RECENT CONCEPTS IN THE ANAETHETIC MANAGEMENT OF PRE-ECLAMPSIA AND ECLAMPSIA

ESSAY

SUBMITTED IN PARTIAL FULFILMENT OF THE MASTER DEGREE IN ANAESTHESIA

BY

MOHAMED AHMED HANAFY MAHMOUD
(M.B., B.Ch.)

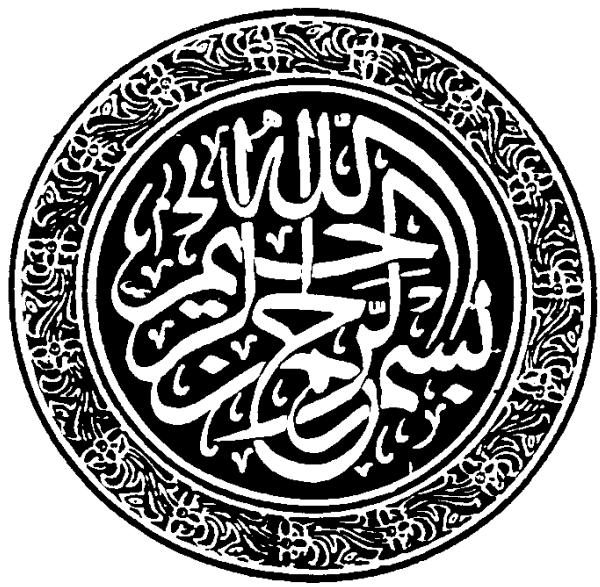
SUPERVISED BY

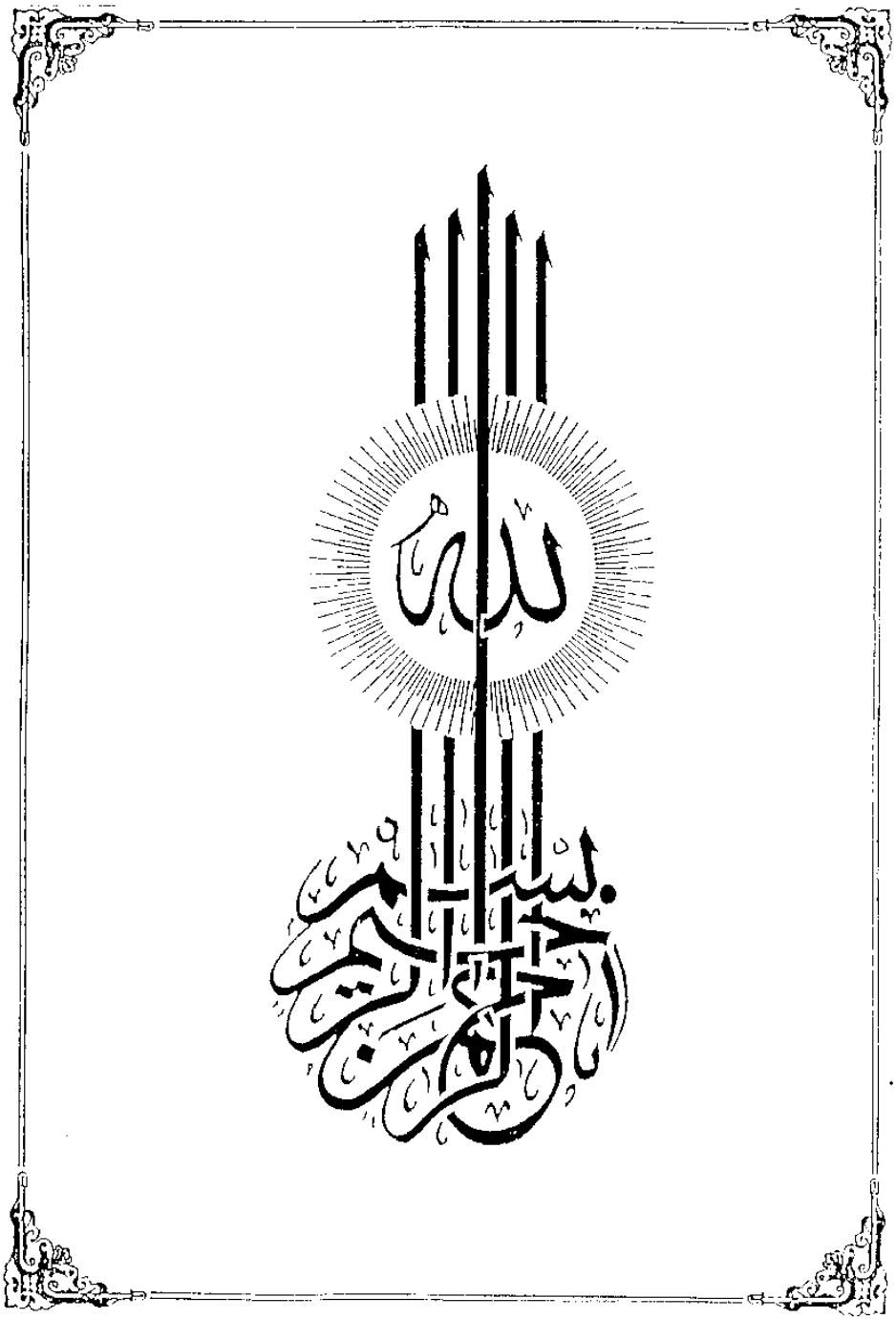
PROF. DR. ABD ELAZIZ MOHAMED YOUSSEF
PROFESSOR OF ANAESTHESIA
FACULTY OF MEDICINE
AIN SHAMS UNIVERSITY

