Heart Disease and Pregnancy

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HEART DISEASE AND PREGNANCY

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Preface

OUR aim is to present an account of the main cardiovascular problems occurring in obstetric practice as has been observed by us at the Newcastle General Hospital over three decades between 1942 and 1971. The observations are based on a study of over 1000 patients with various cardiovascular disorders who have been under our personal care in one or more pregnancies.

As rheumatic heart disease has been and still is numerically and by its nature the most important cardiac disease in relation to pregnancy, more than half of the text is devoted to this section. Many of the patients with rheumatic heart disease have been under observation for years before they reached the childbearing age. It is our intention indeed to underline the point that rheumatic heart disease is a lifelong disorder, and the course of the disease and the quality of its management from its onset throughout the years preceding pregnancy cannot be dissociated from events occurring during pregnancy. In this context, we present the state of pregnancy as a transient natural condition with haemodynamic implications, and discuss the complications encountered in pregnancy in the light of the natural history of rheumatic heart disease. It is for this reason that we have decided to include in this section a brief general review of the nature of rheumatic fever, and of the evolution and preventive aspects of rheumatic heart disease as being relevant to problems occurring later in the childbearing age and in pregnancy.

We have drawn attention to the changing pattern of rheumatic heart disease which is due partly to prophylactic measures, partly to the introduction of cardiac surgery, and more recently also to the natural decline in the severity of the disease. This changing pattern is clearly reflected in the decreasing incidence of serious complications encountered in pregnancy in recent years.

It has been fully appreciated that events like dysrhythmias, thrombo-embolism and infective endocarditis constitute clinical entities with diverse underlying pathology. However, in practice they have been observed mainly in association with rheumatic heart disease. We have, therefore, discussed them in the chapter on chronic rheumatic heart disease in order to present a comprehensive clinical picture of the disease.

The clinical pattern of congenital heart disease as seen in pregnancy has also changed over the years. Since the introduction of palliative or curative cardiac surgery more and more women have been reaching the childbearing age in a more favourable condition to tolerate the haemodynamic burden of pregnancy.

In the assessment of the patient's cardiac status in pregnancy emphasis has been laid on the evaluation of objective signs for the purposes of immediate management. The method of functional classification has been entirely discarded as misleading.

This monograph is primarily intended for readers at postgraduate level. However, it is hoped that it will also be of use to the experienced physician and obstetrician, whose everyday practice does not include the assessment and care of pregnant patients with cardiac disorders, and who are only occasionally called upon to deal with cardiac emergencies during pregnancy.

We owe a special gratitude to the late Professor Sir William Hume whose help was invaluable at the 'birth' of the cardiac antenatal clinic at the Newcastle General Hospital in 1942, and also in the early years of our study. The year 1942 also marks the start of a close collaboration between the Departments of Paediatrics and Cardiology. Children with rheumatic carditis were seen and have been followed up jointly. Many of them have remained under our observation in their adolescent and adult lives and form part of the material we have analysed. For this experience we are greatly indebted to Dr George Davison.

It is a pleasure to acknowledge our indebtedness to our colleagues in the Departments of Cardiology, Medicine and Obstetrics at the Newcastle General Hospital, and indeed to many colleagues in other hospitals in the Newcastle region who have helped us in many ways. We are particularly grateful to Drs W. G. A. Swan, Frederic Jackson and C. B. Henderson of the Department of Cardiology, and to Mr Hugh Arthur, Miss Dorothea Kerslake and Mr John Lawson of the Department of Obstetrics, who shared with us their knowledge and have entrusted to us the management of a number of their patients while pregnant. Thanks to the generosity of many colleagues we have seen in consultation a number of pregnant patients with heart disease who do not form part of the personal statistical analysis, but have provided us with further experience which we have fully utilized in the management of our own patients.

The authors of numerous fundamental studies on cardiovascular disorders in relation to pregnancy have been a constant source of inspiration to us and have influenced us through their writings and often also through personal contact. There are many in this category, but we would like to mention specially the late Dr C. S. Burwell, Drs James Metcalfe, Richard Turner, Curtis L. Mendelson and the late Harold Gorenberg.

