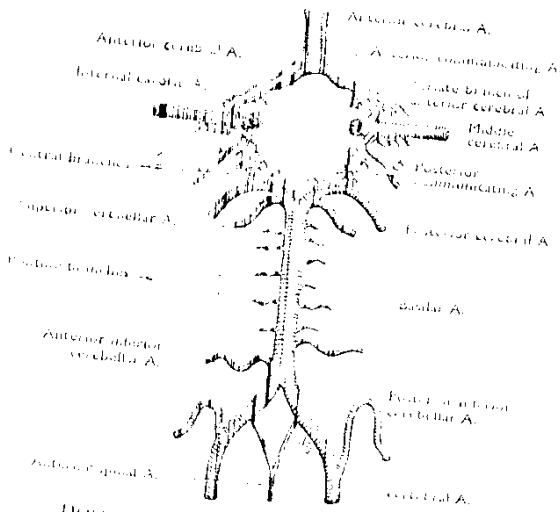

Diagram of the arteries at the base of the brain including the circulus arteriosus.

anastomatic communications does occur, and the importance of the circle of Willis was underlined by Brain (1957) who stated "the purpose served by the circle of Willis is to guarantee that, whatever the position of the head in relation to gravity and to the trunk, and however from one moment to another the relative flow through either carotid or vertebral artery may vary; these variations are always compensated for distal to those vessels and within the cranial cavity by the freest possible anastamsis before the brain is reached".

Another source of collateral cerebral blood flow is the communication between the external and internal carotid arteries through superficial temporal and ophthalmic arteries (Dritz, 1982). The brain can withstand relatively large decreases in the cross-sectional area of the carotid artery without symptoms of cerebral ischaemia (Brice et al, 1964). Even in presence of a complete unilateral occlusion cerebral blood flow is maintained because of this rich collateral vascular network (Symon, 1967).

Two general types of arteries supply the cerebral hemispheres; conducting vessels and penetrating vessels.