Medical Disorders in Obstetric Practice

Cyril G. Barnes

Fourth edition

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Preface

To Fourth Edition

The general arrangement of this book has proved satisfactory and it has not been altered for this edition. Changes have been made in the sections on diabetes and heart disease, that on respiratory function has been expanded and the chapter on thrombo-embolism brought up to date. New material includes vaccination against rubella, modern tests for thyroid function and syphilis, drug addiction and porphyria.

The attention of the physician is drawn to the importance of modern tests for feto-placental function and fetal maturity in those maternal disorders which put the fetus at risk, and that of the obstetrician to Australia antigen and the treatment of hypertensive crises. Many older references have been replaced by new.

I hope that medical and obstetric Registrars and House Officers, for whom this book is written, will find the new edition clear and practical and as up-to-date as one can hope any text to be in these days of rapid progress in Medicine and Obstetrics.

C.G.B.

January 1973

To First Edition

My primary aim in writing this book has been to help obstetric officers and registrars whose patients are suffering from medical disorders of which they themselves have had little experience. I hope, however, that it may also assist general physicians and medical registrars who, because they see little obstetric work, are sometimes uncertain of the effect of pregnancy and labour on the course of medical diseases.

The book is based upon the teaching which I have given to successive generations of students attending the postgraduate courses of the Institute of Obstetrics and Gynaecology at Queen Charlotte's Hospital, and this in turn upon my experience since 1939 while I have been on the staff of Queen Charlotte's and Hillingdon Hospitals in which approximately 5,000 women are delivered each year. I have kept strictly to those disorders in which the obstetrician is likely to seek the help of the physician; thus there is no chapter on pre-eclamptic toxaemia which, I have found, the obstetrician regards as entirely his own concern, although other causes of hypertension, oedema and albuminuria are fully discussed.

I have not tried to write an advanced or exhaustive treatise on medical disorders in pregnancy. For those readers who may wish to delve more deeply into the subject I have added at the end of each chapter a list of references which will provide them with a basis for further reading, and, so far as has been possible, I have confined these references to papers in the English language and published in journals which are likely to be available in the medical library of any large general hospital.

My experience of dermatology in obstetrics was insufficient to allow me to write the chapter on this subject. I was fortunate in being able to persuade my colleague Dr F.J.V. Jenner to undertake the preparation of this section of the book and I am most grateful to him for his collaboration.

It is impossible to write even a simple textbook without relying upon the cooperation of many people, and I gladly acknowledge the help which has been given me most willingly by medical and lay colleagues. I am much indebted to my obstetric colleagues at Queen Charlotte's and Hillingdon Hospitals who have asked me to see their patients and discussed their medical problems with me, and in particular to Dr Joyce Morgan, F.R.C.O.G., who cheerfully undertook the task of reading the script and made a number of constructive suggestions.

Dr J. D. O'D. Lavertine and Dr Gilbert Scott provided the pathological specimens and X-rays respectively, and these were photographed by Mr E. Stride, A.I.B.P., who also prepared the diagrams. Dr A.S.R. Peffers of the British Overseas Airways Corporation kindly checked the section on Air Travel in Pregnancy for me. The book would not have been completed had it not been for the enthusiasm of my secretary, Miss Margaret Hunt, who not only typed the script but also deciphered my handwriting. To all these colleagues I express my thanks for their ready cooperation.

Finally, I would like to acknowledge the courtesy of Mr Per Saugman of Blackwell Scientific Publications who, having asked me to write this book, solved each of my problems as it arose but otherwise left me undisturbed until the book was completed.

Contents

CHAPTER 1

Circulatory Adjustments During Normal Pregnancy

A number of circulatory adjustments take place in a pregnant woman as her uterus enlarges to accommodate the growing fetus, the placental circulation develops to supply it with food and oxygen and widespread hormonal changes occur. These adjustments not only throw a considerable burden on the mother's heart, which may fail if it is diseased, but also produce physical signs and symptoms which may simulate heart disease although none is present. A description of these physiological changes is given in this chapter because it is an essential preliminary to any discussion on the management of pregnancy and labour in patients with heart disease.

