# HYPERTENSIVE DISORDERS IN ANAESTHESIA WITH SPECIAL REFERENCE TO ECLAMPSIA AND PREECLAMPSIA

**Essay**

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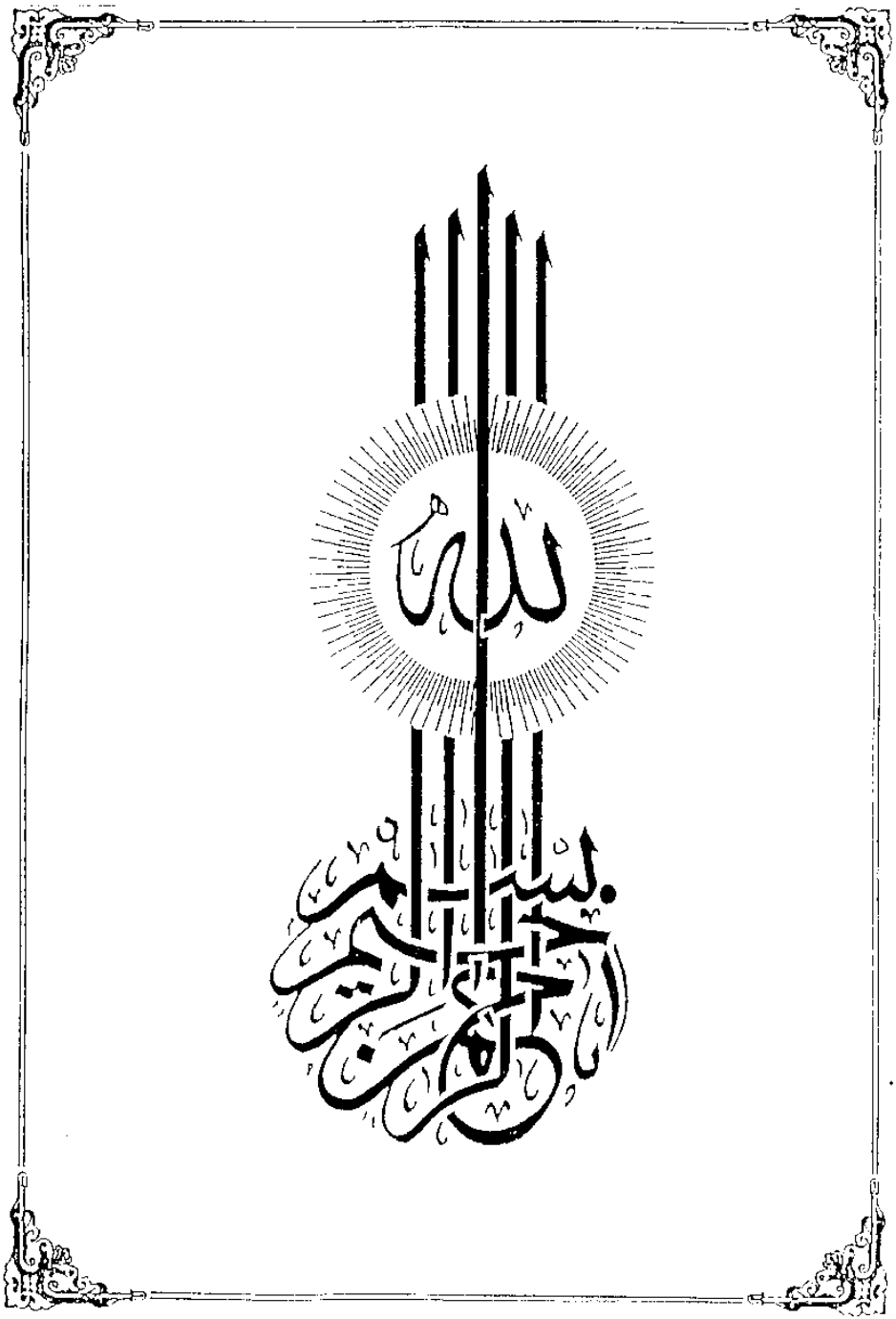

# Contents

# Introduction

# INTRODUCTION

Hypertension is the most common circulatory derangement. It is a significant risk factor for the development of ischemic heart disease and is a major cause of congestive heart failure, renal failure, and cerebrovascular accident. Variety of systems are involved in the regulation of arterial pressure such as, peripheral and/or central adrenergic, renal, hormonal, and vascular. Abnormalities in these systems can lead to hypertension. More than 90% of hypertensive patients have no definite cause and so called primary (essential) hypertension, whereas secondary hypertension has a demonstrable cause, renal disease is the most common cause of secondary hypertension.