PROF. DR. MADIHA METWALY ZEDAN
ASSITANT PROFESSOR OF ANAESTHESIA
FACULTY OF MEDICINE
AIN SHAMS UNIVERSITY

FACULTY OF MEDICINE
AIN SHAMS UNIVERSITY

1990





ACKNOWLEDGEMENT

I would like to express my grateful appreciation to **Professor Dr. Abd EL-Aziz Youssef,** Professor of Anaesthesia Faculty of Medicine, Ain Shams University, for his kind and fathful guidance throughout this work.

My profound thanks are due to **Professor Dr. Madiha Metwaly Zedan,** Assistant Professor of Anaesthesia, Faculty of Medicine, Ain Shams University for her great help and constructive encouragement throughout this work.

Finally, I wish to thank all my staff and colleagues in the Anaesthesia Department for their Kind help.

CONTENTS

INTRODUCTION

INTRODUCTION

The hypertensive disorders of pregnancy are an important topic for all who provide health to pregnant women. Hypertension increases the risk of death to both mother and fetus. In some areas of the world, infection and haemorrhage Complicating pregnancy are now cared for so effectively that the hypertensive complications constitute the leading cause of maternal mortality.

In the past the term "Toxaemia of pregnancy" has been used to include three distinct disease entities, which have a similar triad of hypertension, proteinuria and oedema, and which in certain circumstances, may progress to fits and coma (eclampsia). Since there is no evidence that any of the conditions is due to a toxin, the term is inaccurate. The term of toxaemia of pregnancy, is synonymous with preeclampsia and eclampsia, recently these conditions have been referred as pregnancy induced hypertension (Shnider and Levinson, 1987).

During pregnancy a blood pressure of 130/80 is the upper limit of normal at any time during pregnancy. Types of hypretension seen in pregnant woman:

- Pregnancy induced hypertension (PIH)

Pre-eclampsia a. Mild b. Moderate C.Severe

Eclampsia.

- Coincidental hypertension (Chronic hypertension Preceding Pregnancy): is persistent hypertension regardless of etiology before the 20th week of gestation or beyond 6 weeks after delivery.

- Chronic hypertension with superimposed PIH:

Superimposed preeclampsia.

Superimposed eclampsia

- Gestational hypertension (Late or transient hypertension of the third trimester): Hypertension without proteinuria or generalized oedema during last weeks of pregnancy or immediately after delivery. (Chesley, 1984).

Pregnancy induced hypertension is diagnosed when a pregnant woman has a blood pressure of 140/190 or greater, or a rise of 30 mm Hg systolic or 15 mm Hg diastolic over the baseline, or an increase in mean arterial pressure (MAP) of 20 mm Hg or MAP above 105 mm Hg. Blood Pressure has to be measured at least twice at Six or more hours apart. This patient also has abnormal oedema and significant proteinuria (Presence at 500 mg or more of protein in a 24 hour urine collection) after 20 weeks of gestation. (Shnider and Levinson, 1987).

Eclampsia is the occurrence of convulsions in a patient with PIH. A clear cut syndrome of preeclampsia neerly always precedes the convulsions, but most obstetricians have seen eclampsia to occur in woman who just a few hours earlier showed only modest hypertension and no proteinuria.

It is thus crucial to recognize case of PIH as a potential prelude to eclampsia.

The convulsion is grand mal in character and appears before, during or after delivery. Any seizure that occurs more than 48 hours post partum is due to other CNS lesions (Pritchard and Mac Donald, 1985).