The cardiac output during pregnancy

It has long been known that the resting cardiac output increases during pregnancy but it has proved difficult to obtain precise knowledge of the magnitude of the increase or the stage of pregnancy at which the maximum output is attained.

Early haemodynamic studies during pregnancy depended on the use of techniques based mainly on right heart catheterization and application of the direct Fick principle. They were usually carried out on the supine patient and as single investigations since repeated catheterization could not be justified in normal pregnancy. No clear agreement was reached on the stage of pregnancy at which maximum cardiac output was attained, but it was believed that it was about the 24th week and that cardiac output then remained on a plateau until the last six weeks of pregnancy when it fell rapidly and by term had returned almost to the pre-pregnancy level.

More recent investigations have utilized indicator dilution techniques and so avoided right heart catheterization and allowed repeated investigations to be made on the same patient throughout pregnancy. Even so the results obtained by various workers have not been identical.

Using this method Roy *et al.* (1966) found that the maximum cardiac output was reached between the twenty-eight and thirty-fourth weeks of pregnancy and this was in keeping with the results obtained by Walters and his colleagues (1966) who also reported that the cardiac output was considerably raised as early as the eighth to eleventh week. The work of Lees *et al.* (1967) has made it almost certain that most of the increase in cardiac output during pregnancy has been attained by the end of the first trimester when the resting value is about 40 per cent more than it was when pregnancy began, that it increases only slightly during the second trimester and thereafter remains almost unaltered until term. They found that when the cardiac output was measured with the patient in the lateral recumbent position no decrease was observed during the last weeks of pregnancy and Kerr (1965) had already demonstrated that the fall noted by other workers was due to the occlusion of the inferior vena cava by the pressure of the gravid uterus as the patient lies supine, with resultant diminution in the venous return to the heart.

It is now widely believed that the resting cardiac output has increased by about 40 per cent at the end of the first trimester, that only a small increase occurs thereafter and that the output then remains at this level until term. The work of the heart is proportional to the cardiac output and the mean arterial blood pressure and, since the latter is but little altered in pregnancy, it follows that the basal work of the heart has increased by about 40 per cent at the end of the first trimester. Over and above this basal increase any physical activity, such as walking, on the part of the pregnant woman must demand more and more work from the heart as pregnancy advances, since most women gain about 12·5 kg in weight during the course of pregnancy.

The increase in cardiac output during pregnancy is much greater than the oxygen requirements of the maternal and fetal tissues warrant; a woman's basal consumption of oxygen increases by only about 10 per cent during pregnancy, 80 per cent of the increment being utilized by the fetus. The rise in cardiac output cannot therefore depend solely on the fetal demands for oxygen and may be brought about by the action of ovarian and placental hormones. It is achieved by a combination of tachycardia and augmented stroke volume. The heart rate increases by at least 10 beats per minute (14 per cent) during pregnancy which represents a minimal addition of 14,000 beats in the course of 24 hours but, since the cardiac output rises by 40 per cent, the main factor responsible for this must be an increase in stroke volume.

Since the stroke volume increases whilst the mean arterial blood pressure is little altered it must follow that there is a fall in systemic vascular resistance during pregnancy. The rate of blood flow increases and the circulation time has shortened by about 2 seconds at mid-term.

The augmented stroke volume is dependent on an increase in the venous return to the heart to which alteration in circulating blood volume may contribute. The plasma volume begins to increase about the tenth week of gestation, reaches its peak at the thirty-fourth week and then falls steadily until term; by contrast, the increase in red cell volume, which is less than that in the plasma volume, continues until delivery. The expansion in plasma volume has been measured by a number of methods (Caton *et al.*, 1951), the values obtained varying from 25 per cent to 55 per cent above the figure at the start of pregnancy; it is generally accepted that there is an increase of 40–45 per cent in the plasma volume and of 20 per cent in the red cell volume at the thirty-second week. The plasma volume has returned to normal by the end of the second week of the puerperium. The increase in cardiac output cannot depend entirely on the enlarging blood volume since the maximum output is attained considerably earlier than is the maximum blood volume.