We have fully appreciated the nature of the circulatory changes which take place during pregnancy and their importance for the management of the pregnant patient with heart disease. However, in view of our very limited personal experience in haemodynamic investigations during pregnancy, we have constantly referred to original works, especially to those of Professor Frank E. Hytten and his associates of the M.R.C. Reproduction and Growth Unit, Princess Mary Maternity Hospital, Newcastle upon Tyne.

The cardiac operations we have referred to in this study were performed by the late Mr G. A. Mason, Mr S. G. Griffin and Mr A. Blesovsky. Their help is gratefully acknowledged.

Our thanks are due to the Newcastle Regional Hospital Board for providing us with a research secretary over the years, to the Department of Radiology at the Newcastle General Hospital and to the Department of Photography, University of Newcastle upon Tyne, for the reproduction of X-rays.

Finally, we should like to express our sincere thanks to Messrs Churchill Livingstone for their assistance in the preparation of this monograph.

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1974

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PART I

CARDIOVASCULAR PHYSIOLOGY OF PREGNANCY

1. Circulatory Dynamics of Pregnancy

IMPORTANT changes take place in the maternal circulation during pregnancy. They consist mainly of an increase in cardiac output, in blood volume and in heart rate. Other relevant features are a varying degree of water retention, elevation of peripheral venous pressure and increase in metabolism with a rise in oxygen consumption. There are also slight respiratory changes. The haemodynamic changes of adaptation are well tolerated in the normal pregnant woman and in the majority of patients with structural heart disease. However, in certain patients the increased circulatory load can precipitate serious complications. Accordingly, the rational management of the pregnant patient with heart disease has to be based on the expected haemodynamic alterations peculiar to pregnancy and on the nature of the underlying cardiac disease.

The mechanism of the haemodynamic changes of adaptation encountered in normal pregnancy has not been fully clarified. It cannot be entirely explained on the basis of Burwell's original hypothesis of an arterio-venous shunt in the placenta (Burwell and Metcalfe, 1958, p. 11). More recently, Ueland and Parer (1966) demonstrated that the circulatory changes of pregnancy can be reproduced in the ewe by the intravenous infusion of oestrogens, and they postulated that ovarian and/or placental hormones may have a comparable physiological role in human pregnancy.

Cardiac Output

Catheterization studies by Hamilton (1949) and Palmer and Walker (1949) and later by Bader *et al.* (1955) indicated that the maximum rise in cardiac output is reached at about 20 weeks of gestation. The dye dilution studies by Walters *et al.* (1966) showed that there is already a significant rise in cardiac output before the end of the first trimester.

Current opinion is that the cardiac output starts to rise early in pregnancy, probably already within the first 10 weeks, first quickly then at a slower rate and reaches its maximum probably before the twentieth week (Hytten and Leitch, 1971, p. 72). Contrary to previously held views, this high level is then maintained until delivery. Previous estimations of the cardiac output showing a fall during the last eight weeks of pregnancy were carried out in the supine position in which the gravid uterus compresses the inferior vena cava resulting in a diminished venous return to the right heart (Scott and Kerr, 1963; Kerr, 1965). More recent investigations have shown that the cardiac output when measured with the patient in the lateral position does not fall towards the end of pregnancy (Lees *et al.*, 1967 *a*, *b*; Kerr, 1968; Mulholland and Boyle, 1968). Although Ueland *et al.* (1969) still found in their recent studies a decline in cardiac output in all positions—supine, sitting and lateral—towards the end of pregnancy, their figures show that the fall was only significant in the supine position. Their further statement is significant in this respect, namely that a change from the supine to the lateral position leads to no alteration in cardiac output in non-pregnant patients, but to a rise of 8 per cent in pregnant patients between 20 and 24 weeks of pregnancy, of 13·6 per cent between 28 and 32 weeks, and of 28·5 per cent at term.

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The increase in cardiac output which amounts to between 30 and 50 per cent under resting conditions is brought about by an increase in stroke volume and in heart rate. According to Walters *et al.* (1966) and to Pyörälä (1966) the increase in stroke volume is greater than that in heart rate. On the other hand, Hytten and Leitch (1971, p. 79) have reasoned that as the mean cardiac output in pregnancy rises by about one-third, and the heart rate by about one-fifth, the increase in stroke volume is proportionately smaller than that in heart rate. However, they emphasized that there is no fixed proportioning. In this respect it is noteworthy that the functional state of the heart can remain unimpaired when the pregnant patient has an artificial pacemaker at a fixed heart rate of 70 per minute (Shouse and Acker, 1964).

