HEART DISEASE IN PREGNANCY

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HEART DISEASE IN PREGNANCY.

Heart disease is one of the most serious complications of pregnancy and both its medical and obstetrical care have inspired a rich literature. Foremost amongst the writers on this subject was Sir James Mackenzie who, by his careful clinical studies and development of the polygraph, brought a new concept to the action of the heart in health and disease and, guiding the profession to a more rational approach to the treatment of the damaged heart, succeeded as a truly beloved physician in granting a new life and humanity to the patient. It was soon after his entrance into general practice that Mackenzie's great life-interest was first aroused and stimulated by the tragedy of a young girl who died during labour in acute heart failure with her child unborn and such was his concern and dismay at the death of this young mother that he proceeded to make from his practice in Burnley his special study of the heart. Many of his patients were drawn from his large midwifery practice and his close clinical notes on their daily progress were astounding in their accuracy; and his acuteness of observation of any new change in symptom or sign enabled him to elucidate the various hazards met by

the heart during pregnancy and often successfully to anticipate and prevent their appearance.

Prior to MacKenzie's time pessimism surrounded the outlook of the profession towards the woman whose heart disease was complicated by pregnancy. The Continental writers of the time, led by Peter, (1874) were strong in their disapproval of marriage by women with valvular disease of the heart and MacDonald (1878) in this country was scarcely less severe in his condemnation. Peter (1874) stated that women with heart disease should not marry, nor bear children and if they did, not to nurse them: and MacDonald's (1878) statistics of a 55% death rate explained his gloomy outlook. It needed the new appreciation of the importance of the functional state of the heart, as expounded by MacKenzie, (1921) gradually to establish the view which is today universal that prevents the interdiction of the marriage or pregnancy of a woman suffering from heart disease, but with many years of enjoyable life before her and for whom, as Gilchrist and Murray Lyon (1933) have shewn, the bearing of one or two children need not decrease the expectancy of life.

The interruption of pregnancy was also at this time a subject of much controversy. Jensen (1938) states that the French School of Tarnier and

Champetier de Ribes insisted on the value of this operation in suitable cases and, although for many years opposed to in this country by MacDonald, (1878) their liberalism eventually influenced the general opinion, which, as MacKenzie's (1921) views gained ground, were made more on functional than anatomical grounds and thus logically delimiting the operation. While prevention of interruption of pregnancy was attracting a wealth of dissenting opinions, MacKenzie (1921) was carefully formulating those guiding principles of prenatal care for safe conduct of the woman through her pregnancy and labour and laying down clear and specific directions, which are more comprehensive than those based on the classification of the American Heart Association, in widespread use at the present day.

MacKenzie (1921) states that before the discovery of auscultation and uses of the stethoscope, experienced physicians estimated the state of the heart by its manifest functional efficiency and many of the views which he expressed were, he says, anticipated by them. Hasty conclusions were being made in his day on the importance and significance of heart murmurs, and he set about to make records of the signs and symptoms which he took to be a departure from normal during a confinement and also those which occurred as a consequence of straining at the time

of labour. He soon found a number of changes - for example, modification of the heart sounds, changing murmurs, irregularity of action, displacement of the apex beat to the left, duskiness of the countenance: and some of these signs were modified or disappeared during the puerperium. He was, he states, at a loss to explain the significance of these changes and decided to give the subject a more thorough study, watching for the onset of the signs and the possible causative factors and contriving, with a long follow-up after the confinement, to see if the signs disappeared or remained to give future trouble. He found that one sign or another required particular observation and was of special importance - for example oedema of the lungs was considered to be of special significance in heart failure and he realised that there was a general embarrassment of the circulation, which increased in the last months of pregnancy and that often irregularities of the pulse and pulsation in the neck veins would appear in the patient. It was apparent, therefore, that the heart and circulation underwent definite changes in the course of a normal pregnancy.

CHANGES DURING PREGNANCY IN THE NORMAL HEART AND CIRCULATION.

The vital capacity of the lungs.

MacKenzie (1921) noted that towards the sixth month of pregnancy the patient's response to effort became restricted, breathlessness appearing after an amount of exertion which had hitherto been performed in comfort: as a rule this breathlessness never became extreme and he found the patient able to attend to her household duties, but in a more restricted fashion and she felt breathless chiefly on going up-stairs. The splinting of the diaphragm by the growing uterus was in part the cause of breathlessness, but MacKenzie (1921) considered that the diminished vital capacity of the lungs on this account was offset by the widening of the chest-wall which allowed a greater lateral excursion of the lungs. In one patient he described the chest circumference below the breasts was $5\frac{1}{2}$ inches greater at term than it was three weeks after delivery. In more recent studies some authors, such as Alward, (1932) published numerous readings to shew that there is a definite tendency for the vital capacity of the lungs to decrease to about 20% below the estimated normal just

before labour; while Jensen (1938) quotes yet others such as Hasselbach, studying single cases, ascertained a definite but slight increase in vital capacity; and more recently Burwell (1937) was struck by the fact that the vital capacity was not essentially diminished during pregnancy. Hamilton and Thomson (1941) were able to confirm the above observation of Burwell (1937) but, in striking contrast with the normal heart, found that there was a decrease in the vital capacity of the lungs in the presence of cardiac decompensation and these changes were noted in some patients before chemical signs of failure made themselves manifest. They concluded that a decrease of vital capacity, between monthly reading, of 10 per cent or less are of no significance. A decrease of 15 per cent or more, or a continued decrease from month to month of 10 per cent or more on successive determinations, Hamilton and Thomson (1941) believe however to be a definite sign of impending heart failure during pregnancy, in the absence of some obvious extracardiac cause.

Displacement of the heart.

There is a displacement and rotation of the heart outwards and upwards during pregnancy, due to the diaphragm being pushed up by the abdominal contents and growing uterus. Before the advent of

X-rays the normal displacement and rotation of the heart by the growing uterus was noted by MacKenzie (1921) frequently to be as much as 1 inch outside the left nipple line and upwards to the fourth interspace and that after labour the heart gradually swung back into its normal position. X-ray examination confirms this displacement, although it reveals, as a rule that the heart is smaller than anticipated from physical examination.

Hypertrophy of the heart.

MacKenzie (1921) was adament that there was no hypertrophy of the heart although the current view of his time, supported by MacDonald (1878) and Handfield-Jones (1928) was that of Larcher, who, quoted by Jensen (1938), stated that there was hypertrophy of the left ventricle during pregnancy. Jensen (1938) goes on to state that observations were made by Bonomi in 1900 that there was an increase in the size of the myocardial fibres of pregnant guinea pigs, but later experiments by Petren and Sylven shewed that there was no increase in the absolute weights of the hearts. More recently still Liere and Sleeth (1938) conducted similar experiments, which consisted of taking one group of young pregnant guinea pigs and weighing and killing the mother 72 hours after its young was born and then weighing the

hearts; in a second group of guinea pigs, pregnancy was allowed to proceed for seven weeks and then the animals were weighed and killed and the hearts weighed. The conclusions reached by Liere and Sleeth (1938) were, that pregnancy does not cause cardiac hypertrophy in guinea pigs and they state that since pregnancy does not produce cardiac hypertrophy in different types of animals (guinea pigs, cats, dogs) it seems doubtful that it would produce it in human beings. Increased cardiac work, they conclude, does not necessarily produce cardiac hypertrophy. MacKenzie (1921) wrote that it took long years of effort to produce hypertrophy of the left ventricle and he said it was a matter of observation that strong healthy vigorous men do not have larger hearts than men carrying on less vigorous work. Enlargement of the cardiac shadow in the normal heart can be accounted for by the displacement and rotation of the heart, although Burwell and Strayhorn (1933) stated that they observed cardiac enlargement during pregnancy before such displacement could account for it. Gammeltoft, (1928) too, states, following cardiac X-ray examination of women throughout pregnancy, that during the first six months the left ventricle undergoes a relative hypertrophy in proportion to the right ventricle and from the sixth month this

hypertrophy is compensated by a hypertrophy of the right half of the heart: and, like Burwell and Strayhorn, (1933) Gammeltoft (1928) says that these changes can be demonstrated as early as the third or fourth month of pregnancy. Post-mortem findings have, however, failed to shew either hypertrophy or dilatation of the heart, and the evidence of such change in the normal heart during pregnancy would appear still to be inclusive.

The work of the heart.

The work of the heart is increased in order to drive the blood round a greatly expanding vascular area, with an added volume of circulating blood and an extra body weight which increases by an extra 25 to 30 pounds by term, as shewn by the studies of Thomson and Cohen (1938). There is no agreement, however, Jensen says, whether the increase in work is considerable or whether it is so slight that it cannot add materially to the heart's work, which is further eased Cohen and Thomson (1936) state by the diminished viscosity of the blood. Jensen (1938) goes on to state that the metabolic rate is increased often out of proportion to the increase in weight and the heart meets the extra demands made upon it by increasing its minute volume; and, he adds, it is not definitely decided if this is accompanied by an increase in the

rate or the stroke volume of the heart and there is the further possibility that not all hearts react in the same way. Stander and Cadden (1932) have shewn that in the normal gestation of animals and humans the cardiac output rises above the normal level in the fourth month and steadily increases, until at term it amounts to approximately fifty per cent above normal. The increase in cardiac output, they state, is due to the increased nervous return and greater diastolic filling of the auricles and consequently a greater stroke volume output.