The expansion in circulating blood volume is probably hormonal in origin, retention of sodium and water being brought about by aldosterone secreted from the adrenal glands. The secretion of this hormone increases markedly after the third month of pregnancy and continues until delivery (Venning and Dyrenfurth, 1956); this is reflected in the urinary excretion of aldosterone which increases until it is two to seven times that in non-pregnant women (Rinsler and Rigby, 1957). The high levels of oestrogen and progesterone in the blood during gestation have also been held responsible for the expansion in blood volume.

It has been suggested that the developing placenta contributes to the rise in cardiac output by acting as an arterio-venous fistula which allows a rapid return of blood to the right atrium and so causes a rise in venous filling pressure. This hypothesis has been criticized by Winner (1965) and seems the more improbable since we have learnt that most of the increased cardiac output is established by the end of the first trimester when the choriodecidual blood flow is still small.

Cardiac output during delivery

During labour so many variable factors are present that we have little satisfactory information about circulatory haemodynamics at that time. We know that each uterine contraction is accompanied by a transient rise in arterial blood pressure and oxygen consumption, that ectopic beats are frequent during the second stage, that tachycardia occurs during delivery and that there is a period of bradycardia as soon as labour is completed. With each contraction up to 500 ml of blood is expelled from the uterus augmenting the venous return to the heart and temporarily increasing the cardiac output by at least 20 per cent (Adams and Alexander, 1958). Even between contractions the cardiac output is increased during labour above that in late pregnancy, but this may be the result of pain and apprehension since Hansen and Ueland (1966) found that it was not the case in women kept free from pain by continuous caudal anaesthesia.

Immediately after delivery the cardiac output rises by about 30 per cent as the involuting uterus empties its blood into the circulation, assisted perhaps by the action of oxytocic drugs, but intra-partum blood loss may modify the effect of this auto-transfusion. Removal of the pressure of the uterus from the great veins of the pelvis and abdomen also increases the venous return to the heart and thus the cardiac output. The cardiac output returns to the non-pregnant level about the end of the second week postpartum (Walters *et al.*, 1966).

Physical signs in the cardiovascular system during normal pregnancy

The physiological changes in the maternal circulation which have been described above produce a hyperkinetic circulatory state during pregnancy resembling that found in hyperthyroidism. The hands are warm and may show capillary pulsation, the arterial pulse is of large volume and the venous pressure in the neck increases by about 1 cm. The cardiac impulse is forceful, the first sound at the apex loud and split and systolic ejection murmurs, caused by increased blood flow through the great vessels, can usually be heard over the base of the heart. These murmurs increase in intensity during inspiration and therefore probably originate in the pulmonary artery. Rapid ventricular filling during early diastole accentuates the normal third sound of the

heart which can be heard in 75 per cent of pregnant women after the sixth month of gestation.

As the uterus enlarges the diaphragm rises, lifting the heart and rotating it about its antero-posterior axis. This leads to displacement of the apex beat upwards into the fourth space and outwards to the left by as much as 3 cm during the third trimester, giving a false impression of cardiac enlargement and sometimes increasing the cardiothoracic index in the chest radiograph to more than 50 per cent. Some workers have described an increase in the diastolic size of the heart shadow in the chest radiograph during pregnancy, but the change in position of the heart makes it difficult to be certain whether any cardiac enlargement has actually occurred. The pulmonary artery pressure does not increase during normal pregnancy so that the pulmonary vessels must dilate to accommodate the augmented blood flow. The increased size of the pulmonary vessels is responsible for the exaggerated vascular markings in the lung fields seen on chest radiographs of the pregnant patient. There is some backward displacement of the barium-filled oesophagus in the right anterior oblique view of the heart.

The rotation of the heart causes changes in the E.C.G., a deep Q wave developing in standard lead 3 although not in lead a VF. T wave inversion may also occur in lead 3, and as pregnancy advances flattening of the T wave and occasional depression of the S–T segments may develop in chest leads facing the surface of the left ventricle (Oram and Holt, 1961).