In spite of the increased stroke volume there is no rise in arterial pressure, presumably because of the fall in systemic vascular resistance. As the mean arterial pressure is only slightly altered in pregnancy, if at all, the work load of the left ventricle is directly proportional to the cardiac output (Kerr, 1968).

Blood Volume

There is an increase in total blood volume of the order of approximately 40 per cent. This increase is mainly brought about by an increase in plasma volume. The total red cell volume also increases but to a much lesser degree. As a result the haemoglobin concentration in the maternal blood declines during pregnancy.

The hypervolaemia is mainly responsible for the increase in cardiac output (Hytten and Paintin, 1963). It is of interest that this degree of hypervolaemia continuing until term was already reported by Hytten and Paintin in 1963 when it was not yet fully appreciated that the cardiac output did not fall during the last few weeks of pregnancy.

The plasma volume starts to increase as early as six weeks of gestation, rises rapidly until about mid-pregnancy and then slowly until term (Lund and Donovan, 1967). According to Rovinsky (1970) the plasma volume in normal single pregnancy is about 34 per cent above the non-gravid level at 21 to 24 weeks, rises to a peak of about 49 per cent at 33 to 36 weeks of gestation, and remains essentially at this level until term. However, Hytten and Paintin (1963) found no close correlation between the non-pregnant plasma volume and the actual increase during pregnancy.

The mean peak plasma volume increment is higher in twin pregnancy than in single pregnancy (Rovinsky and Jaffin, 1965), and also greater in multigravidae than in primigravidae (Hytten and Leitch, 1971, p. 16).

There is marked individual variation in the increase in red cell volume depending on several factors including supplemental iron intake. In the absence of supplemental iron intake the physiological increment of red cell volume in pregnancy is about 250 ml; when iron is taken the mean rise is of the order of 400 to 450 ml (Hytten and Leitch, 1971, p. 24).

The mean peak red cell volume increment is higher in multiple pregnancies than in single pregnancy (Rovinsky and Jaffin, 1965).

Heart Rate

The resting pulse rate increases during pregnancy by approximately ten beats per minute

(Burwell and Metcalfe, 1958, p. 12). According to Metcalfe (1968) the maximum is reached at term in patients who are studied lying either supine or in the lateral position, but when studies of seated patients are made the maximum heart rate is found at approximately 30 weeks of pregnancy with a demonstrable decrease during the last eight or ten weeks before delivery.

Rovinsky and Jaffin (1966) reported a gradual increase in heart rate to a maximum of 21 per cent at term in single pregnancy. In twin pregnancy there was a similar degree of increase already between 33 and 36 weeks of gestation which was followed by a rise to 40 per cent above non-gravid levels at term.

Circulation Time

Pyörälä (1966) found by the photo-electric method a decrease in the median circulation time from about 23 seconds in a non-pregnant control group to about 17 seconds in a pregnant group. However, there is considerable individual variation. Hytten and Leitch (1971, p. 90) expressed the opinion that the circulation time changes during pregnancy only very slightly, if at all.

Intracardiac Pressures

According to Werkö (1950) there is no significant change in right heart or pulmonary artery pressures. However, Bader *et al.* (1955) observed a slight rise in right ventricular end-diastolic pressure. The presence of an increased pulmonary blood flow without a rise in pulmonary arterial pressure indicates that there is a fall in pulmonary vascular resistance.

Arterial Blood Pressure

There is a marginal fall in systolic blood pressure and a slightly greater fall in diastolic pressure resulting in slight increase in pulse pressure. This change starts early in pregnancy and is accentuated if the patient lies flat on her back for a time (Adams, 1954). The total peripheral vascular resistance is considerably below normal in mid-pregnancy (Adams, 1954; Bader *et al.*, 1955). Both total peripheral vascular resistance and mean arterial blood pressure probably reach their lowest level at the time the cardiac output reaches its peak.

Supine Hypotensive Syndrome

Some patients experience fainting attacks in late pregnancy due essentially to a fall in arterial blood pressure especially when they lie in the supine position.

