"Statistical study of pregnant females who underwent a cardiac surgery in Ain-Shams University Hospitals in the last 5 years, 1983-1987".

Thesis

Submitted in Partial Fulfilment For the Master Degree in Obstetrics and Gynaecology

By

Usama Abdel Samie Mohamed Elwan

M.B., B.Ch.

Supervised By

Prof. Dr. Ahmed Roshdi Ammar

Prof. of Obstetrics and Gynaecology

Dr. Gamal Abdel Salam Wafa

Lecturer of Obstetrics and Gynaecology

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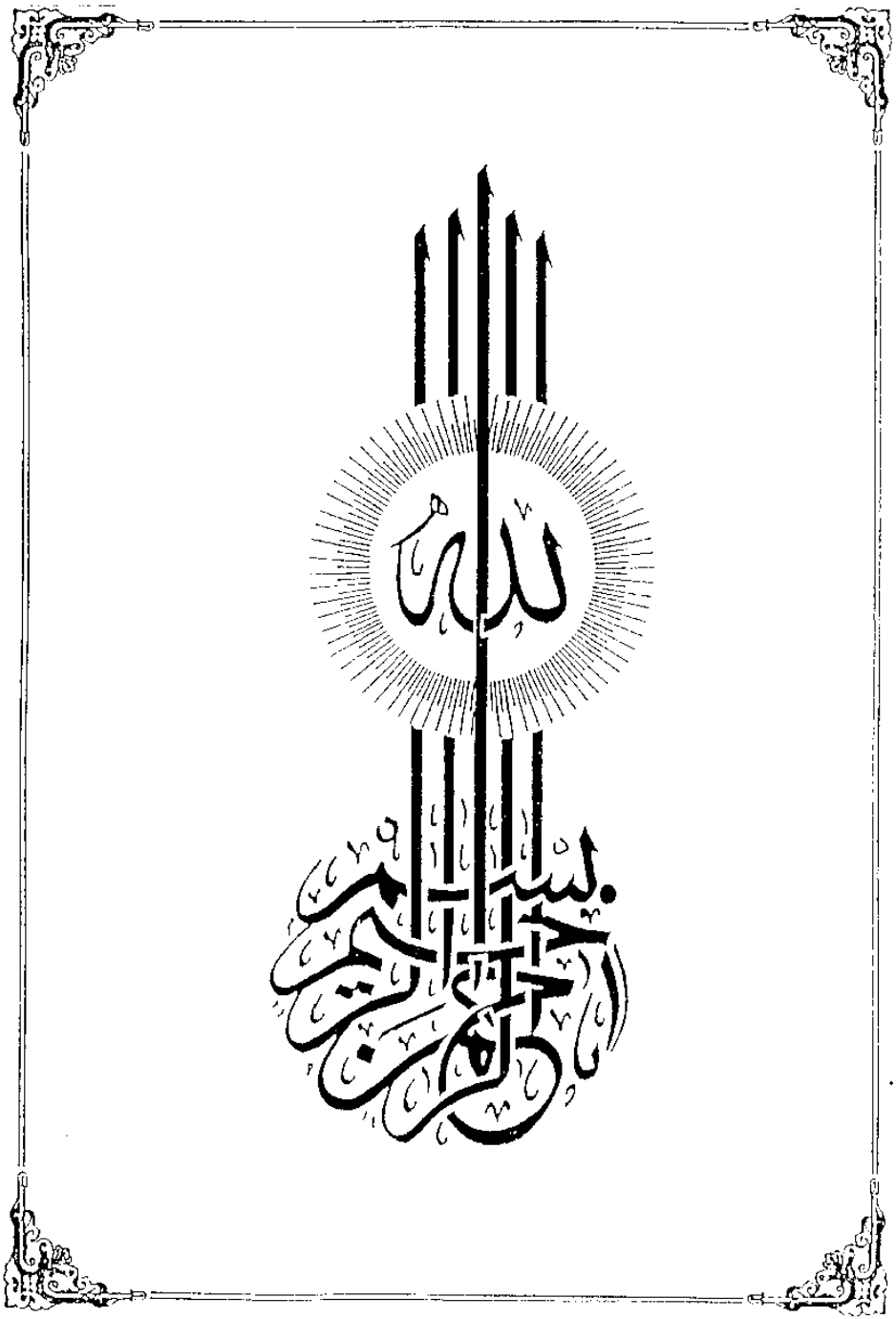