'MAMMARY MURMURS' IN PREGNANCY

As the breasts enlarge they become more vascular and it is not uncommon for bruits, produced in the mammary blood vessels, to be heard over the second and third intercostal spaces close to the sternum, more frequently on the left side than on the right. These murmurs are usually high-pitched in quality and systolic in time and are loudest when the patient lies supine. They start after the first heart sound and occasionally extend into, or throughout, diastole when they may be mistaken for the murmur of a patent ductus arteriosus. Each year one or two patients are referred to me in whom such a murmur is loud and extends well into diastole, but cases in which the murmur is confined to systole are more common. The bruits do not develop until late in the second trimester and can be recognized by the fact that their pitch and intensity can be varied by the pressure exerted with the bowl of the

stethoscope on the chest wall. The murmurs are extremely localized and can be abolished by gentle pressure of the finger in the intercostal space lateral to the stethoscope. Tabatznik *et al.* (1960) have suggested that the murmurs are produced by turbulence at the site of anastomoses between the aortic intercostal arteries and the branches of the internal mammary artery, and they noted that arterial pulsation can often be observed in the intercostal space at which the murmur is audible. I have noticed that the intercostal pressure required to obliterate the murmur is sometimes so slight that the origin of the sound must surely lie in thin-walled, low pressure anastomotic vessels. The murmur continues during the first few weeks of lactation but gradually fades towards the end of the second month after delivery. It is likely to recur in subsequent pregnancies.

POSTURAL HYPOTENSION DURING PREGNANCY

In the last trimester the enlarged uterus compresses the inferior vena cava against the spinal column, interfering with the venous return from the legs in which the venous pressure increases by 10 or 15 cm of water. The effect is enhanced by the lumbar lordosis of late pregnancy and it is in part responsible for the oedema of the feet and varicosity of the leg veins which are common among women at this time.

The compression of the I.V.C. is greatest when the patient lies supine. The pressure in the lower I.V.C. may then exceed 20 mmHg and the venous return to the heart be so much impaired that the cardiac output falls abruptly leading to syncope, low blood pressure and sometimes bradycardia. Holmes (1960) found that in 8·2 per cent of pregnant women the systolic blood pressure fell by more than 30 per cent when they lay supine for a few minutes and that in 2 per cent of patients the fall in pressure was over 50 per cent. The symptoms, which are most marked when there is polyhydramnios or multiple pregnancy, are rapidly relieved when the patient turns onto her side and becomes less severe once the head has been engaged in the pelvis. Kerr *et al.* (1964) showed that the I.V.C. is virtually occluded in all pregnant women in the supine position near term. It appears that susceptibility to the supine hypotensive syndrome may turn on the ability of the azygos and vertebral venous systems of the individual to maintain an adequate venous drainage from the legs and lower half of the body.

It is known that compression of the I.V.C. leads to increased pressure in the chorio-decidual space and it was suggested by Mengert *et al.*

(1953) that this may sometimes cause mechanical separation of the placenta and so contribute towards accidental antepartum haemorrhage; but confirmation of this theory in Man is lacking. Nonetheless, it is advisable to nurse patients with accidental haemorrhage in the lateral position.

In the pregnant woman enlargement of varicose veins of the legs and vulva is not due only to compression of the I.V.C. by the gravid uterus or to the increase in circulating blood volume. There is a generalized relaxation of smooth muscle during pregnancy leading to diminished venous and arterial tone, so that the dorsal veins of the hands also become engorged. This is probably a hormonal effect since it can be observed to some degree in women taking oral contraceptive agents. The blood flow through the skin of the hands and forearms increases and patients with Raynaud's phenomenon find that their symptoms improve with pregnancy.

