Ain shams University
Faculty of Medicine

ANAESTHESIA FOR NEUROSURGICAL EMERGENCIES

ESSAY

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The Master Degree in Anaesthesiology

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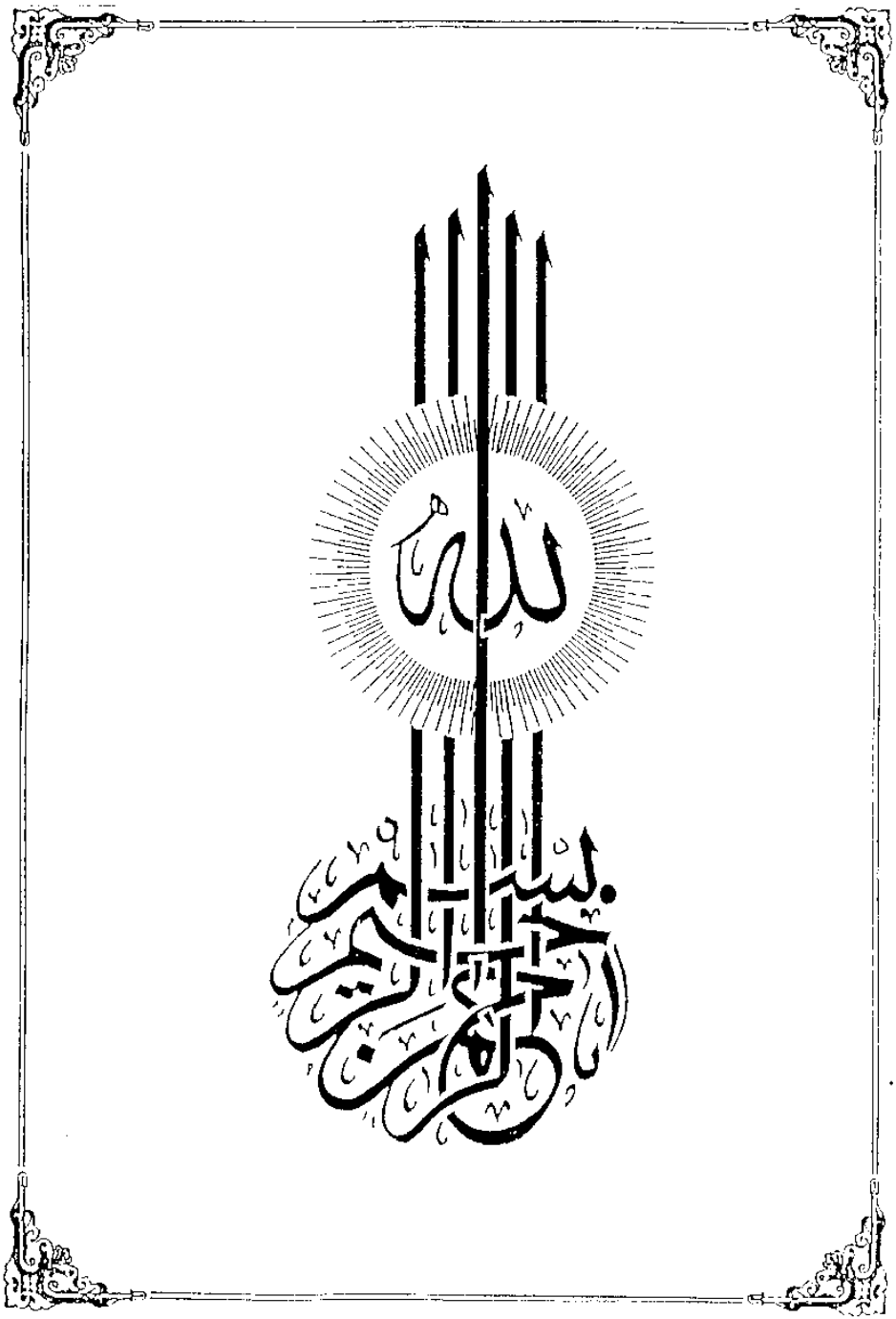
وَعَلَّمَكَ مَا لَمْ تَكُنْ تَعْلَمُ

وَكَانَ فَضْلُ اللَّهِ عَلَيْكَ عَظِيمًا

صَدَقَ اللهُ العَظِيم

سورة النساء آية ١١٣



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Introduction

Anaesthesiologists are usually involved in the management of neurosurgical emergencies whether due to head trauma or due to pathological cerebrovascular strokes.

