Ain Shams University
Faculty of Medicine

MAGNESIUM AND CALCIUM LEVELS IN PREGNANCY INDUCED HYPERTENSION

A thesis
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of
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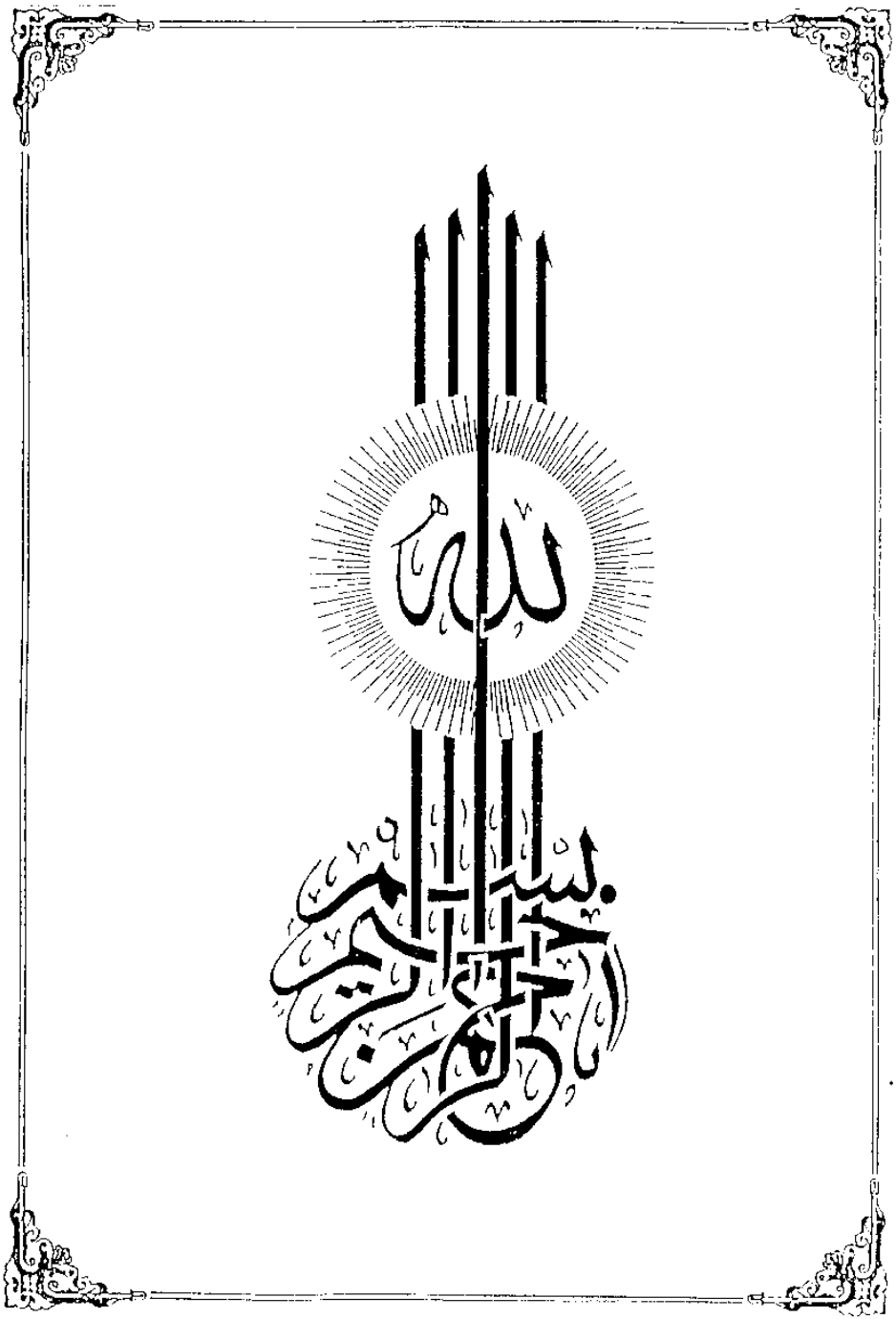
TO MY FAMILY



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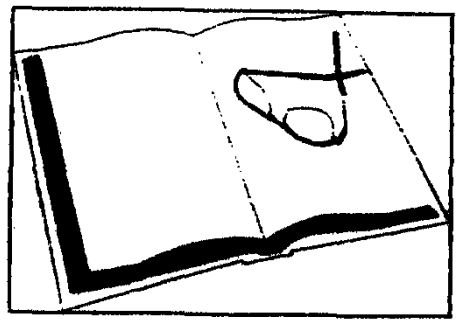


INTRODUCTION AND AIM OF THE WORK

INTRODUCTION

Preeclampsia-eclampsia remains one of the most commonly encountered pregnancy related disorder in Egypt.

It is a multifaceted syndrome with variable involvement of several organ systems. The classic triad of hypertension, oedema and proteinuria is still the most common presentation (Entman, 1983).