Cohen and Thompson (1936) in their work found an increase in the cardiac output up to the ninth lunar month, with subsequent decrease prior to labour at term. Burwell (1938) et al., in a study of the output of the heart and other related observations, confirmed Cohen and Thompson's (1936) findings that there is an increase in cardiac output per minute demonstrable at the third and fourth month, falling towards normal in the last weeks of pregnancy and being within normal limits for the non-pregnant woman after delivery: the increase in output was, however, greater in proportion than the increase in oxygen consumption and hence the arterio-venous oxygen difference is diminished.

Blood Changes.

In addition to the increased return Dieckman and Wegner (1934) have shewn that there is an increase in the volume of circulating blood, with an increase in plasma, a relative or absolute diminution in the number of formed elements and a relative anaemia.

Basal Metabolic rate.

Plass and Yoakum (1929) have shewn a progressive increase in the Basal metabolic rate from the fourth month of gestation to term and a subsequent fall during the puerperium.

Blood Volume.

In their studies of blood volume changes Hamilton and Thomson (1941) state that there is a progressive increase in plasma volume from early pregnancy to the ninth lunar month, when a maximum is reached; and this is an average increase of 65 per cent over the average normal non-pregnant value. During the tenth lunar month there is a definite decrease in plasma volume, so that at this time it is 50 per cent above the normal non-pregnant level. This decrease continues post partum to reach the normal non-pregnant level by the end of the second puerperal week.

Total blood volume: Hamilton and Thomson (1941) state that there is a progressive increase in the total blood volume from early in pregnancy to the

ninth lunar month at which time the average increase was 45.5 per cent over the average of a comparable normal non-pregnant control group. During the tenth month there was a definite diminution, they state, in the total blood volume to 32.4 per cent above the normal non-pregnancy level, with a prompt return to normal limits by the second week of the puerperium. Observations which were made on one normal patient before conception and at frequent intervals during the first few weeks of pregnancy shewed that a 5 per cent increase in total blood volume was present three weeks after conception and a 12.5 per cent increase at seven weeks gestation, the remainder of the blood volume curve following the course already described.

Total blood cell volume.

Hamilton and Thomson (1941) point out that, contrary to popular belief, there is an actual increase in the total mass of circulating red cells and haemoglobin during the latter months of pregnancy and hence in one sense no anaemia exists. They add, however, that on the basis of haemoglobin content per unit volume of blood there is an anaemia due to the fact that the increase between the plasma and cellular elements of the blood is disproportionate, with resultant relative hydration.

The factors producing the changes in blood volume during pregnancy are not clear. It may be as some

suggest that the natural loss at parturition is being anticipated, or that more maternal blood is needed to fill the enlarging vascular bed of the utero-placental circulation. These reasons Hamilton and Thomson (1941) state do not readily explain the early increase in blood volume before the utero-placental circulation is fully established nor the predelivery decrease during a period when the placental circulation is functioning to full capacity. The work of Brown, Henry and Venning (1938) on the excretion of gonadotropic substance suggests that this may, on the other hand, be the controlling factor. This hypothesis has been supported by the demonstration of Thorn and Harrop (1937) that both androgenic and oestrogenic substances exert a retaining effect on water and sodium.

The circulation.

There is a tendence MacKenzie (1921) states to a circulating stasis at the base of the lungs, which accompanies this widening of the chest and in the healthy heart in pregnancy crepitations may be present after lying in bed all night, to disappear, however, with a few breaths: and these were observed to return for a few days after the confinement. Venous stasis as seen in the legs during pregnancy were not, MacKenzie (1921) postulated, a sign of heart failure

but were the result of pressure on the veins in the pelvic or abdominal cavity and occurred in healthy people with efficient hearts. This tendency to venous stasis is thought to be accentuated by the relaxation of unstriped muscle which occurs in pregnancy and Burwell et al., (1938) describe the general distention of the venous system of the abdomen and neck which may appear, and which return to their usual degree of filling and visibility after pregnancy. These writers found, too, that in pregnant women by the beginning of the second trimester, the pressure in the leg was notably higher than that in the arm and it continued so throughout pregnancy: but after delivery, it was found to be no higher than the pressure in the arm; and they thus concluded that these changes were due to obstruction of the venous return by the pelvic pressure of the pregnant uterus. The distension and pulsation in the neck veins MacKenzie (1921) found to vary among healthy women and to be present without any sign of dilatation of the right heart, and he goes on to explain why there is no jugular pulsation during labour in the absence of acute failure - since there is a dilatation of the peripheral vessels with effort, the jugular veins do not receive a sufficient amount of blood for the movements occurring in them to be perceived: and,

further, the right auricle and veins leading into it act as a reservoir - although, again, the pressure in the veins and auricle are never enough to distend the ventricle and cause dilatation: and, therefore, MacKenzie (1921) says there is no jugular pulsation during labour, but it explains why the pulsation may return a day or two after delivery and then attain an even greater size, as he shews in his polygraphic records. He further dismisses these records of the jugular pulsation as being of no use in estimating the efficiency of the heart. Grienstein and Clahr (1937) in their circulation time studies in pregnant women were able to demonstrate a slight, but progressive retardation of flow of blood during the course of pregnancy and they thought that symptoms and signs which simulate cardiac weakness may be due to a sluggish circulation and increased pulmonary congestion. Harrison (1935) was able to produce dyspnoea and diminished vital capacity by injecting blood into the pulmonary artery of dogs. He attributed the dyspnœa to a vagal reflex excited by pulmonary congestion. The symptoms and signs which may simulate cardiac weakness during pregnancy, Greinstein and Clahr conclude, may therefore be due to increased pulmonary congestion and may be of respiratory rather than cardiac origin. In a further study by Clahr, Greinstein and Klein (1939) of the circulation time in pregnant

women with Rheumatic heart disease they came to the following conclusions:-

1. The arm-tongue circulation rate in pregnant women with well-compensated rheumatic heart disease is within normal limits.

2. The arm-tongue circulation rate in pregnant women with cardiac insufficiency is prolonged: although the arm-lung time remains within normal limits.

These observers were able to demonstrate a mild degree of cardiac decompensation in a rheumatic heart patient, who developed an upper respiratory infection and dyspnoea at the thirty-eighth week of pregnancy: and the following week when her respiratory condition cleared the arm-tongue circulation time estimation also improved.

The pulse rate.

That the heart could meet the extra demands made upon it by an increase in its rate has frequently been postulated but, because of the many complicating factors to an accurate estimation of this, such a change is not accepted. Burwell et al., (1938) however, calculated that the basal pulse rate is higher during pregnancy than after delivery: but the so-called Tuszkai sign of disappearance of the normal lability of the pulse during pregnancy has had no adequate fact or truth to support it.

The Blood Pressure.

Blood pressure changes consequent on pregnancy are similarly difficult to assess, since there are many factors both psychogenic and metabolic which may tend to produce an alteration, since these vary considerably in each individual, even in the same person at different times.

While, therefore, Hare and Karn (1928) state, after careful recordings, that the mean pressure below the general mean and during the last three months above it, gradually increasing to term, Burwell et al. (1938) assert that the basal blood pressure, particularly the diastolic phase, is lower during pregnancy than after its termination. It is true that many observations reveal a lowered blood pressure in the early months and a tendency for vascular tone to recover towards the end of pregnancy, but individual variations are so considerable that it is difficult to generalise with certainty.

Henry (1936) shewed that the blood pressure in normal pregnancy tends to be lower than in the non-pregnant state. This is especially true, he states, of the diastolic pressure and as a result the pulse pressure tends to be 10 mms. greater than in the non-pregnant woman. He regards this as a mechanism by which the heart is enabled to meet the increased demands made on it by the increase in blood volume and vascular area of normal pregnancy.

Thomson, Reed and Cohen (1939) in their studies on the circulation in pregnancy came to the conclusion that of previous investigations there are a number of possible errors due to the enlargement of the breasts obstructing the arm veins, contractions of the uterus (Braxton Hicks) being followed by a rise in the venous pressure to as much as 10 inches, the position of the arm - for example, internal rotation or abduction giving rise to falsely high readings, insufficient general relaxation of the patient. Eliminating all these possible errors these observers came to the conclusion that the arm venous pressure, as measured by the Mority and von Tabora method, is within "normal limits" during normal pregnancy. This corroborates the findings of Cohen and Thomson (1936), Landt and Benjamin (1936). Explaining the "Normal limits" Thomson Reed and Cohen (1939) further state that the venous pressure of normal pregnant women tends to diminish from early pregnancy to the sixth month, remain fairly constant throughout the remainder of the pregnancy, rises slightly in the early puerperium and returns to the early pregnancy level later post partum. The same trend as this latter, they add, is present in pregnant women with compensated heart disease. The measurement of venous pressure cannot be used, they finally state, to predict or diagnose early congestive heart failure in pregnant cardiac women.

Electro-Cardiographic Changes.

An accurate knowledge of the various types of cardiac arrhythmias dates from MacKenzie's (1921) researches with his polygraph and his accurate work was confirmed by the electro-cardiograph which is now used. Electro-graphic changes in the normal heart during pregnancy are said to be associated with the displacement and rotation of the heart, but they are not constant in the time or extent of their appearance. Feldman and Hill (1934) in an E.C.G. study of 36 pregnant women with normal hearts found a left axis deviation in 58.33 per cent of cases and it occurred during the time when the uterus was at its highest level in the abdomen: the smallest deviation in a counter-clockwise direction was 13^{o} and the largest 80^{o}, the average being 38^{o}. The axis shifted back to the right after delivery. Landt and Benjamin (1936) corroborated this left axis deviation and considered it due to the mechanical shifting of the heart: and Carr and Palmer (1932) in their E.C.G. study considered that the estimation of axis deviation, as an aid in the diagnosis of acquired valvular disease or congenital defects during pregnancy, is unreliable since they found that the axis tends to shift towards the left during the first two trimesters and to return towards the right during the third trimester and since accurate average corrections for this shift cannot be

made without doing great numbers of serial E.C.G. estimation throughout pregnancy. They add that inverted Lead III is of no significance, probably being due to transverse position of the heart: and, in association with Hamilton, (1933) these authors concluded that a large Q3, as defined by Pardee, occurring, it would seem, relatively frequently during pregnancy was not a reliable sign of heart disease in patients with normal hearts.