Ruptured aneurysms, arteriovenous malformations, intracranial tumours, hydrocephalus or congenital malformations are examples of non traumatic head injuries.

Proper understanding of the pathophysiological changes associated with such conditions should help in their management.

Initial assessment, first-aid management, rapid preoperative preparation, choiced anaesthetic technique and postoperative intensive care should end in improving the outcome of this critical category of surgical emergencies.

PHYSIOLOGY & PHARMACOLOGY OF CEREBRAL BLOOD FLOW

Physiology and Pharmacology of Cerebral Blood Flow

A normal functioning, resting, normothermic human brain consumes oxygen at a rate of approximately 3.4 ml.100 g^{-1}. min^{-1}. Because the brain weigh 1200 to 1400 grams in the average adult, it consumes 40 ml O_2 per minute or 15 percent of the oxygen utilized by the entire body. Normally 35 percent of arterial oxygen content is extracted as blood traverses the cerebral circulation, resulting in 65 percent venous saturation in the jugular bulb. The high substrate requirements of the brain; together with its limited capability to store oxygen , contribute to its extreme vulnerability to hypoxic and ischaemic insults. Under normal circumstances, 40 to 50 percent of oxygen consumed by the brain is needed to maintain cellular integrity , while the remainder is used to perform electrophysiologic work (Prough and Rogers, 1989).

PHYSIOLOGIC REGULATION OF CEREBRAL VASCULAR RESISTANCE

Cerebral blood flow (CBF) is tightly controlled under normal physiologic circumstances by alteration of cerebral vascular resistance (CVR). CVR changes in response to changes in cerebral metabolic rate

of oxygen consumption (CMRO2), Pa O_2, Pa CO_2 and Cerebral perfusion pressure (Prough and Rogerss 1989).

CMRO2

CBF and CMRO2 are tightly coupled (i.e. CBF increases or decreases as CMRO2 rises or falls) assuming that other factors regulating CVR remain constant (Roland and Fribreg, 1985).

Although it is controversial, the most likely mechanism of tight coupling of CBF and $CMRO_2$ is the local release of adenosine (Berne et al., 1981).

Arterial Oxygen Content

Severe reductions in arterial oxygen tension (PaO_2) are associated with substantial increases in CBF (Fig. 1). CBF increases rapidly as PaO_2 is decreased below 60 mm Hg (i .e. below 90 per cent haemoglobin saturation). As with neural activation ,much of the change in CBF produced by decreases in PaO2 may be mediated by the local release of adenosine (Hoffman et al., 1984).

Decreases in haemoglobin concentration are associated with increases in CBF that offset approximately one half of the decline in oxygen content (i.e. if haemoglobin declines from 14 to 7 gm. , CBF will increase by 50 percent) (Poole et al., 1987).