It is known that in the supine position there is a fall in cardiac output in late pregnancy. This reduction in cardiac output is as a rule not associated with a significant change in heart rate or a fall in arterial pressure, probably because of a concomitant rise in vascular resistance, and does not lead to syncope. However, in some patients this situation is followed by a parasympathetic response with bradycardia which prevents a rise in systemic vascular resistance and leads to a further and critical fall in arterial blood pressure and to a hypotensive state. The parasympathetic response with bradycardia is therefore

a critical factor in the production of the supine hypotensive state (Kerr, 1965; Courtney, 1970).

Venous Haemodynamics

The venous pressure in the arm is not significantly altered. On the other hand, it is increased in the legs and abdominal veins.

The venous haemodynamics were extensively studied by Scott and Kerr (1963). They have shown that the pressure in the inferior vena cava is markedly raised when the patient is lying in the supine position. As soon as the patient is turned on her side a marked fall in caval pressure occurs, although it still remains above normal. These results indicate that there is an obstruction to the flow of blood in the inferior vena cava in the supine position in late pregnancy caused by the gravid uterus. There is probably also an increased blood flow into the inferior vena cava from the uterine veins.

Ikard *et al.* (1971) investigated recently women in all stages of pregnancy and in the postpartum period by the ultrasonic flow detector method in order to determine the degree of venous obstruction in various positions and its possible role in the causation of venous complications. They found evidence of obstruction to venous return from the lower extremities throughout the whole of pregnancy. However, leg vein obstruction greatly varied with the position of the patient: it was almost universal in the third trimester on standing and to a much lesser extent in the supine position.

The increased venous pressure in the lower limbs is an important factor in the development of ankle oedema and also of varicosities.

Peripheral Oedema

Oedema of the ankles and legs is a common feature in normal pregnant women and is in the absence of cardiovascular or renal disease of little pathological significance.

Hytten *et al.* (1966) observed oedema in about 50 per cent of primigravidae who had perfectly normal pregnancies in all other respects. They found a direct relationship between the degree of oedema and the increase in body water. Between 10 and 38 weeks of pregnancy the gain in body water was 6·85 kg in women with no oedema, 7·19 kg in those with oedema of the legs and 9·8 kg in those with generalized oedema. The average woman accumulates about 8·5 l of water in a normal pregnancy (Hytten and Robertson, 1971).

Water retention can be mainly accounted for by the salt retention that takes place in pregnancy. There is an increase in total exchangeable sodium and according to Hytten and Thomson (1968) normal pregnant women accumulate about 500 to 600 mEq of exchangeable sodium. There is also an increase of about 170 mEq of body potassium in late pregnancy.

Thomson *et al.* (1967) found in normotensive pregnant women that the incidence of peripheral oedema increased with age, namely from 14 per cent in those under 20 to 29 per cent in those over 30 years of age.

Oedema in late pregnancy can be attributed partly to the relatively high venous pressure in the lower limbs, and partly to the reduced colloid osmotic pressure of plasma.

More recently Robertson (1971) studied again the natural history of oedema during

pregnancy. He found clinical oedema in 83 per cent of women who had no evidence of renal or cardiac disease and were considered healthy at the onset of pregnancy. Thirteen per cent of all patients developed some hypertension during late pregnancy. There was no consistent relationship between the presence of oedema and the later development of hypertension. He concluded on the basis of measurements of several parameters such as body weight, blood volume, ankle circumference and finger size that increased tissue hydration is universal in normal pregnancy. He also emphasized that the presence of oedema cannot be regarded as an indication that hypertension is likely to develop in the later stages of pregnancy.

RESPIRATORY FUNCTION IN PREGNANCY

The oxygen consumption rises progressively during pregnancy and reaches its maximum of about 20 per cent above the non-pregnant level near term (Burwell, 1954).

Gazioglu *et al.* (1970) reported that in normal pregnant women the vital capacity tended to increase with a decrease in expiratory reserve volume and in residual volume. However, these changes were only slight and statistically not significant. Hytten and Leitch (1971, p. 114) concluded that there was no significant change in vital capacity and little or no increase in respiratory rate. There is hyperventilation as a rule with a lowered alveolar and arterial carbon dioxide tension (Hytten and Leitch, 1971, p. 126).

