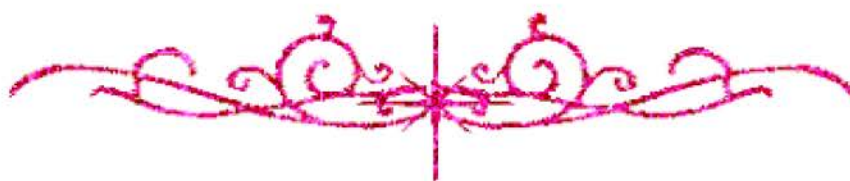


سامية محمد مصطفى



شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم



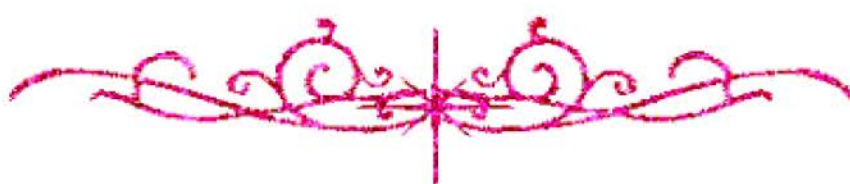
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شبكة المعلومات الجامعية



شبكة المعلومات الجامعية التوثيق الالكتروني والميكروفيلم



سامية محمد مصطفى



شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

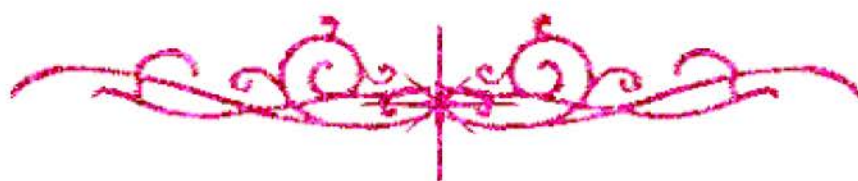
قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأقراص المدمجة قد أعدت دون أية تغيرات



يجب أن

تحفظ هذه الأقراص المدمجة بعيدا عن الغبار



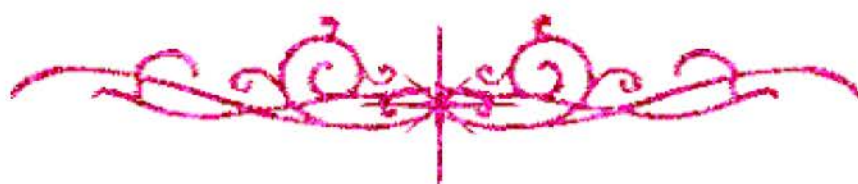
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بعض الوثائق الأصلية تالفة



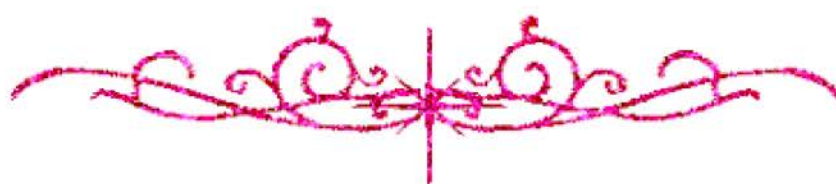
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شبكة المعلومات الجامعية



بالرسالة صفحات لم ترد بالأصل



THE EFFECT OF CARDIOPULMONARY BYPASS ON CEREBRAL METABOLISM DURING OPEN HEART SURGERY

THESIS

Submitted in Partial fulfillment for the Degree of MSc.