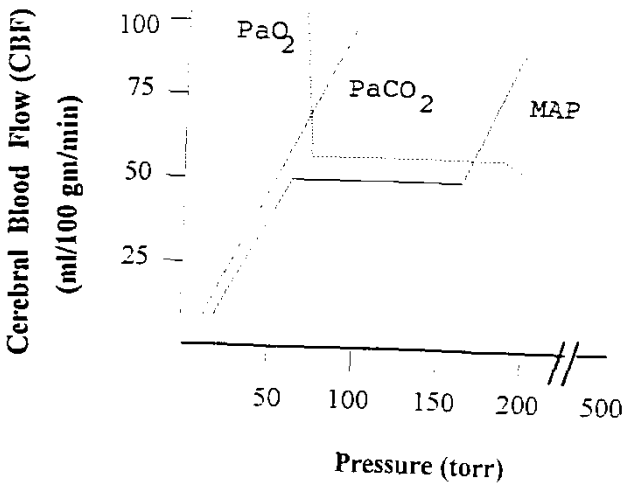

Figure 1. Effects of major physiologic variables on CBF. Arterial partial pressure of oxygen (PaO_2), arterial partial pressure of Carbon dioxide ($PaCO_2$), and mean arterial pressure (MAP) are three variables that exert important effects on CBF. PaO_2 below 60 mmHg (approximately 90 percent Hgb saturation) is a powerful vasodilator. $PaCO_2$ changes CBF by as much as 4 percent per mmHg change in $PaCO_2$. Pressure autoregulation describes the stability of CBF across a wide range of cerebral perfusion pressure (CPP) (Shapiro,1975).

$PaCO_2$

Because of the powerfull control of CVR exerted by changes in $PaCO_2$ (Fig. 1), hyperventilation has become one of the most commonly employed interventions to help for the acute reduction of intracranial pressure (ICP) in patients with intracranial hypertension. Changes in $PaCO_2$ appear to alter CVR by changing the hydrogen ion concentration of brain extracelluar fluid. Over the range of $PaCO_2$ from 20 mmHg to 80 mmHg , CBF is halved if $PaCO_2$ is halved, and doubled if $PaCO_2$ is doubled (Lassen, 1980).

Because rapid changes in $PaCO_2$ produce rapid changes in both CBF and cerebral blood volume, acute hypercarbia represents a major threat to patients with decreased intracrainal compliance, whereas acute hypocarbia frequently provides effective temporizing therapy in patients with intracranial hypertension. If $PaCO_2$ is abruptly changed and then maintained at a new level for 24 to 36 hours, CBF will return towards normal values as brain extracellular hydrogen ion concentration returns to normal (Severinghaus et al., 1966).

Arterial Blood Pressure And Autoregulation

Autoregulation is the term used to describe the maintenance of a constant CBF across a broad range of cerebral perfusion pressures. As a result of this physiologic control mechanism, termed pressure autoregulaton, cerebrovascular resistance varies directly with changes

in cerebral perfusion pressure (CPP) to maintain constant CBF. The generally accepted lower limit of autoregulation in healthy adult human is 50 to 60 mm Hg and the upper limit is 130 to 150 mm Hg (Fig.1), (Strandgaard and Paulson, 1984).

A cmmon cause of altered cerebral autoregulation is chronic hypertension, which shifts the lower and upper limits of autoregulation toward higher pressures (Barry and Lassen, 1984).

Although there are several proposed mechanisms for autoregulation, the most defensible now appears to be the myogenic theory, which postulates that an increase in wall tension within the cerebral vasculature produces a reflex increase in smooth muscle tone. The lower limits of autoregulation are in part determined by the physiologic or pharmacologic change that produces a reduction in blood pressure. Trauma and haemorrhagic shock shift the lower limits toward higher pressures (rendering the brain more vulnerable to hypotension) and also shift the upper limit, potentially causing cerebral hyperperfusion if hypertension is inadequately treated (Fitch et al., 1975).

PHARMACOLOGY OF THE CEREBRAL CIRCULATION

Diuretics

Diuretic drugs are usually given to patients with acute brain injury to reduce ICP and therefore to maintain CPP. The two diuretics most frequently used are mannitol and frusemide. Mannitol osmotically removes water from brain tissue. Frusemide also effectively reduces ICP without the transient increase in cerebral blood volume (CBV) associated with mannitol. Although frusemide has achieved some popularity in the management of intraoperative reduction of brain bulk , it is less frequently used in the critical care setting (Samson and Beyer, 1982).

Aminophylline

The bronchodilator theophylline which is commonly employed in patients who suffer from hypoxaemic lung disease , antagonizes the cerebral vasodilatory effects of hypoxaemia in animal models (Berne et al., 1981) .

Recently , Bowton and colleagues reported that theophylline reduced CBF by about 30 percent in patients with chronic obstructive lung disease (Bowton et al., 1987) .