The anaesthesiologist should plays a major role in the delivery of the toxemic patient, because of his experience in pain control, airway management, ventilatory care and monitoring and the significant effects that both toxaemia and its management may have on anesthesia, the anesthesiologist should be consulted early to assume a major role in the care of these patient, callig the anesethiologist just before delivery and instructing him to "take her down now" is unacceptable medical practice.

AETIOLOGY AND CLINICAL PICTURE

AETIOLOGY:

The cause of preeclampsia is not known but a number of deductions can be made from the available evidences. The presence of trophoblast is essential while a fetus is not, as preeclampsia can develop with hydatiform mole. Preeclampsia must therefore be associated with either an abnormality of trophoblast itself or of the maternal adaptation to the presence of trophoblast. The latter is more likely as there are a number of maternal-specific risk factors for the development of preeclampsia such as primigravidity, family history and underlying medical disorder. (Chun et al., 1964).

Fetal or paternal factors have been implicated (Need et al., 1983) but the evidence is circumstantial.

The various manifestation of preeclampsia are caused by either the presence of some abnormal circulating factors, or the absence of normal ones.

Placental ischemia is a central part of the process (Page, 1972) and could be the cause of preeclamptic syndrome. Placental ischemia could occur via a number of different mechanisms, such as the failure of the normal adaptation of

the spiral arteries occurring in early pregnancy.

In other situations, an excessive placental mass or sclerotic uterine vessels could result in placental ischemia.

Failure of normal trophoblast invasion of spiral arteries could be due to immunological factors. The hypothesis is that the maternal immune response to trophoblast needs to be down-regulated to permit normal invasiveness. The down-regulation may depend directly on immune responses generating "blocking antibodies" or suppressor cells (Redman, et al., 1984).

A number of the epidemiological features have provided circumstantial evidence for the importance of immunological mechanisms but so for there is little direct evidence.

The protective effect of first pregnancy is possible, but unconfirmed (Campbell et al, 1983). protective effect of previous abortion, and of blood transfusion (Feeney et al., 1977), the increased incidence in multigravidae who change partners (Feeney and Scott, 1980) or have donor insemination pregnancies (Need et al., 1983) are difficult to explain except on an immune basis. The assumption is that a beneficial (i.e. immuno-regulatory) response to fetal antigen occurs, and is absent in women developing preeclampsia (Scott et al., 1978).

If there is an inadequate maternal immune response to trophoblasts in preeclampsia, it is probably genetically determined. A familial factor is well established (Chesley, 1980). The incidence of severe preeclampsia can be explained by assuming that the affected women must be homozygous for a single recessive gene (Cooper and Liston, 1979).

Incidence:

Preeclampsia is mainly a disease of the primigravidous woman and is not usually a recurrent condition. The incidence in the multiparous woman is significantly lower than in the primiparous woman (Sibai, 1988).

The overall incidence of preeclampsia is 10% of all pregnancies (Goldkrand and Fuentes, 1986). It affects 6% of nulliparous pregnancies and many others complicated by chronic hypertension, renal disease and diabetes (Thomas, et al., 1989). While the incidence of eclampsia is 0.05-0.2% (Sibai et al., 1981b).

There has been a marked decline in the relative number of women with eclampsia over the last four decades with little change in the incidence of preeclampsia, this emphasizes the preventability of the convulsive phase.

Preeclampsia is also more common in black women than in white ones. Although the mechanisms for these racial differences are not clear, there is accumulating evidence to indicate that differences in cellular cation metabolism may partially be responsible for this racial differences (Cooper et la., 1987).

Clinical Picture and Laboratory data:

In general, pregnancy induced hypertension (PIH) is a disease of slow onset and steady progression, but occasionally it may arise quite suddenly and develop into eclampsia in a few days. For this reason the early signs must be sought at each visit. Three signs are usually considered to indicate the development of PIH, these are:

Hypertension, Proteinuria and Oedema.

Hypertension: A blood pressure of 130/80 mm Hg is the upper limit of normal at any time during pregnancy as recommended by the American Obstetric Committee. A diastolic blood pressure of 90 mm Hg is considered diagnostic. If the diastolic blood pressure exceeds 110 mm Hg severe PIH is diagnosed, and if exceeds 120mm Hg urgent action to reduce its level is indicated. The range of moderate preeclampsia is 150-169/96-109 mm Hg and of mild preeclampsia is less than 150/96 mm Hg.

Proteinuria: This is a sign that moderate or severe PIH has supervened. It indicates that renal spasm is marked. Small amounts of proteinuria (<300mg/L) may be found in normal pregnancy, but until PIH, chronic nephritis, and urinary tract infection have been excluded, the finding must