The World Health Organization defined hypertension as a single sitting or recumbent blood pressure exceeding 160/95 mmHg. This elevation in blood pressure has broad implications for the anaesthesiologist because it denotes potential underlying structural and functional changes in various organs of the body. So proper understanding of the pathophysiological changes and effects of hypertension is important in the anaesthetic management. However, well assessed and well treated cases presenting for anaesthesia and surgery present no operative risk so long as hemodynamic stability is provided during the course anaesthesia, surgery and post operative period.

Preeclampsia as a hypertensive disorder of pregnancy, is one of the most outstanding problems in obstetrics and has been so far many decades.

Preeclampsia is mainly a disease of primigravidous women and is not usually a recurrent condition. Preeclampsia and eclampsia are among the leading causes of maternal morbidity

and mortality. Preeclampsia becomes apparent after 20 weeks of gestation and is characterized by the triad of maternal hypertension, proteinuria and generalized edema. With the occurrence of a grandmal convulsions, the condition is known as eclampsia, and the prognosis for both mother and fetus worsens.

Its etiology is still unsolved. There is no evidence that a toxin either initiates the disease or is an association of it. So, it is more logical to use the descriptive phase pregnancy induced hypertension rather than etiologic term such as toxemia of pregnancy.

It is important to realize that the underlying pathophysiology and management of preeclampsia and eclampsia has advanced rapidly during the last 10 years. Proper understanding of pathological changes in pregnancy induced hypertension is essential in the anaesthetic management. So the anaesthetist must be aware of the problems which result from the effect of the disease on the cardiovascular system, respiratory system, liver, kidney, and blood.

Anaesthetic complications are still a major cause of maternal mortality in obstetrics, especially in emergency and high risk cases, such as preeclampsia and eclampsia.

To reduce the maternal mortality rate from anaesthesia, emergency obstetric anaesthesia should be carried out by an expert anaesthetist, also with proper preoperative assessment and preparation of the patient.

So full compound anaesthetic - obstetric care is essential in most modern maternity units.

# Pathophysiology of Hypertension

# PATHOPHYSIOLOGY OF HYPERTENSION

## HYPERTENSION :

Hypertension is a disease process in which the patient has higher than normal blood pressure on more than one occasion. It is defined by two or more blood pressure measurements of greater than 160/90 mmHg. It may be categorized by diastolic and/or systolic hypertension.

## TYPES OF HYPERTENSION :

a) **Essential hypertension :**

In most patients (90-95%) no obvious etiology is present and the hypertension is called essential or primary. The physiological derangements which cause essential hypertension are not known. Abnormal sympathetic nervous activity and altered renin-angiotensin system regulators are implicated.

b) **Isolated systolic hypertension,** occurs as a result of the loss of elasticity of the arterioles with increased age. With advanced age systolic blood pressure increases while diastolic pressure decreases, so no changes occurs in the mean arterial pressure. Treatment of this type in the elderly has been recommended, as it reduces the incidence of strokes and coronary artery diseases *(Dahlof et al., 1991).*

c) **Secondary hypertension :**

In (5-10%) of hypertensive Patients, the hypertension is secondary to an underlying cause which is renal or endocrine in most cases. The endocrine causes of hypertension include, primary aldosteronism, cushing's syndrome, pheochromocytoma, and thyrotoxicosis. Also the

renal causes, include, renal parenchymal disease or renal artery stenosis.

Other infrequent causes of secondary hypertension include, coarctation of aorta, increased intracranial pressure, pregnancy, and oral contraceptives *(Domino, 1995)*.