The clinical aspect.

Non-pathological arrhythmias.

Sinus Arrhythmia is less common during pregnancy, Jensen (1938) states, but it is at all events of no pathological significance: while extrasystoles MacKenzie (1921) found in over 50 per cent of 100 pregnant women whom he examined. In functional heart disease of pregnancy, Jensen (1938) found an even higher incidence but he quotes Frey as giving an incidence of only 4 per cent in organic heart disease. The condition is one, which occurring at rest with the slowing of the heart in those patients, calls merely for assurance and sedatives. An irregularity of the pulse is more likely to be due to sinus arrhythmia or premature beats and not auricular fibrillation or heart block. In a recent series of a hundred patients with heart disease Jones (1944) found premature beats

present in fifteen cases, auricular fibrillation in two cases and heart block in one case: and from the clinical aspect he observed that, on exertion, premature beats usually disappeared but that the irregularity of auricular fibrillation became most prominent and electro-cardiography was advisable in the doubtful cases.

Murmurs of the normal heart.

The heart murmurs, on the other hand, which may occur during pregnancy in the normal heart have been well known and extensively studied, though with an apparent uncertainty of their significance until MacKenzie's time. Jensen (1938) states that the incidence of functional murmurs in pregnancy varies with the assiduity with which the patients are examined. A large proportion of such murmurs are not incidental to pregnancy, especially those heard over the mitral area, while the typical gestatory murmur, Jensen (1938) states, is heard in the second and third intercostal spaces to the left of the sternum, and he further states that he sometimes found very loud and rasping murmurs accompanied by thrills in some cases. The variation in intensity in these murmurs can readily be demonstrated by examining the patient in different postures and they often disappear entirely, when the patient is sitting up: and MacKenzie (1921) states that they also varied

from day to day in their intensity. Sodeman (1940) thinks the rough gestatory murmur, which may appear over the base of the heart during pregnancy may be due to insufficiency of the tricuspid valve and hypertrophy of the heart with the strain of pregnancy. Changes in the cardiac tones, on the other hand, are frequent during pregnancy, states Sodeman (1940) with the mitral sound becoming louder or softer, more often the former and splitting of the apical first sound may be heard. Accentuation of the pulmonary second sound was found by Jensen (1938) to occur in 27-50 per cent of pregnancy women: while Sodeman found this change in 42.5 per cent of his series and considered it due to displacement and rotation of the heart. In an interpretation of these murmurs Jones (1944) states that the systolic heart murmurs, which are soft, short and localised to the apex or base should be ignored in the absence of other signs of heart disease: and the systolic murmur, which diminishes or disappears with change of posture can be disregarded. A loud, harsh or prolonged systolic murmur over a wide area is, however, usually a sign of heart disease. The extracardiac murmur, Jones (1944) says, is audible over a wide area, is equally loud everywhere over the heart and has often a palpable thrill: but with this exception, a systole murmur with a palpable thrill, he states, is always due to organic disease and indicative of an obstructive valve lesion, or a

congenital abnormality of the heart.

The changes in the cardio-vascular system during normal pregnancy are therefore numerous and often perplexing and in many normal women raise the question as to whether or not heart disease exists. It is conceivable, too, how the increasing load of pregnancy, causing such complaints as dyspnoea, especially towards the latter half of pregnancy, irregular action of the heart (extra systoles) and palpitations, may be a strain even to the normal heart.

THE INCIDENCE AND MORTALITY OF HEART DISEASE IN PREGNANCY.

In an analysis of nearly 9000 reported cases of heart disease in pregnancy Jensen (1938) concluded that about 1.15 per cent of all obstetric patients have clinical evidence of heart disease.

Among patients at their own clinic Jensen et al. (1940) found the incidence to be 1.27 per cent: Sodeman (1937) 0.82 per cent: Stander (1942) found heart disease present in 2 to 3 per cent: Fitzgerald (1935) 0.66 per cent: Daley and Strouse (1931) 0.25 per cent: Sparks (1929) 0.25 per cent: Fitzgibbon (1927) 0.16 per cent, while Lamb (1934) discovered heart murmurs in pregnant women to be 6.8 per cent, of which 4.8 per cent were functional and 2.06 per cent due to organic disease. In Sheehan and Sutherland's (1940) series 1.5 per cent of all obstetric patients had chronic valvular disease. Out of 29,713 patients who attended Queen Charlotte's Hospital during the years 1937 to 1946 inclusive, the total number with heart disease was 225, an incidence of 0.8 per cent.

The mortality rate, too, is high and occupies a prominent place among the causes of maternal deaths.

Jensen states that the general over-all figures are, however, a great improvement on previous ones, having fallen in recent years from about 8 to 10 per cent to 2 to 3 per cent and he attributes this to a greater appreciation of the danger and a better understanding of the heart problem, and to the more extensive antenatal care. As regards the importance of the latter, Lamb found that the death rate in heart disease was 2.7 per cent among those who had antenatal care, whereas it was 20 per cent among those patients who had not had such care: and Crighton Bramwell's (1935) figures shew that there is a death rate of almost 70 per cent amongst patients admitted as emergencies, as compared with a mortality rate of less than 3 per cent amongst those who had antenatal treatment. It is less common nowadays, however, for heart cases to be admitted as emergencies, that is in heart failure, and the supervision at antenatal clinics serves to lessen this catastrophe.

At the same time, while Jensen's (1938) evidence is that there has been this substantial fall in the death rate in pregnancy from heart disease, Hoffman and Jeffers, (1942) of the University of Pennsylvania, in a study of sixty-one fatalities of rheumatic heart disease complicating pregnancy, found the death rate

of two to three per ten thousand live births remained constant throughout the ten years which they reviewed, and that this offered a striking contrast, they state, to the total maternal death rate during the same period - this latter shewing a steady decline from 7.7 deaths per one thousand live births in 1931 to 3.1 per one thousand in 1940: and they considered that there was room for great improvement in the mortality figures from heart disease, regarding twenty of the sixty-one deaths, which they reviewed, as preventable.

The part played in maternal mortality by heart disease has, therefore, assumed bigger proportions owing to the spectacular lowering of deaths from other causes, so that Eastman (1936) believes that only sepsis and toxaemia claim more lives during pregnancy and the puerperium; and Jensen (1938) says that heart disease comes fourth or fifth among the fatal causes and is possibly the most important during labour and the puerperium, and he adds that it claims close to a thousand deaths per year in the United States. Stander (1942) found in his series of patients that heart disease was responsible for 10 per cent of all maternal deaths, and Munro Kerr and McLennan (1932) found that it causes 7.7. per cent.

Jensen (1938) found the mortality among the 9000 cases he reviewed to be 4.3 per cent; Sheehand and Sutherland (1940) gave a death rate of 6.3 per cent but state that 2.5 per cent of the deaths were due to reasons other than the heart disease, giving therefore a corrected mortality rate of 3.8 per cent. In the present series of cases the maternal mortality was 3.1 per cent.

TABLE I.

OTHER REPORTED SERIES OF MORTALITY RATES IN HEART DISEASE WITH PREGNANCY.

AUTHOR	Pregnant women suffering from cardiac disease.	Deaths during pregnancy or the puer-perium.	Percen-tage death rate.
Bramwell and Longson (1938)	350	26	7.43
Gilchrist (1931)	95	7	7.36
Harris (1937)	100	10	10.0
Henderson (1936)	35	2	5.7
McIlroy and Rendel (1931)	200	5	2.5
McClure (1936)	69	3	4.3
Nelson and Eades (1935)	495	23	4.64
Pardee (1937)	48	4	8.3
Reid (1930)	528	21	3.97
Turino and Antony (1938)	102	6	5.88
Naish (1937)	450	14	3.1

Sheehan and Sutherland (1940) 10.1 per cent. In the present series it formed 11.6 per cent of the maternal deaths from all causes during the ten years reviewed.

TABLE II.

THE RELATIVE IMPORTANCE OF HEART DISEASE AS A CAUSE OF DEATH IN OBSTETRICS .. Modified from Jensen (1938)

Cause of Death	British Maternity Hospitals	Queen Charlotte's Hospital	Hendry	New York City	Heyremann
Sepsis, puerperal and otherwise	90	11	8	510	80
Toxaemia	140	13	19	231	48
Obstetrical complications, including haemorrhage ..	58	15	28	488	24
Embolism	-	4	4	-	15
Acute infections, including influenza, and pneumonia	-	2	17	-	-
Abortion	-	1	-	262	-
Heart disease ..	36	7	5	173	24
Other causes ..	131	6	5	377	17
Totals	455	59	86	2041	208

TABLE III.