A C K N O W L E D G E M E N T

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Usama Abdel Samie

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C O N T E N T S

INTRODUCTION

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Many significant circulatory changes accompany pregnancy in some women with pre-existing cardiovascular disease, these alterations in hemodynamics can be dangerous. In a few others, events peculiar to pregnancy can occur, rendering them at significant risk of circulatory failure, a thorough understanding of these hemodynamic stresses is essential if the physician is to provide optimal care for the pregnant woman with heart disease **(Ueland, 1977).**

The advances in surgical management of patients with cardiac anomalies have increased the number of women with cardiac problem in the childbearing years.

In our developing country where the incidence of Rheumatic heart disease have not yet been reduced and where resources for its treatment are limited, there is still a place for simple operative technique, e.g. closed valvotomy, but open heart surgery and valve replacement operation are becoming quite common.

The likelihood of successful pregnancy after valve replacement depends on the type and site of the valve, the cardiac status of the woman and the need for anti-coagulants **(Bortolotti et al., 1982).**

REVIEW OF LITERATURE

PHYSIOLOGICAL CIRCULATORY ADJUSTMENTS DURING NORMAL PREGNANCY

A- Hemodynamic adaptations to pregnancy:

Cardiac output rises early in pregnancy and a significant elevation has been demonstrated by 12 weeks of gestation **(Barnes, 1970).**

It begins to increase as early as eight weeks' gestation to reach the level of 30 to 50 percent above values in non pregnant levels; it remains elevated to this degree throughout the rest of pregnancy **(Katz et al., 1978).**

If cardiac output is measured in the left lateral recumbent position, it will be shown to increase until approximately 36 weeks' gestation, at which time the output will stabilize and remain at its highest level until delivery **(Burch, 1977).**

A change in maternal position from supine to lateral produced a rise in C.O.P. of only 8% at 20-24 weeks of gestation, 14% at 28-32 weeks and 29% at term **(Walters et al., 1966).**

Scott and Kerr, (1963), convincingly demonstrated by radiographic means that the posture related hemodynamic changes were produced by uterine compression of the inferior vena cava, which resulted in marked diminution in venous

return to the heart, and a fall in cardiac output, even in women with adequate venous collaterls.

In a few women, presumably those whose collaterals are not well developed, maintenance of the supine position may result in alarming hypotension and bradycardia, a vaso-vagal syndrome which has been called the "supine hypotensive syndrome of pregnancy" **(Kerr et al., 1964).**

Heart rate increases by approximately 10-15 b.p.m. during pregnancy. The peak change occurs in most maternal positions near term. Stroke volume, on the other hand appears to be highest in early to mid pregnancy and then declines progressively to term, reaching levels at or below those of the non-pregnant individual. Thus early in pregnancy the increase in resting cardiac output is due mainly to an increase in stroke volume, a variable which falls later in pregnancy coincident with the progressive elevation in heart rate **(Ueland and Hansen, 1969 a).**

Blood pressure decline near the end of the first trimester and generally through the mid-trimester of pregnancy. The diastolic fall is greater than the systolic and results in a rise in pulse pressure. The change is accentuated in the supine position. Both total peripheral vascular resistance and mean arterial blood pressure reach their lowest level at the time the C.O.P. reach its peak **(Adams, 1954).**

Burch, (1977), demonstrated that blood pressure remains relatively unchanged, and those observations in which the blood pressure decreases during the second trimester of pregnancy were often made with the patient in the supine position and are artifactual. When measured in the left lateral recumbent position , blood pressure remains relatively constant throughout pregnancy.