## PATHOPHYSIOLOGY OF ESSENTIAL HYPERTENSION:

The underlying mechanism of this form of hypertension is unknown. Accelerated atherosclerosis is an invariable companion of this type of hypertension. Independent risk factors associated with the development of atherosclerosis (elevated serum cholesterol, glucose intolerance, and/or cigarette smoking) enhance the effect of hypertension on mortality, regardless of sex, age, or race. There is a positive correlation between obesity and hypertension. More than 50% of untreated hypertensives will develop end-organ disease, such as cardiomegaly, congestive heart failure, retinopathy, cerebrovascular accident, and renal insufficiency *(Wilson et al., 1991)*.

The primary difficulty in uncovering the mechanism(s) responsible for the hypertension in these patients is attributable to the variety of systems that are involved in the regulation of arterial pressure. Peripheral and/or central adrenergic, renal, hormonal, and vascular and to the complexity of the relationships of these systems to one another. Several abnormalities have been described in patients with essential hypertension, often with a claim that one or more of these are primarily responsible for the hypertension. While it is still uncertain whether these individual abnormalities are primary or secondary.

**INCREASED VASCULAR RESISTANCE:**

It is suggested to be the major hemodynamic alteration in hypertension. Which is achieved through an active increase in the state of tone of vascular smooth muscle in both arterioles and venules. This state of vessel tone can be achieved whether the myocyte is stimulatd by enhanced adrenergic input (elevated circulating levels of humoral agents e.g. catecholamines, angiotensin II, serotonine, or vasopressin.), local vasoactive peptides (e.g. angiotensin II, vasoactive intestinal poly peptide, endothelium), or ions e.g. (calcium); or by other causes. *(Froblich, 1983).*

Another factor that participates in the increased vascular resistance is an increased wall-to-lumen diameter of the arterial and arteriolar wall. This structural alteration of the vessel wall in hypertension serves to augment vascular responsiveness to constrictor stimuli, thereby serving to perpetuate the hypertension. Recent investigations have suggested that the hemodynamic stress of vessel stretch may not be the sole responsible mechanism accounting for the vessel wall thickening.

Recently it is found that, numbers of growth factors, such as platelet-derived growth factor, have been shown to participate in the process of vessel wall thickening in hypertension. Some of which are vasoconstrictors (e.g. angiotensin II, endothelin), and are produced within the arteriolar wall. These growth factors and vascular smooth muscle cell growth represent common features of hypertension and atherosclerosis *(Dzau and Gibbons 1991)*

**- Nitric oxide "NO" and hypertension:**

It is now recognized that, the vascular endothelium actively participates in the regulation of vascular tone through the release

of several factors that modulate the contractile activity of the underlying vascular smooth muscle. One of these factors is endothelium-derived nitric oxide NO which is a soluble gas that produces smooth muscle relaxation by activation of intracellular guanylate cyclase. NO is synthesized by the endothelial cells using the amino acid L-arginine as a substrate in a process catalyzed by the enzyme No synthase *(Moncada et al., 1991)*. The constitutive form of this enzyme, and therefore, the production of NO, can be stimulated by several agonists acting on different cell-surface receptors and using distincts intracellular signal transduction pathways *(Birnbaumor, 1990)*. The contribution of NO to the regulation of vascular tone in humans has been demonstrated by showing that inhibition of its synthesis results in significant vasoconstriction and reduction in the response to endothelium - dependent vasodilators. The critical role of endothelium in the regulation of vascular tone has been emphasized by the finding of abnormal endothelial function under several cardiovascular condition *(Panza et al., 1995)*.

However, patients with essential hypertension have blunted endothelium-dependent vasodilator responses to acetylcholine and bradykinin, largely due to reduced bioactivity of NO. The precise location of the defect responsible for this endothelial abnormality is uncertain *(Panza et al., 1994)*.

It was postulated that, acetylcholine-stimulated endothelium released a substance causing smooth muscle relaxation, which later became known as endothelium-derived relaxing factor (EDRF). It is demonstrated that EDRF and NO are identical : they possess the same biologic activity, stability, and susceptibility to an inhibitor and to a potentiator, so it is suggested that EDRF is NO *(Loskove and Frishman 1995)*. NO