CARDIAC ARRHYTHMIAS

Ectopic beats either ventricular or supraventricular in origin occur in about 15 per cent of normal women during pregnancy and, provided that no heart disease is found on clinical examination, they need cause no anxiety to the obstetrician although he should be alert for evidence of hyperthyroidism both in these patients and in those who suffer from attacks of paroxysmal tachycardia. Ectopic beats, however, may cause considerable discomfort to the patient who should avoid distension of her stomach with large meals, reduce her smoking and take sodium amytal 60 mg t.d.s., since this sedative often seems either to reduce the number of ectopic beats or to make the patient less conscious of them. Ventricular ectopic beats may sometime be abolished by practolol 100 mg four times a day or by procainamide 250 mg thrice daily. When atrial ectopic beats occur in a patient with mitral disease they may presage the onset of atrial fibrillation.

Bouts of supraventricular paroxysmal tachycardia are an infrequent but annoying accompaniment of pregnancy and the puerperium, and occur both in normal women and in patients with rheumatic heart disease. It is probable that pregnancy increases the susceptibility to paroxysmal tachycardia because women who are subject to the bouts between pregnancies may find that they are more frequent during gestation, whilst some patients experience attacks only during pregnancy or the puerperium. Szekely and Snaith (1953) observed ten patients

with paroxysmal tachycardia, all supraventricular in origin, among 10,746 pregnant women, but they point out that the incidence was probably greater than this as there were other patients who complained of symptoms which were probably caused by the arrhythmia but from whom no clinical or cardiographic confirmation was obtained.

The onset of tachycardia is abrupt; the heart rate suddenly increases to 150 or 200 per minute and is unaffected by posture or exertion. The attack may last from a few seconds up to 12 hours or more, and usually stops as abruptly as it began. Isolated ectopic beats may occur between the paroxysms of tachycardia. During the attack the patient is anxious and restless, and may vomit. She complains of throbbing in the arteries, especially those of her head, and a sense of great physical exhaustion.

The prognosis of paroxysmal tachycardia depends upon the presence or absence of underlying heart disease. If there is no cardiac lesion the attacks are not dangerous but are a great nuisance to the patient. If there is heart disease, and especially if mitral stenosis is present, they may precipitate pulmonary oedema by shortening diastole and thus interfering with the flow of blood from the left atrium into the ventricle.

Some paroxysms of tachycardia can be terminated by carotid compression which the patient may learn to apply for herself, others can be stopped by a cold drink or by breath-holding. The majority, however, cease spontaneously and it is necessary only to reassure the patient and give her a sedative such as sodium amytal 180 mg whilst awaiting the return of normal rhythm. Attacks which continue for several hours may be treated either with digoxin 0·75 mg intravenously, followed by 0·25 mg orally every 4 hours, or by practolol 100 mg by mouth three times a day. After the third month of pregnancy quinidine can be used in a dosage of 500 mg, followed by 300 mg every 6 hours; the drug has no effect on the pregnant human uterus unless contractions have already started but there are still fears that it may be teratogenic in the first trimester. When the bouts of tachycardia are frequent the patient should be kept digitalized and she should avoid smoking, large meals and effervescent drinks.

Diagnosis of heart disease in pregnancy

The hyperkinetic circulation of pregnancy accentuates the cardiac murmurs which accompany organic heart disease. For example, it is not uncommon for the crescendo presystolic murmur of mitral stenosis to

be clearly heard during pregnancy and then, a month after delivery, for it to become inaudible until the circulatory rate is once more increased by the patient taking exercise. For this reason organic valvular disease of the heart seldom passes unrecognized during pregnancy.

By contrast, many patients whose hearts are normal are suspected of cardiac disease in the course of pregnancy on account of the murmurs and other physical signs produced by the hyperkinetic circulatory state. It is my experience that nearly 50 per cent of the patients referred to me from antenatal clinics for assessment of their cardiac status have no heart disease. It is sometimes extremely difficult to be certain whether organic heart disease exists in a pregnant woman. She should then receive the same antenatal attention as a patient with known heart disease and the final diagnosis delayed until after delivery. It is common experience among physicians to find that as many as 5–10 per cent of the patients whom they considered during pregnancy to have organic cardiac disease, especially with congenital lesions, show no evidence of heart disease a month after delivery. One must then tell the patient without equivocation that her heart is normal as otherwise the increased attention directed towards it during pregnancy may leave her with the belief that she has cardiac disease and cause her unnecessary anxiety or even disability.

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