CAUSES OF MATERNAL MORTALITY FOR THE PERIOD SEPTEMBER 1st, 1932 to DECEMBER 31st, 1941. Stander H.J. (1942)

Sixty-seven deaths in 34,353 cases, as follows:-

	No. of cases	Percentage.
Infection (antepartum, postpartum, postabortal)	13	19.4
Pulmonary complications	10	14.8
Postpartum haemorrhage	9	13.4
Cardiac disease	7	10.4
Pneumonia	6	8.9
Toxaemia of pregnancy	4	6.0
Premature separation of placenta	3	4.5
Cerebrovascular accident	3	4.5
Pyelonephritis	2	3.0
Circulatory collapse	2	3.0
Post-operative Haemorrhage	1	1.5
Tuberculosis, miliary	1	1.5
Placenta praevia and antepartum haemorrhage	1	1.5
Chorionepithelioma	1	1.5
Blood dyscrasia erythroblastic splenomegaly	1	1.5
Psychosis, re-active panic (suicide)	1	1.5
Peritonitis, after appendicitis	1	1.5
Not determined, insufficient data	1	1.5
	67	99.9

Total maternal mortality, 1.98 per one thousand pregnancies.

TABLE IV.

CAUSES OF MATERNAL DEATHS (EXCLUDING ABORTIONS) IN ENGLAND AND WALES FOR THE YEAR ENDING 31st MARCH 1946.

REGISTRAR GENERAL'S REPORT.

	No. of deaths	Percentage.
Obstetric complications including haemorrhage	179	25.9
Toxaemia	163	23.6
Sepsis	110	15.9
Heart Disease ...	59	8.6
Lung infections (including Tuberculosis) ...	54	7.8
Embolism	40	5.8
Other conditions ...	85	12.3
TOTAL	690	99.9

TABLE V.

TABLE OF MATERNAL DEATHS FROM ALL CAUSES IN PRESENT SERIES.

Fifty-nine deaths in 29,713 patients, for the period 1937-1946.

	No. of deaths.	Percentage.
Obstetrical complications, including haemorrhage.	15	25.4
Toxaemia	13	22.0
Sepsis	11	18.6
Heart Disease	7	11.6
Embolism	4	6.8
Acute lung infections ...	2	3.4
Abortions	1	1.7
Other causes	6	10.2
	59	99.7

Total maternal mortality, 1.95 per one thousand pregnancies.

The other causes were:-

epilepsy, carcinomatosis following breast cancer, intestinal obstruction, uraemia, appendicitis, uncertain.

Of the fifteen obstetric complications, seven were due to placenta praevia and postpartum haemorrhage.

THE DIAGNOSIS

The diagnosis is a matter for careful history of previous health and thorough and, if necessary, repeated examination of the heart. Only a fully trained observer, aware of the variations from the normal which a healthy heart may undergo during pregnancy and who is experienced in the detection of the early lesion, the congenital murmurs and the various arrhythmias, can truly assess the condition. An early mitral stenosis, for instance, may easily be overlooked and the exact nature of a congenital murmur requires the most expert assessment. Reis and Frankenthal (1935) commenting on the variation in the incidence of heart disease in pregnancy consider that it is always lower where a Cardiologist is attached to the hospital staff. Sheehan and Sutherland (1940) said that the accuracy of clinical diagnosis was much greater in patients who had symptoms suggesting a heart condition, and they ascribed this to the fact that these patients were examined more carefully than the others. Examinations in their series were made with reasonable competence, but not by a cardiologist and they found that the clinical diagnosis of severe mitral stenosis was clearly more reliable than that of other lesions.

In the case of aortic lesions they state that the scarring of the cusps, seen post mortem, was not sufficience in many instances to produce very gross mechanical insufficiency and this may account for the failure to recognise an aortic lesion in so high a proportion of cases.

TABLE VI.

ACCURACY OF CLINICAL DIAGNOSIS

Sheehan and Sutherland (1940).

Pathological Findings	Clinical Diagnosis		
	Correct	Wrong	Unrecognised
Severe mitral stenosis ..	30	6	14
Slight mitral stenosis ..	6	4	7
Mitral incompetence .	3	3	7
Aortic lesions ..	3	5	25

Various authors including Hamilton and Kellog (1928), Jensen (1938) have commented on the inaccuracy of clinical diagnosis of heart disease in pregnancy, but Sheehan and Sutherland (1940) find it impossible to accept Fellner's (1901) conclusion on

clinical grounds that only one-seventh of the cases of heart disease in pregnancy are recognised clinically.

Jones (1944) states that the difficulty in a diagnosis of the early mitral lesion is such that it is sometimes to be suspected only by a loudness in the first sound accompanied by a rather sudden cardiac impulse, and in such a finding an attempt must be made to elicit the typical presystolic murmur by turning the patient on her left side or examining after exercise: since, too, the murmur may be heard only one inch across at, or just medial to, the cardiac impulse, it can be readily missed if the displacement upwards and outwards of the heart with pregnancy has taken place. Similarly in aortic incompetence, an increase in pulse pressure or striking arterial pulsation in the neck should lead to careful search for the characteristic high-pitched, soft-blowing diastolic murmur, usually maximum in the third or fourth left interspaces near the sternum. This murmur, Jones (1944) states, is rarely loud and is best heard when the patient stops breathing in full expiration, the most favourable position for her being either supine or erect.

Besides the heart lesions, there may be other signs and symptoms suggestive of heart disease. Dyspnoea on effort is an early symptom and usually

means that the patient is exceeding the restriction placed upon her efforts by the damage to her heart: cyanosis can be expected if there is slowing of the circulation, and oedema of the ankles and legs at the end of the day may also appear. A cough is common and, being one of the earliest symptoms of oedema of the lungs, necessitates careful and frequent examination of the chest: and at times the sputum may be tinged with blood, although a frank haemoptysis is usually a terminal sign in failure of the heart. Pain beyond a feeling of constriction and tightness of the chest is not common but may, by causing apprehension, tend to keep the patient from sleeping. Sleep itself is apt to be fitful and disturbed in the cardiac and, not uncommonly, the patient will admit to nightmares, which may be the result of a developing anoxia. The symptoms and signs of established decompensation with its marked dyspnoea, cyanosis, rapid pulse, spreading oedema of body and lungs, enlarged and tender liver and oliguria constitute an entity which is both ready to be diagnosed and one often avoided by antenatal care. Finally there is the patient without any history of rheumatic disease and unaware of any heart trouble, and in whom a routine examination leads to its discovery. A

careful and complete antenatal examination will reveal many asymptomatic lesions, which thus would not otherwise be suspected.

The differential diagnosis may have to be made from the following :-

1. Functional murmurs of the heart.
2. Symptoms associated with pregnancy.
3. Anaemia or toxaemia of pregnancy.
4. The effort syndrome.

Functional murmurs of the heart are not peculiar to pregnancy and may therefore precede it, but they may, on the other hand, make their first appearance then; and as Jensen (1938) states there is also a rough, basal murmur, systolic in time, which he ascribes as gestatory, which sometimes appears and may give a palpable thrill and is almost indistinguishable from congenital murmur. The aid of electrocardiograms and X-ray pictures may be required finally to place such murmurs; for instance, a definite enlargement of the left auricle seen in the X-ray film, in a patient with an apical systolic, would indicate mitral disease, and, with a basal murmur, enlargement of the pulmonary artery or right ventricle usually suggest the possibility of congenital heart disease.

Pregnancy causes a certain strain and embarrassment of varying degree in all patients and

the burden of increasing weight and an enlarging uterus is prone to set up more easily breathlessness and palpitations, while swelling of the ankles is common in pregnancy, and these changes are present with a normal heart. Anaemia and toxaemia of pregnancy may similarly be associated with breathlessness and oedema. Palpitations are often present in pregnancy and are due to premature beats or to the effort syndrome, and are rarely a symptom of heart disease. Premature beats usually occur when the heart is slow and therefore are noticed by the patient when she is at rest in bed.

Jones (1944) states that left submammary pain, breathlessness and palpitations may also be due to the effort syndrome. When dyspnoea on exertion is due to heart disease, it begins during exertion and is relieved by rest: in effort syndrome, on the other hand, although the patient may complain of breathlessness on exertion, further enquiry often reveals that the difficulty in breathing sometimes starts at rest, particularly if the patient is startled or worried. Such dyspnoea, too, varies from day to day, depending on the patient's mental state: whereas the exertion of dyspnoea from heart disease is constantly present whenever a certain amount of effort is undertaken. Therefore, as Jones

(1944) says, the first problem is to decide when such findings are the result of heart disease. One type of patient, for instance, who shews functional dyspnoea is the woman whose previous pregnancy had been a distressing experience to her, possibly associated with a difficult delivery and a still-born child, and she is therefore fearful of the future confinement.

In the vast majority of patients, a history of rheumatic fever can be obtained, to account for the heart disease: and in others, there is a vague uncertainty about growing pains in childhood, sore throats or scarlet fever having been present at some time or another, so that the incidence of rheumatic fever as the causal agent is probably higher than the figures given. A rheumatic fever history was found in their respective series by Stander (1942) in 95 per cent; Carr and Hamilton (1933) 94.4 per cent; Lamb (1934) 91.8 per cent; Gilchrist (1931) almost 90 per cent; Jones (1944) 87 per cent, and in the present series 93.0 per cent.

The mitral valve is the seat of the lesion in most patients.

TABLE VII.

VALVULAR LESIONS ENCOUNTERED IN RHEUMATIC HEART DISEASE.

Lamb (1937).