Till now and as stated by Zweifel, 1916, preeclampsia is the disease of theories. However, it is widely accepted that increased blood pressure is due to increased sensitivity of the blood vessel wall to the constrictor action of endogenous neurohormonal substances (for example: adrenergic amines, angiotensin and vasopressin). On the other hand, it may be due to decreased sensitivity of the blood vessels to endogenous vasodilators such as prostaglandins (Genest, 1977).

Calcium is known to play an important role in the excitation-contraction coupling of the vascular smooth muscle (Ganong, 1981).

Magnesium ions on the other hand, may act physiologically to regulate entry and exit of calcium (Altura, 1980). Several recent studies were conducted to

elucidate the role played by calcium and magnesium in the pathogenesis of pregnancy induced hypertension. Such studies are warranted since a confirmation of the association between calcium and/or magnesium status and this disease could have significant public health implications in the prevention and treatment of pregnancy induced hypertension.

After all, magnesium sulfate remains to be the drug of choice for this diffuse vasospastic disease.

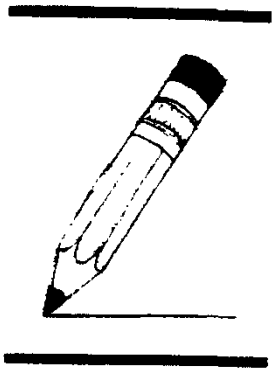
AIM OF THE WORK:

The aim of this study is:

a. To review the literature about calcium and magnesium role in the pathogenesis of pregnancy induced hypertension.

b. To evaluate and to compare calcium and magnesium levels in normal pregnancy and that complicated by preeclampsia-eclampsia.

c. Further, it holds a comparison between calcium and magnesium levels in hypertensive patients before and after receiving magnesium sulfate therapy.



Review of Literature

CARDIOVASCULAR CHANGES DURING PREGNANCY

Cardiac output changes:

In normal individuals pregnancy is associated with a rise in cardiac output of approximately 40%, i.e. from 3.5 L/min to 6.00 L/min at rest (Bader, 1955).

The time of this rise is still a matter of discussion. However, it is already accepted that cardiac output shows marked increase as early as the first trimester. What is disputed is whether the cardiac output falls at the end of pregnancy and, if so, by how much. During the last 10 weeks of pregnancy, Ueland demonstrated in 1969 a fall in the cardiac output of 4% to 20% of the non pregnant levels with measurements taken in the lateral position. More recent studies using Doppler estimation of aortic velocity and electrical impedance cardiography suggested a fall in the cardiac output to non pregnant levels at term (Davies, 1986).

The increase in the cardiac output is caused partly by an increase in the heart rate and partly by an increase in stroke volume (Clapp, 1985).

Oestrogens, prostaglandins, including prostacyclins together with a recently described potent vasodilator, calcitonin gene related peptide may contribute to the increase in cardiac output (Stevenson, 1986).

Blood pressure changes:

Arterial pressure falls during pregnancy, beginning in the first trimester, when the cardiac output is already rising and reaching a nadir in mid pregnancy (Mac Gillivray, 1967). Arterial pressure is determined by total peripheral resistance and cardiac output, so there must be a profound fall in systemic arterial resistance (Adams, 1961) in the first trimester because the uteroplacental circulation is not large enough at this time to affect total peripheral resistance.

During the third trimester, both systolic and diastolic readings slowly rise to about the pre-pregnant levels (Mac Gillivray, 1967). The measurements are however dependent on posture.

In the supine position, with vena-caval compression and reduced venous return, arterial pressure is maintained by reflex vasconstriction (Lees, 1967). The systolic pressure may fall nevertheless by more than 30 percent in 10 percent of cases (Holmes, 1960). There may be also aortic compression in the supine position (Beinarz, 1968). This can mimic a minor degree of aortic coarctation causing higher arterial pressures in the arm than in the leg. At least some cases of late gestational hypertension may be caused by this change alone (Scanlon, 1974).

The reduced peripheral resistance of normal pregnancy is associated with relative arterial refractoriness to the constrictor actions of exogenous angiotensin II but not noradrenalin (Gant, 1973). Reduced reactivity to Angiotensin II is already detectable at 8 weeks gestation and maximal at mid pregnancy (Gant, 1973). The mechanism of this blunted responsiveness has not been elucidated but it may be related to a balance between prostacyclin and thromboxane (Ylikorkala, 1986).

HAEMODYNAMICS OF PREECLAMPSIA

Before further discussion we should recall Ohm's Law which described the relationship between mean arterial pressure (MAP), cardiac output (CO) and systemic vascular resistance (SVR):
MAP = CO x SVR/80. Thus hypertension can be a result of elevated resistance or cardiac output.

Many researchers categorically state that vascular resistance is elevated in preeclampsia (Lindheimer, 1985). The available data do not support such a uniform picture. While some preeclamptic patients have cardiac output as low as 4.5 L/min with a systemic vascular resistance as high as 2300 dyne.cm.sec^{-5}, (Cotton, 1984). Others have cardiac