In

Anesthesiology

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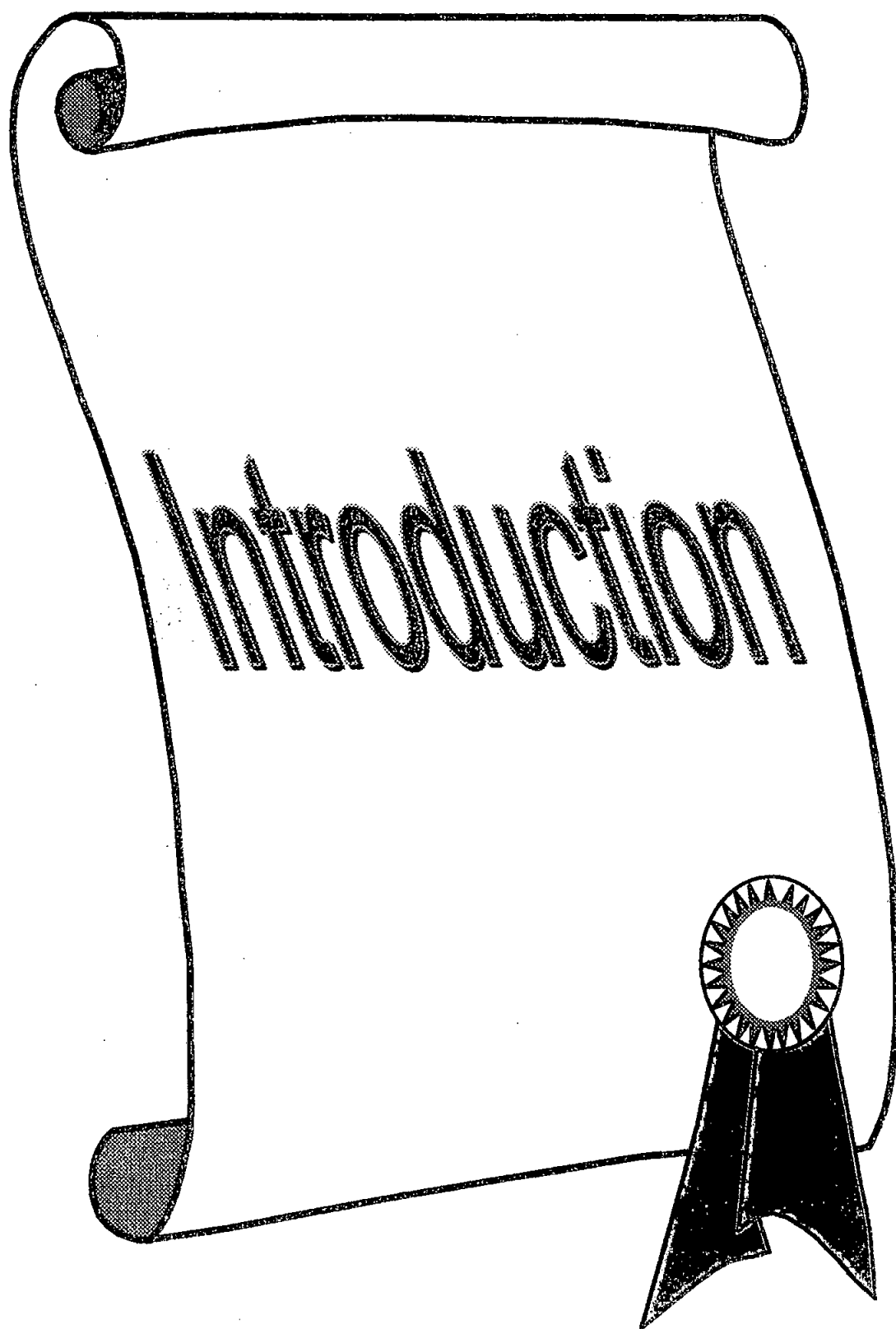
*First of all and above all, great thanks to **ALLAH** whose blessings on me can not be counted.*

*The sincerest thanks, deepest appreciation and greatest admiration to my **Prof. Dr. Kamal ElDeen Ali Heikal** Professor of Anesthesiology, Faculty of Medicine, Tanta University, for his constructive keen supervision, fruitful criticism, continuous support and encouragement to complete this work. He continuously advised me and spared no time or effort to offer his help and skill that made the completion of this work possible. I owe special feelings of gratitude and thanks to him.*

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INTRODUCTION

For patients experiencing cardiopulmonary bypass (CPB), overall cardiovascular morbidity and mortality have been declining steadily in recent years⁽¹⁾. However, because of changing population demographics, more elderly patients are now presenting for cardiac surgery. These older patients have a greater risk of stroke^(1,2). Those who suffer neurologic injury have a substantially prolonged hospital stay, with an attendant increase in cost⁽³⁾, that is why neurologic and neuropsychologic dysfunction are major risks of cardiac surgery⁽⁴⁾.

During cardiac surgery procedures mild hypothermia 25-30°C was induced to facilitate myocardial cooling and protection; such maneuver also provides cerebral protection while the brain is subjected to the insult of non pulsatile bypass^(5,6).

The major advantage to the hypothermia is a reduction in the metabolism and oxygen consumption. The mechanism for this reduction is quite complex and not entirely understood. At a biochemical level, hypothermia changes the reaction rate of all biochemical processes especially enzymatic reactions⁽⁷⁾.

Neuronal cells normally utilize glucose as their primary energy source. Brain glucose consumption is approximately 5mg/100g/min of which over 90% is metabolised aerobically.

Cerebral metabolic rate for oxygen therefore normally parallels glucose consumption, this relationship doesn't hold during starvation, when ketone bodies also become major energy substrates. The brain can also take up and metabolize some lactate⁽⁸⁾.