Valvular Lesion		Cases	Percentage
Mitral insufficiency	...	13	12.7
Mitral stenosis with and without insufficiency	...	76	74.5
Aortic insufficiency with mitral stenosis and insufficiency	...	10	9.8
Aortic stenosis and insufficiency with mitral stenosis and insufficiency	...	2	2.0
Tricuspid stenosis with mitral stenosis and insufficiency	...	1	1.0

Mitral Valve involved in 100 per cent.

Mitral stenosis with and without mitral insufficiency occurred in 87 per cent.

TABLE VIII.

TYPES OF HEART LESION IN THE PRESENT SERIES.

Mitral stenosis with or without mitral incompetence	174
Mitral stenosis, mitral incompetence and aortic incompetence	23
Aortic incompetence	2
Myocarditis	3
Auricular fibrillation	2
Paroxysmal tachycardia	3
Auricular flutter	1
Mitral incompetence	4
Congenital lesions	13
TOTAL	225

THE ASSESSMENT.

The patient's ability to go through pregnancy and labour with the minimum risk to life and health must be assessed with the greatest care. As previously described, pregnancy increases the work of the heart and places a strain on even the normal heart, so that it is conceivable that under such conditions slight overtaxing of the damaged heart may produce irreparable change and precipitate heart failure.

It was here, however, that MacKenzie's (1921) approach to the heart problem veered away from contemporary teaching to emphasize that although the damaged valve with a systolic murmur may be an indication that the heart has been invaded by a disease process, yet the estimation of the significance of those murmurs, as of all other signs, should be based not on the murmur itself, but on the functional efficiency of the heart and the presence or absence of additional symptoms and signs of cardiac disease. The functional efficiency of the heart muscle was to be estimated, therefore, on the patient's response to effort and each heart he found, had a certain reserve force, within the limits of which the patient must keep. The heart, he postulated has an inherent quality of adapting itself to the body's needs and has two forms of force: a rest

force, and a reserve force to accommodate itself to the extra work, which is used in increased effort: this reserve force is limited even in health, so that if exhaustion sets in there is distress - particularly dyspnoea. Therefore, with effort, the heart responds and continues to use its reserve force and so long as this is not exhausted, effort causes no distress. Undue effort, may thus not cause impairment but exhaustion of the heart. To illustrate this principle MacKenzie (1921) relates the example of an individual, who can walk a certain distance with comfort, but who, when carrying a heavy load will suffer breathlessness and distress before that distance is accomplished - his heart is not, however, in any way impaired but its reserve force is exhausted sooner. And so with the increased burden of pregnancy there is a tendency to use up the reserve force of the heart more quickly to cause a greater limitation of its field of response to effort but cardiac exhaustion may follow an excess of effort beyond this reserve, which if persisted in may lead to other signs of failure. The broad general principle stands out from this teaching, that those patients without much limitation of their heart reserve, as estimated by its functional efficiency, will go through pregnancy and labour without

undue risk: and this reserve of the heart can be measured by the patient's response to the effort of her daily duties. This was an important forward step by MacKenzie, (1921) from the undue significance previously placed on the type and loudness of the heart murmur, to a clinical assessment of the reserve force of the heart.

The next step MacKenzie (1921) took was of similar importance and significance. The practice of his time was to treat heart failure when it appeared and was already a fully developed entity; but by looking to the heart for the approaching signs of failure such a change might not be anticipated. MacKenzie (1921) realised that the first signs of heart failure were to be found not in the heart itself, but in the various organs and systems which it supplied and which, therefore, would reflect this failure and show diminished efficiency. Impaired circulation for instance reveals itself in a progressive oedema of the dependent parts and the stasis of the circulation in the vast capillary fields of the lung produces the early crepitation and cyanosis and aggravates the sensitive respiratory centre to cause additional rapid breathing: the blood supply to the heart muscle too, becomes defective causing a

sense of constriction and tightness across the chest. Undue exertion which requires an additional effort in force and speed by the heart may thus lead to impairment in blood supply to these organs, whose sensitivity to this lack produce the early unfavourable signs of oedema, cyanosis, breathlessness and constriction of the chest, which reveal themselves at the antenatal examination.

A major advance in the problem of the care of the patient with heart disease is, therefore, an avoidance of undue effort outside the limits of the reserve of the heart muscle and frequent examinations for the detection of those early signs of heart failure, especially the persistent crepitations at the bases of the lungs. Patients with such signs of failure MacKenzie (1921) would immediately advise to rest confining them to their beds and instruct them in deep breathing, to assist the right heart in maintaining an efficient flow through the lungs. The stage at which signs of failure appear in any one instance varies, since each heart has its own standard in health and disease and only constant care can reveal this individuality. The progressive signs of failure of the heart are to be seen in an acceleration of the pulse and an increase in size of the right

heart, in an oedema which reaches the abdominal wall and sacrum and in an increase in sputum, which may be blood stained. Fibrillation of the auricle may develop and at once be accompanied by worsening of the heart effort and circulatory flow, so that in a few days, or even a few hours, there is a great breathlessness at rest, lividity of the face, enlargement and pulsation of the liver.

Since the maternal mortality in heart failure is so much greater than in patients with a fully compensated heart, it is necessary to determine the degree of cardiac reserve in each individual and to anticipate, and if possible prevent, the development of heart failure. This estimation, as MacKenzie (1921) first shewed, can be made from the patient's response to the routine of daily life, and, although this is not an infallible guide, it has shewn itself to be so much better than any other single means of measuring the heart's response to effort and the degree of cardiac reserve present. On this principle, Jensen (1938) says the New York Heart Association based their classification of heart patients into four groups and in most American clinics today, this functional classification for patients with heart disease accepted by the American Heart Association,

is used as an index of whether women with heart disease will be able to withstand the strain of pregnancy and labour. Cases are, therefore, grouped according to the ability of the heart to withstand effort, rather than according to the structural damage and other factors. The grouping is as follows :-

Group 1. Patients with no limitations to normal active life; no symptoms of heart disease and in whom the only sign is the heart lesion itself.

Group 11. Patients with slight limitation of the amount of work done without breathlessness. Such patients are breathless at the end of effort, e.g., climbing flights of stairs they have to rest at the top, or they have to rest at the end of some routine housework.

Group III. Patients with definite limitation of the amount of work done without breathlessness. They have to rest two or three times whilst climbing a flight of stairs, or at intervals in their housework, and they stop to rest on their way home from shopping.

Group. IV. Complete limitation of the amount of work which can be done, so that there is heart failure at rest.

This classification has also been used in this country by Gilchrist (1931), McIlroy and Rendell (1931), McLennan (1933) and others, and it has been shewn to be a satisfactory guide, in the large majority of cases, to the patient's response to the effort of labour and it forms a valuable means of determining the help which may be necessary to effect delivery.

Bramwell and Longson (1938) classify their patients according to the estimated state of cardiac reserve, as judged from their history, prior to their becoming pregnant. Sheehan and Sutherland (1940) commenting on the American Heart Association classification, consider that it may have some significance when based on the patient's condition in the last quarter of pregnancy, but they say it is of relatively little value before the middle of pregnancy. Since, as mentioned, it is known that the patient's condition may change for various reasons during pregnancy such criticism would seem without point, Oppel (1940) found the classification invaluable in his management of heart disease in pregnancy. It is

true that the heart lesion itself being a progressive one and the patient's own response to the pregnant state a varied factor, the patient may start pregnancy in one group and deteriorate to another. This is, however, an accident which is largely avoidable by careful antenatal supervision and examinations at frequent intervals so that, for instance, should a Group II patient tend to regress into Group III she can, with prompt treatment, be brought back into Group II. This has also been noted by Lamb (1934), Pardee (1934) and Fitzgerald (1935); but Hamilton and Thompson (1941) go further and finding that 18% of their patients whom they classified as "favourable" changed to "unfavourable" during the course of pregnancy and, concluding that "any cardiac may fail at any time," recommended that all cardiac patients should be seen at weekly intervals throughout their pregnancy. Mendelson (1944), however, found no such instability of patients in this functional classification, which he considers very valuable in management and prognosis. That a patient's condition, originally placing her in the more favourable Groups I and II, can deteriorate during pregnancy, so that she becomes a Group IV case with heart failure and a risk of a fatal ter-

mination, has been seen in the series reported here (Case 2705/46 page 286) and in previous cases. As a general rule, and according to the finding of this series, however, the majority of cardiac cases do not vary much from their original classification, and a prediction as to their behaviour during the strain of labour can be made. It is, however, the occasional failure of the Group II patient which is a disturbing feature in assessing the cardiac case and suggests that some of those additional factors described are required for a more accurate prognosis.

Decompensation of the heart.

The additional factors which Lamb (1934) takes into consideration to augment the American Heart Association classifications are :-

1. The structural changes in the heart itself. These are judged by the size of the heart and the extent of the valvular damage.
2. The duration of the disease.
3. The presence of signs indicating activity of the rheumatic process.
4. The presence of auricular fibrillation.

As regards the size of the heart Lamb (1934) has used the cardio-thoracic ratio as an indication

of heart damage; this is difficult to judge with accuracy, however, because of the displacement and axial rotation of the heart which occurs in pregnancy. Jones (1944) considers that an X-ray picture of the heart is of value in the assessment, but Gilchrist (1931) found that the degree of cardiac enlargement is not always a helpful sign and says that some of the largest hearts may be quite capable of the strain of pregnancy and labour, while many of the smaller ones show evidence of failure. A routine picture of the size and shape of the heart is considered essential by the cardiologists, however, since as previously discussed, the heart murmur may be difficult to elicitate; and, as an example of this, one of the patients in this series (2705/46) in whom the heart murmur was difficult to elicit, shewed on X-ray considerable cardiac enlargement, which her Group IV symptoms suggested.