Gazioglu *et al.* (1970) found a slight decrease in pulmonary diffusing capacity in the majority of normal pregnant subjects while the pulmonary capillary blood volume was unchanged. They contrasted these findings with those in patients with mitral valve disease who showed a much more marked decrease in pulmonary diffusing capacity with an increase in pulmonary capillary blood volume.

Mechanical effects of pregnancy and hormonal influences are probably responsible for some of the respiratory changes in normal women (Novy and Edwards, 1967).

CIRCULATORY CHANGES IN LABOUR AND PUERPERIUM

Each uterine contraction leads to a rise in right atrial pressure, to an increase in cardiac output of the order of 15 to 20 per cent and at the same time to a rise of the mean arterial pressure by about 10 per cent. This combination of a rise in cardiac output and in mean arterial pressure implies increase in left ventricular work (Kerr, 1968).

Serial haemodynamic measurements throughout labour including periods between uterine contractions have also shown a cumulative rise in cardiac output by about 40 per cent above the values found in late pregnancy. However, this cumulative rise is mainly caused by pain and apprehension as patients free from pain owing to continuous caudal analgesia did not show it. On the other hand, caudal analgesia did not affect the haemodynamic changes caused by uterine contractions (Hansen and Ueland, 1966; Kerr, 1968).

Following delivery there is a rise in blood volume which may be partly due to re-absorption of tissue fluid accumulated during labour or during the whole of pregnancy,

and partly to the quantity of uterine blood squeezed into the maternal circulation after completion of labour. The non-gravid blood volume level is restored within 4 to 6 weeks after delivery (Rovinsky, 1970).

There is also a postpartum rise in cardiac output of about 10 to 20 per cent associated with bradycardia (Brown *et al.*, 1947; Metcalfe, 1963; Hansen and Ueland, 1966). After a variable tachycardia during labour a relative bradycardia sets in immediately postpartum. According to Adams (1954) the puerperal relative bradycardia persists for about two weeks. The combination of a raised cardiac output and bradycardia indicates an increase in stroke volume. This is brought about by an increase in venous return to the heart following reduction of the blood volume in the utero-placental vascular bed and the relief of caval obstruction by the uterus. This increase in stroke volume starts immediately after delivery and is said to persist for about two weeks (Adams, 1954).

There is a decrease in red cell volume at delivery the magnitude of which is directly related to blood loss. A return to non-gravid red cell volume level takes place within 60 days after delivery (Rovinsky, 1970).

There is also a postpartum decrease in total body water with increased diuresis (Haley and Woodbury, 1956; Metcalfe, 1963; Walters *et al.*, 1966).

Kerr (1968) has, however, pointed out that some of the haemodynamic data obtained after delivery may reflect a simple postural phenomenon as the studies during labour were carried out with the patient in the supine position in the presence of caval occlusion and consequent reduction in cardiac output. Furthermore, the post-delivery circulatory changes are necessarily variable and modified to a lesser or greater extent by the amount of blood loss at delivery. In any event, owing to the circulatory readjustments the first week and especially the first few days of the puerperium constitute a potentially hazardous period in the presence of structural heart disease.

HAEMODYNAMIC RESPONSES TO EXERCISE

Haemodynamic responses to exercise in the pregnant woman assume real significance in the presence of structural heart disease.

Ueland *et al.* (1969) found in normal women that the absolute rise in cardiac output following light exercise was greater during pregnancy than in the non-pregnant state. In further studies, Ueland *et al.* (1972) also observed that the altered haemodynamic response to exercise which they found even in symptomatically mild mitral stenosis was accentuated by pregnancy. The increase in cardiac output with exercise was achieved more and more by an increase in heart rate as pregnancy advanced. The stroke volume response to exercise became gradually less, although it remained higher at all stages of pregnancy as compared with the non-pregnant state.

Bader *et al.* (1955) pointed out earlier that in patients with mitral valve disease the post-exercise increase in cardiac output takes place at the expense of a raised pulmonary capillary, pulmonary venous and left atrial pressure.

Following slight to moderate amount of work there is also an additional increase in the oxygen consumption during pregnancy as compared with the non-pregnant state (Hytten and Leitch, 1971, p. 129).

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