Hickey and Hoar have shown in human that a reduction in flow rate from 2.1 to 1.2 litres/min/m² at 25°C did not alter oxygen consumption or tissue perfusion⁽⁹⁾. It has long been established that temperature directly affects cerebral metabolism with experiments conducted in vitro⁽¹⁰⁾. and in vivo⁽¹¹⁾.

Review
Of Literature

REVIEW OF LITERATURE

CEREBRAL PHYSIOLOGY

The adult human brain weighs approximately 1350 g and therefore represents about 2 percent of total body weight, however, it receives 12 to 15 percent of cardiac output. This high flow is a reflection of the brain's high metabolic rate⁽¹²⁾. At rest, the brain consumes oxygen at an average rate of approximately 3.5 ml of oxygen per 100g. of brain tissue per minute, whole brain oxygen consumption ($13.5 \times 3.5 = 47$ ml/min) represents about 20 percent of total body oxygen utilization⁽¹³⁾.

Normal cerebral physiological values:

Cerebral blood flow (CBF):

Global	45-55 ml /100g/min
Cortical CBF (mostly gray matter)	75-80 ml /100 g /min.
Subcortical CBF (mostly white matter)	20 ml/100 g/min

Cerebral metabolic rate of oxygen (CMRO₂) 3 -3.5 ml/100 g/min.

Cerebral vascular resistance (CVR) 1.5-2.1mmHg/100g/min

Cerebral venous PO₂. 32-44 mmHg

Cerebral jugular oxygen saturation 55-70%

Intracranial pressure (ICP) Supine 8-12 mmHg

A large proportion of the brain's energy consumption, approximately 60 percent, is used to support electrophysiologic function⁽¹⁴⁾. The depolarization-repolarization activity that occurs and that is reflected in the EEG requires energy expenditure for the maintenance and restoration of ionic gradients and for the

synthesis, transport and reuptake of neurotransmitters. The remainder of the energy consumed by the brain is involved in cellular homeostatic activities, which include maintenance of the neurons relatively large membrane mass. Local CBF and local CMR within the brain are very heterogenous, and both are approximately four times greater in gray matter than in white matter. The cell population of the brain is also heterogenous in oxygen requirements⁽¹²⁾.

Cerebral blood flow:

The brain has more control over its blood flow and the distribution within the brain than any other organ, primarily because of autoregulation⁽¹⁴⁾. The anterior carotid circulation distributes four to five times the blood volume of posterior vertebrobasilar system, just as locally gray matter receives four times the blood flow of the white matter⁽¹⁵⁾, change in blood flow correlates with cortical neuron electrical activity as detected by surface electroencephalography and evoked potentials. Regulation of blood flow to specific areas of brain corresponds to metabolic demand in these areas⁽¹⁶⁾. Cerebral metabolic function is dependent on circulatory delivery of glucose and oxygen⁽¹⁷⁾. Aerobic metabolism of glucose provides basically all the energy required by neuronal tissue. Glycogen stores are lacking and ketones are metabolised only in extreme substrate deprivation. Local glucose metabolism is reflected by the local cerebral metabolic rate of oxygen consumption (CMRO₂)⁽¹⁸⁾. With ischemia or hypoxemia, the brain quickly depletes glucose and anaerobic metabolism occurs,

which inefficiently produces cellular energy substrates and locally increases lactic acid and H^+ ion⁽¹⁹⁾.

When there is neuronal injury, this autoregulatory coupling of metabolism and blood flow can be disrupted in brain tissue.

Factors affecting cerebral blood flow:

The brain substantial demand for substrates must be met by adequate delivery of oxygen and glucose. However, the space constraints imposed by the noncompliant cranium and meninges require that blood flow not to be excessive⁽²⁰⁾. Not surprisingly, there are elaborate mechanisms for regulation of CBF, which include many factors. While the quantitative effects of many of the factors that influence CBF are recognized, the precise mechanisms of these effects are not well understood. However our understanding is growing. A substantial volume of recent research indicates that modulation of arginine-nitric oxide (No), Cyclic guanosine monophosphate system (GMP)⁽²¹⁾, is central to the changes in the cerebral vascular tone caused by several processes. There is evidence that (No) is a mediator of cerebral vasodilatation caused by hypercapnia^(22,23), increased cerebral metabolic rate⁽²⁴⁾, volatile agents⁽²⁵⁾.

Chemical factors:

Cerebral metabolic rate:

Increased neuronal activity results in increased local brain metabolism, and this increase in CMR is associated with a well matched, proportional change in CBF⁽²⁶⁾. Regional CBF and CMR measurements performed in humans during maneuvers designed to