Lamb (1937) also emphasises the importance of a long rumbling diastolic murmur, existing throughout diastole, as indicative of a high degree of structural damage of the mitral valve and definitely increasing the risk of failure, and states that 62% of these women decompensated.

STRUCTURAL DEFECT.

Table by Lamb (1937) to shew the importance of the degree of structural damage to the heart valve as indicated by the type of murmur.

TABLE IX.

Relation of anatomic lesion to decompensation and death based on analysis of murmers - Lamb (1937)

	Number of cases	Number of decomp.	per cent of decomp.	Deaths	Post mortem.
Systolic at apex	13	0	0	0	
Presystolic or short diastolic at apex	45	7	15	3	
Presystolic or short diastolic at apex associated with aortic and tricuspid lesions	7	2	28	1	
Long diastolic at apex filling diastole	32	20	62	8	4
Long diastolic associated with aortic lesion	5	3	60	1	
Totals	102	32		13	4

Lamb (1937) concluded that there is a definite relationship between the extent of the structural damage in the heart valves and decompensation : no matter what the functional classification may be - if the size of the heart is more than 55 per cent of the total diameter of the chest, or if the patient presents a long rumbling diastolic murmur at the apex

the likelihood of a cardiac insufficiency developing during pregnancy is great. And in order to examine the accuracy of these statements patients in class I and II who decompensated were analysed according to the size of the heart and the presence of a long diastolic murmur at the apex - and all but one of these patients shewed either one or both of these signs. Sheehan and Sutherland (1940) also consider that there is a definite relationship between the mechanised severity of the stenosis of the valves and the tendency of the patient to become decompensated but, they say, this stenosis is difficult to assess clinically.

TABLE X.

Relation of the Cardiac Murmur to decompensation - Present Series.

Heart lesion	No. of cases	No. of decompensations (Group 3)	(Group 4)	per cent De-comp.	Deaths	Deaths per cent
Mitral stenosis with or without incompetence	174	35	11	26.4	6	3.4
Mitral stenosis mitral incompetence & aortic incompetence	23	1	1	4.3	1	4.3
Aortic incompetence	2	1		50.0		
Congenital Lesions	13	0	2	15.3		

Generally speaking, Lamb (1937) further states, the longer the duration of the disease, the greater the possibility of failure developing during pregnancy, and decompensation occurred in over 50% of the seven cases seen with active rheumatic disease.

AURICULAR FIBRILLATION.

MacKenzie (1921) identified auricular fibrillation as a clinical entity and studied its relationship to pregnancy. He stated that it is the form of irregularity, which is most commonly associated with heart failure and, observing the clinical changes, described the disappearance of the mitral presystolic murmur and the development of dropsy, pulmonary oedema and enlarged liver - stating that 80-90% of heart failure cases with the latter signs shew auricular fibrillation. He saw only six cases of auricular fibrillation during pregnancy and all had a history of rheumatic fever and limitation of effort with oedema of the lungs and premature labour: all lived through their confinement he adds, but none regained her previous health and all but one died within two years of the pregnancy. Auricular fibrillation, he, therefore, concludes, is a bar to pregnancy: and if oedema of the lungs, orthopnoea, or enlarged liver supervene, the pregnancy should be terminated.

These are still the views generally held except that some authors consider auricular fibrillation a constant danger and always advise termination of the early pregnancy. Gilchrist and Murray-Lyon (1933) consider auricular fibrillation a definite manisfestation of increasing cardiac embarrassment and often an indication of the early appearance of congestive heart failure, being present in about half the more severe cases of mitral stenosis.

Naish (1937) found auricular fibrillation to be rare, but occurring at an earlier age in parous than in nulliparous women, and being of bad prognostic significance in all patients. Lamb (1937) found the complication present in 7.8% of heart cases during pregnancy and stated that decompensation occurred in 75% of patients with it.

TABLE XI

Cardiac decompensation in relation to auricular fibrillation. Lamb (1937)

	No of Patients	Decompensation No. of cases During Preg.	Decompensation No. of cases After Preg.	Deaths No. of cases During Preg.	Deaths No. of cases After Preg.
Rheumatic	8	6	2	0	3
Arterio sclerosis	1	1	0	1	0
Time of decomp.		1 at 3 mo. 2 at 7 mo. 3 at 8 mo. 1-2 days P.P.	1-1 year P.P. 1-5 years P.P.		
Time of Death				1 During labour	1-5 mo. P.P. 1-1 yr. P.P. 1-5 yr. P.P.

The ages of the patients in the above table varied from 23 to 39 years with one exception - a girl of 19 years with active rheumatism.

They further stated that :

The duration of the heart disease from onset of rheumatic fever to onset of auricular fibrillation varied from eleven to twenty-three years. The

reason that auricular fibrillation is not encountered more often in pregnancy they suggest is that women in the child bearing age are not, as a rule, old enough for the development of this irregularity

De Graffe and Lingg (1935) have shewn that auricular fibrillation is usually a late manifestation in rheumatic heart disease as it was most commonly observed in the longstanding cases: and they found that auricular fibrillation is usually associated with heart failure and of 8 cases in rheumatic women, 6 decompensated.

In the present series five patients with auricular fibrillation were seen, two in Group IV one of whom, Gravid 1, aet. 23, was treated by hysterotomy and sterilisation and the other, Gravid 1, aet. 32, by Caesarean section and sterilisation, their condition being only fair on discharge: there was one Group III patient, Gravid 2, aet. 34, who had a normal delivery: the remaining two had heart failure and died in the puerperium, one, Gravid 1, aet. 23, following Caesarean section, the other, Gravid 1, aet. 25 following premature labour at 36 weeks with spontaneous delivery.

Finally, in a resume of auricular fibrillation complicating pregnancy Jones (1944) considers it a grave complication, and states that of 85 recorded

cases 35 died during or soon after pregnancy. Bramwell and Longson (1938) describe 17 cases in which fibrillation was present when first seen and in 8 of which therapeutic abortion was performed without a death: of the remaining 9, - 4 died - one from cerebral embolism, and 3 from heart failure. They considered that a fatal issue might have been avoided by adequate supervision and concluded that under the best conditions it was sometimes justifiable to allow pregnancy to continue even in the presence of fibrillation; but unless the circumstances are exceptionally favourable they consider it best to perform a therapeutic abortion early in pregnancy.

Sheehan and Sutherland (1940) found that women with auricular fibrillation died at a much younger age than women suffering from chronic valvular disease without decompensation and they give the following ages at death of their 12 cases :-

Age at Death	Cases
20 - 25	4
26 - 30	6
over 30	2

A G E.

Cardiac failure in relation to Age.

The greater chance of auricular fibrillation appearing in the older patient, as shewn by Gilchrist and Murray-Lyon (1933), may form one reason why age may unfavourably influence the course of pregnancy.

Hamilton and Carr (1933) assert that patients 35 years or older are twice as likely to fail under the same conditions as those under 35 years of age: and Lamb (1934) considers the duration of the disease to be a factor.

Gorenberg and McGleary (1941) give the following table :

TABLE XII.

Cardiac Failure in Relation to Age.

Years	Total	Failed
Less than 20	32	4 - 12.5%
21 - 25	135	13 - 9.6%
26 - 30	100	26 - 26.0%
31 - 35	49	16 - 32.6%
36 - 40	25	14 - 56.0%
More than 40	4	4 - 100%
	345	77 22.3%

In general they say the older the cardiac patient the greater the possibility of a breakdown occurring in the pregnant state.

The minor discrepancy shewn in the less than twenty group is possibly due to the relatively small number in this class.

Further the failure rate in the less than 30 age group was 16.1 per cent compared with 43.6 per cent in the 30 years and over group.

The older groups they conclude contained the higher percentages of functionally bad hearts.

Sheehan and Sutherland (1940) give the following table :

TABLE XIII.

Relation of Age to Decompensation

Sheehan and Sutherland (1940).

	Age		
	16-25	26-35	36-45
Not decompensated			
Non-cardiac death	3	15	10
Cardiac death	2	3	5
Slight decompensation			
Non-cardiac death	0	1	3
Cardiac death	3	10	1
Severe decompensation			
Cardiac death	4	8	2
Control obstetric patients without any valve lesion	190	303	212

The age of the patient they conclude does not appear to have much influence in decompensation.

TABLE XIV.

Decompensation in Relation to Age - Present Series.

Years	Total in series	Group III	& per cent	Group IV	& per cent
20 and less	13	1	7.7	1	7.7
21 - 25	59	5	8.5	4	6.8
26 - 30	85	14	16.5	4	4.7
31 - 35	44	12	27.3	6	13.6
36 - 40	15	5	33.3	0	
more than 40	9	0	-	2	22.2
	225	37		17	

It will be seen from the above table that the average decompensation rate in patients in the 30 years and below age groups was 8.6 per cent as compared with 24.1 per cent in the over 30 years groups.

Small statistical figures cannot be taken as a true guide, since should it be deduced from the above table that patients in the 30 years and below group ran less of a risk one has only to consider that the average age of the fatalities in the present series was 27.6 years.

Jensen (1938), perhaps correctly, surmises that the evidence available does not suggest that decompensation of the heart is a phenomenon specifically related to pregnancy.

TABLE XV.

Time of Decompensation.

Relation of Decompensation to Duration of Pregnancy - Sheehan and Sutherland (1940).