One further physiologic consideration should be noted in pregnancy, a significant portion of the increase in cardiac output is delivered to the uterus via the uterine arteries. This flow is delivered largely to the placental bed. At this point large arterioles open directly into the placental Lacunae, where the maternal blood comes into contact with the foetal placental membrane. These maternal Lacunae are quite large and result in a shunting of blood from arterioles to venules without passage through a capillary bed. Perhaps because of the action of the myometrium, most women do not have symptoms consistent with a large arteriovenous shunt. However, symptomatic arteriovenous shunting is occasionally noticed and in such women the condition may deteriorate with high-output cardiac failure **(Burch, 1977).**

By the end of the first trimester, uterine flow has increased by 50 to 100 ml/min. over that of the non-pregnant

state. It increasesto 200 ml/min. by 28 weeks of pregnancy and reaches 1200 ml/min. above the baseline at term, in twin pregnancies uterine flow is even higher **(Assali et al., 1960).**

This progressive expansion of uterine blood flow is out of phase with the total rise in resting maternal cardiac output, which reaches its peak in the middle trimester **(Greenberg et al., 1982).**

When redistribution of flow is required in order to serve the mother, uterine blood flow falls. Excitement, heat, exercise, or decrease in venous return have all been shown to result in decreased uterine blood flow. Though exercise may temporarily divert blood flow away from the pregnant uterus, it is not apparent that this transient diversion has any deterimental effect on the foetus **(Roman-Ponce et al., 1978).**

The hemodynamic responses of a pregnant woman to exercise are somewhat different from those of non-pregnant women. For a given level of exercise in sitting position there is a greater increase in oxygen consumption compared to that of the non-pregnant state. This is associated with a relatively smaller rise in arteriovenous oxygen difference than that produced by the same exercise in non-pregnant women **(Ueland et al., 1973).**

The increment in blood volume during gestation varies considerably in different individuals, ranging from 20-100% **(Ueland 1976).**

It increases most rapidly during the first 20-30 weeks of pregnancy, but it continues to rise gradually to term **(Lund and Donovan, 1967).**

On occasion, the increase in plasma volume exceeds that of the red cell mass, and there is a slight decline in hemoglobin values. However, this phenomenon appears to be entirely dependent on the availability and utilization of iron. Patients treated prophylactically with oral iron do not have a significant decline in hemoglobin concentration late in pregnancy **(Hytten and Leitchi, 1971).**

Ikard et al. (1971), found evidence of obstruction to venous return from the lower extrimities throughout the whole of pregnancy with some variation in the position of the patient, and this increased venous pressure in the lower limbs is an important factor in development of ankle oedema and varicosities.

Oedema of the ankle and legs is a common feature in normal pregnant women and is in the absence of cardiovascular or renal disease of little pathological significance **(Szelky, 1974).**

Roberts, (1970) studied the natural history of oedema during pregnancy and found clinical oedema in 83% of women who had no evidence of renal or cardiac disease and were considered healthy at the onset of pregnancy.

Hytten and Robertson (1971), found a direct relationship between the degree of oedema and the increase in body water, between 10-38 w. The gain in body water was 6.8 Kgm in women with no oedema, 7.2 Kgm with oedema in the legs and 9.8 Kgm with generalized oedema. It can be attributed partly to high venous pressure in lower limbs and partly to reduced colloid osmotic pressure of plasma.

B- Hemodynamic alterations during Labor, Delivery, and the early puerperium:

The cardiovascular response to uterine contractions is influenced by two major factors - the maternal posture and the site where the measurements are made.

Ueland and Hansen in (1969 a), compared the hemodynamic responses to contractions in patients, both supine and in lateral recumbency, during the first stage of labour and prior to the administration of analgesia and anesthesia. It is of interest to note that, the cardiac output is very similar in both groups at the peak of uterine contraction - 6.522 liters supine and 6.830 liters in lateral position. The basic difference lies in the fact

that the cardiac output is considerably lower in the patients who are supine between contractions, a reflection of inferior vena caval obstruction and diminished venous return. These direct measurements were made from catheters inserted into the brachial artery and superior vena cava.

If on the other hand, measurements are taken from the femoral artery, there is a marked but transient hypotention in the supine position during contractions **(Metcalf and Ueland, 1974).**

Bieniarz et al. (1968), using arteriography, clearly demonstrated complete occlusion of the aorta and common iliac vessels at the height of uterine contractions in the supine position. Thus, the assumption of the supine position during labor produces a transient but complete obstruction of both the venous and arterial circulations and results in significant flactuation in matenral hemodynamics. As labor progresses, there is a progressive rise in cardiac output in patients receiving only local anesthesia during labor "paracervical block" and pudendal block for delivery. This steady rise occurs between contractions and is associated with a further increment during contractions.

The rise in cardiac output is considerably less throughout labor in patients receiving caudal analgesia