	Duration of Pregnancy in weeks			
	To 19	20-29	30-35	36 and over
Not decompensated.				
Non-cardiac death	2	1	8	17
Cardiac death	1	1	1	7
Slight decompensation				
Non-cardiac death	0	0	1	3
Cardiac death	0	1	5	8
Severe decompensation				
Cardiac death	0	3	6	5
Control obstetric patients without any valve lesions	56	42	106	501

There is state Sheehan and Sutherland reviewing the above table apparent tendency for patients with decompensated hearts to die or to be delivered before the last month of pregnancy.

Gorenberg and McGleary (1941) consider that 80 per cent of the cardiac failures in pregnancy occur during the first 8 months.

TABLE XVI.

Cardiac Failure in Pregnancy. Time of Occurrence - Gorenberg and McGleary (1941).

Time	No.	Per Cent
1-6 months	40	51.9
7-8 months	22	28.5
9 months	8	10.3
Labour	5	6.4
Puerperium	2	2.5
	77	

Gorenberg and McGleary (1941) conclude therefore that from the clinical standpoint cardiac difficulties begin when pregnancy begins and reach a maximum point of intensity 4 - 6 weeks before term.

Therefore therapeutic measures to avoid decompensation must they insist be instituted in the beginning of pregnancy.

They have been so impressed by the improvement shewn by rheumatic heart patients during the last month of gestation that they now anticipate such amelioration: and this further confirms the improvement suggested by the physiological changes.

P A R I T Y.

The relation of Parity to decompensation.

McIlroy and Rendel (1931) found that limited cardiac reserve was more common amongst multiparae. Jensen (1938) states that this idea was introduced by Fritsch (1876) and has survived with exceeding tenacity and has been so taken for granted that few had seriously questioned it.

Jensen (1938) supplied figures sufficiently large to be reliable, and came to the conclusion that increased parity had no effect on the death rate.

TABLE XVII.

Effect of Parity on the death rate: Jensen (1938).

Parity	1	2	3-5	5 and over
1100 surviving cases	37.64	20.64	27.73	14.00
461 fatal cases	40.13	18.44	25.38	16.05
Difference	-2.49	2.20	2.35	-2.05
Standard error of difference	±2.66	±2.65		±1.93

Sheehan and Sutherland (1941) consider that there is no relation between the parity of a patient and cardiac decompensation.

TABLE XVIII.

Relation of Parity to decompensation -

Sheehan and Sutherland (1941)

Decompensation		Parity 1	2 and 3	4 and over.
None				
Non cardiac death	....	10	6	12
Cardiac death	....	3	3	4
Light				
Non cardiac death	....	0	1	3
Cardiac death	....	10	2	2
Severe				
Cardiac death	....	6	3	5
Control obstetric patients without any valve lesions	....	282	160	254

TABLE XIX.

Relation of Parity to decompensation - Present Series

	Gravid					
	1 : total No. 133		2 and 3 total No.65		4 and over total No.27	
Group	Decom-pensated	per cent	Decom-pensated	per cent	Decom-pensated	per cent
III	16	12	16	24.6	5	18.5
IV	13	9.8	4	6.2	-	-

From a small series of cases deductions can be erroneous but when the above table is considered with the knowledge that all 7 deaths reported in this series (page 118) were in primigravidae it cannot be deduced that parity plays a role in determining cardiac decompensation.

THE MANAGEMENT.

Medical Treatment.

The patients should be advised to report at frequent intervals for examination and advice, at least every two weeks during early pregnancy and weekly after the child has reached viability. It has been frequently observed that about the twenty-sixth to the twenty-eighth week of pregnancy patients complain of more breathlessness and limitation of their physical activity than before and, although this may be due to the embarrassment of the growing uterus, rest at this period of pregnancy achieves remarkable response with improvement in the patient's condition. It might be considered advisable, therefore, that each patient be admitted for one week during her pregnancy for observation and assessment, and that the 28th week would be an advantageous time.

The medical treatment concerns itself with the patient obtaining the maximum amount of rest and working only within the limits of her ability, avoiding breathlessness and exhaustion. An adequate iron intake should be ensured in order to avoid anaemia, which of itself will add to breathlessness and cyanosis: and respiratory infections, which at other times might be considered trivial, should be treated

with the utmost care. Both Stander (1942) and Jones (1944) stress the bad influence of anaemia and lung infection and Mendelson (1944) admits to hospital a patient with serious upper respiratory infection. Jones (1944) states that bronchitis and influenza are often the precipitating cause of heart failure: and since these infections are common, twenty per cent of the patients in his series developing upper respiratory infections, it is wise to warn the patient of this risk and confine her to bed at once, to stay there until she is better. Stander (1940) goes so far as to state that upper respiratory infections are such an ominous development that they may be the first signal in a break in compensation. McKenzie (1921), too, devoted much time and care to examination of the chest, looking out for the appearance of crepitations; and it may be that such assiduity on the part of the physician will enable him to anticipate any change towards decompensation of the heart which may be threatening. The medical care, then, largely resolves itself into the prevention of cardiac decompensation and although this may creep on slowly, Carr and Hamilton (1933) state that in the majority of instances it occurs quickly as the result of over exertion, such as

prolonged housework and shopping expeditions, or from an intercurrent infection as mentioned above: and they go on to say that these causes are largely preventable by a stubborn medical control of the patient, fitting a daily routine to the case and modifying it to individual requirements and visiting the patient to see that she strictly observes all instructions; as regards the onset of respiratory infection, they state that if the patient with cardiac disease goes to bed at the first signs nothing untoward happens as a rule, but if she stays up on her usual regime she may develop heart failure. Clear instructions should, therefore, be given about dangerous subjective symptoms, such as a cough or haemoptosis, and since they may mean a failing heart the patient is asked immediately to report them. To ensure this, the writers advocate a weekly visit, whether apparently needed or not; and they finally add, that the earliest sign of failure in their opinion, too, is persistent râles at the lung bases.

Fitzgerald (1935) recommends immediate hospitalisation of the patient if she is breathless, or unable to do light household work without breathlessness, if there is a persistent cough, or if the pulse is over 100 or if she needs more than one pillow to sleep: he advocates too a social service which visits patients in their homes advising the patient on the

care of her children and arranging for such help which may be necessary and discussing financial arrangement especially free hospital treatment in order to remove all unnecessary worries. Hamilton (1947) considers that the reduction of the maternal mortality requires a great deal more time and money spent on the antenatal care of the patient and also further stresses the importance of the same daily regimen and a lessening of effort if breathlessness appears. Sheehan and Sutherland (1940) found in their series that about half of their patients who had congestive heart failure in pregnancy gave a history of some dyspnoea before its onset, although perhaps it was never severe, and it tended to become worse in the first few weeks of pregnancy. The majority of the remaining half, they state, developed the congestive failure either in the first twelve weeks of pregnancy or at about twenty-four to thirty weeks, and in these the onset was rather sudden so that the patient was unable to date it.

Decompensation, therefore, as stated, is best avoided by careful and frequent examination of the patient, by regulating her routine of daily life within the bounds of her ability, avoiding tiredness and breathlessness, and by securing her full co-operation in this and in the reporting of any

departure from the normal in her health. For the established heart failure, complete rest in hospital, where full nursing care is obtainable, is a prime necessity and it is a moot point if such a patient, having recovered, should be allowed home without the observation of a trained staff until she has been delivered.

If heart failure has set in and is attributable to auricular fibrillation confinement to bed and treatment with digitalis are indicated. Bramwell (1935) recommends 20 minims of the tincture or 2 grains of the powdered leaf given three times a day will suffice, but in cases of extreme urgency three doses each of 1 drachm of the tincture may be prescribed at intervals of six hours; alternatively 1/100 grain strophanthin may be injected intravenously and repeated in two hours if necessary. Care is taken, Bramwell continues, if the massive dose method is used, that the patient has not been taking the digitalis group drugs during the preceding three weeks and toxic symptoms such as nausea, vomiting, coupling of the heart beats and oliguria must be watched for.

Opinion is divided on the value of digitalis in heart failure with normal rhythm. Bramwell (1935) however is convinced that it does good, although not giving the dramatic results seen in patients with

auricular fibrillation, especially if used in small doses such 10 minims of the tincture, or 1 grain of the powdered leaf, or 1/600 grain Nativelle's digitaline, given twice or three times a day: there is also a place for such cardiac stimulents as coramine and cardiazol 1 cc of either drug injected every 4 hours.

Sedatives to lessen anxiety and relieve undue restlessness are always indicated and the Bromides are well tolerated, morphia being reserved for actual pain such as may accompany enlargement of the liver. Diuretics to remove oedema are often dramatic in their action and it is revealing to watch the improvement they obtain: diuretin 10-15 grains three times a day or Mersalyl 0.5 cc increasing to 1 cc and to 2 cc daily for a few days; and the salt intake is restricted. Oxygen may be required and a B. L. B. mask is usually well tolerated for short periods: and bronchitis is helped by a suitable stimulant mixture. Finally, tonics of iron and arsenic and a little wine with dinner do much to revive the flagging spirit.

A routine course of instructions and procedure is followed in the present series on all heart groups.

1. The patient is asked to take the maximum rest possible during pregnancy - at least two hours in the afternoon lying on her bed, and twelve hours nightly.

2. She reports for fortnightly examination until the 28th week of pregnancy and then at weekly intervals.

3. There is immediate admission of all group III and IV cases.

4. Instruction is given to the patient to avoid chills and upper respiratory infection and to report them immediately should they occur. Anaemia is avoided by the free use of iron.

5. All patients are admitted to hospital for one week - preferably about the 28th week - for observation and assessment and one week before delivery for rest and reassurance.

OBSTETRIC TREATMENT.

A B O R T I O N.

Spontaneous abortion is not more common in patients who have heart disease. Mendelson (1944) for instance gives the incidence as 4% in groups III and IV as compared with 5% of his total clinic patients. In Stander's (1942) series of cases, spontaneous abortion occurred in 5.1% of the patients, and his total clinic incidence was 6.8%. Malpas (1938), in a careful study of abortion sequences did not mention heart disease among the defects found in women who had two or more successive spontaneous abortions.

Therapeutic Abortion: Jensen (1943) states that only when medical treatment has failed or when mild decompensation remains, after the physician has done his best, should interruption of pregnancy be considered. At the same time, he says, the outlook for the patient is profoundly affected by the appearance of decompensation and generally speaking, the earlier it appears, the less readily does it respond to treatment. It is also true that the greatest strain upon the heart is seen in the latter half of pregnancy, and nice judgment must be shewn in deciding to allow the patient in groups III and IV to

proceed with the pregnancy. Should the decompensation be caused by an acute cold, or transitory strain, Jensen (1943) considers the outlook better, since compensation is readily regained when the cause is removed. Generally speaking, in early pregnancy a group III patient who fails to respond to medical treatment, and group IV patients should be advised to have the pregnancy terminated.

The previous history of the patient is of value in the doubtful Group III case. Should she reveal, for instance, that during a previous pregnancy there was cardiac decompensation, Gilchrist (1931) says there is a likelihood of a recurrence of this complication with future pregnancies; and Lamb (1937) considers that a history of cardiac decompensation, apart from one associated with a previous pregnancy, is an indication for its termination. Maurice Campbell (1926) reviewing Hunt's (1926) cases found that of the patients with a history of previous heart failure 50% developed failure during their present pregnancy as compared with 17% of failures amongst those with no such history.

All authors are agreed, as previously mentioned, on the dangers of auricular fibrillation, and, although it may be controlled, there is a grave risk in

continuing with the pregnancy, and in eight patients observed by Lamb (1937), six became decompensated. The duration of the disease, he states, is not an indication for interruption, unless there are other signs of extensive heart disease. Active rheumatic infection during pregnancy is not common, but Lamb (1937) states that over 50% of patients with this complication failed during pregnancy, and he advocates its prompt termination; he also advocates that a heart size in excess of 55% of the diameter of the chest is an indication for terminating the pregnancy.

Jones (1944) advises a patient against having a third pregnancy, and states that it is often necessary to recommend termination, if the patient is seen earlier than the twelfth week; when pregnancy is already advanced, however, he considers the dangers of therapeutic abortion little less than those of allowing the pregnancy to continue. The figures in this series do not bear this out and there is no doubt, as Gilchrist (1931) says, that although the pregnancy may not end fatally in groups III and IV patients, the risk of permanent increase in damage to the heart is great, so that a further inroad is made on the cardiac reserve and the response of the heart to effort is even more limited after pregnancy than before.

Lamb (1937) found the greatest number of decompensations occurring in the latter months of pregnancy; and since, as Stander (1942) has stated, the patient is to expect the impact of a heavier burden and cardiac strain as pregnancy progresses, should she shew signs of failure in the early months, the likelihood of another breakdown is ever present. Such a patient, with a history of failure, should therefore be seriously considered as endangering her life to continue with a pregnancy, although a temporary improvement is possible in her condition.

Before termination of pregnancy is undertaken, the patient should be brought into as good a condition as possible, and this includes a period of complete rest, thorough digitalisation of the heart, adequate sedation to ensure sleep, and the treatment of any respiratory tract infection; if there is oedema, fluid and salt intake are restricted, and mercurial diuretics are exhibited. Intervention thus becomes a reasonable risk and, if performed by the vaginal route, is relatively safe. Hysterotomy, however, does carry a definite risk, so that in the first three months evacuation by the vaginal route is advisable, and a decision is made, if possible, early enough to allow this to be carried out. There

were twelve hysterotomies in this series an incidence of 5.3 per cent one in group I - a patient gravid 3, with mitral stenosis and aortic regurgitation who did not respond to medical treatment and died three weeks after the operation; six cases in group III: and five cases in group IV - with one dead in this group - a mortality of 8.3 per cent for the series. In groups III and IV of the series reported here, the maternal mortality rate was 14.2 per cent in those proceeding to term, suggesting, therefore, that this is the greater risk to the patient. Fatal cases amongst hysterotomies doubtlessly include patients who would, in any event, have died from heart lesion, since, as Jensen (1938) states, no matter what the treatment, some patients will become worse and die; and patients have been seen, who succumbed to the heart disease as early as the sixth or eighth week of pregnancy. Should hysterotomy be performed, the indications are present for simultaneous sterilization.

CARE OF THE PATIENT ACCORDING TO THE CLASSIFICATION OF THE AMERICAN HEART ASSOCIATION.

Group I. Normal delivery at term can be expected.

Group II. Normal delivery at term, aided by forceps if there is delay in second stage.

Group III. Forceps delivery early in the second stage, unless advance is rapid and delivery quick. The large majority of patients in this group, however, with careful medical treatment during their pregnancy, can be brought into Group II and so treated. It is common to find that many of the patients in this group have been doing too much work, and complete rest in bed produces marked improvement. Once a patient has recovered from cardiac decompensation it is important that she should be closely watched for the remainder of her pregnancy and in many cases it is wise to keep her in hospital. Should there be a failure to respond to treatment in early pregnancy, therapeutic abortion should be advised.

Group IV. No obstetrical treatment of any kind should be attempted until the patient is thoroughly rested and the heart fully digitalised, as the patient in such an extreme stands interference badly.

After full rest and improvement, termination of an early pregnancy is advised; seen in the latter part of pregnancy, complete rest in bed should continue, and a spontaneous vaginal delivery, aided by forceps, is considered as the best method of delivery for the large majority of these patients.

There is an unfortunate tendency amongst patients in this Group to go into premature labour before medical treatment has had a chance, and in such a situation the patient is a bad surgical risk and it is safer to allow her to continue with a well-sedated first stage of labour and to deliver her with froceps, than to submit her to an abdominal operation.

In the Group IV case late in pregnancy, however, where after a period of medical treatment the progress is not maintained, or is stationary and an optimum time in

Group IV. (Cont'd)

her improvement is considered to have been reached, termination of the pregnancy may be advisable. In such an instance, Caesarean section is likely to be less of a risk than a delayed labour from an artificial induction.

COURSE OF LABOUR IN HEART DISEASE.

There is a definite course of labour peculiar to the patient with heart disease, which allows the great majority to have a spontaneous and easy delivery.

The Maturity.

It is a general rule that patients with heart disease are not late in going into labour. The average length of pregnancy (omitting the hysterotomies and Caesarean Sections) in the present series was 39.1 weeks. Prematurity is likewise more common in the Group III and Group IV patients, in whom the average duration of pregnancy in this series was 38.1 weeks.

Length of Labour.

McLennan (1933) states that labour in a patient with heart disease is frequently short and precipitate, and in his series the average duration of labour in primiparae was slightly less than eight hours, and in multiparae, six hours.

Both Reis and Frankenthal (1935) and Fitzgerald (1935) also report a short duration of labour in patients with heart disease.

Reis and Frankenthal (1935) :-

Average total length of labour in primigravidae - 12 hours.

Average total length of labour in multigravidae - 7 hours 24 minutes.

Fitzgerald (1935) :-

Average total length of labour in primigravidae - 10 hours 50 minutes.

Average total length of labour in multigravidae - 5 hours 40 minutes.

Mendelson (1944) asserts, however, that his data do not support the statement that patients with heart disease have short labours. He gives, further, the following figures :- Average duration of labour in Group III and IV patients was 17 hours for Primiparae and 10 hours in Multiparae and that dispite the fact that 50 per cent of the deliveries were by forceps. The average length of labour in healthy patients he gives as 18 hours in primiparae and 12 in multiparae. Both Carr and Hamilton (1933) and Nelson and Eades (1935) say that labour in the patient with heart disease is neither longer nor shorter than in other women.

In the present series the average length of labour in primigravidae, first stage, seventeen hours; second stage, one hour seven minutes; and in cases of forceps delivery, second stage, one hour four minutes. In multigravidae the average length of labour was, first stage, six hours fifty-seven

minutes; second stage, twenty-four minutes, forceps being used in only one instance.

While it may be considered therefore that the total length of labour in women with heart disease is the same as for the average patient it would appear that the second stage is shorter but even more important is the clinical fact that it is typically accomplished with ease.

Weight of the Babies.

The average weight of babies in this series was seven pounds four ounces, which, considering the prematurity, shews that they are well up to average size. In Mendelson's (1944) series, the average weight of babies in Groups III and IV patients was 3,440 grams and that of babies of the total clinic population was 3,428 grams.

There is evidence for believing therefore that the babies are not small in patients with heart disease.

The Still-birth rate and Neo-natal death rate.

The still-birth rate in the present series of cardiac patients was 2.8%, including one case of antepartum haemorrhage, and the neo-natal death rate was 2.3%, which compares well with the average rates for all patients over a period of ten years

which were : still-births, 2.9%, and neo-natal deaths 1.6%. The higher neo-natal death rate associated with the cardiac patients may, at least in part, have an explanation in the frequency of premature labour. There was, however, only one neo-natal death, in one of twins born at the thirty-fourth week, among the ten Group IV patients who went beyond the twenty-eighth week and were delivered.

In a study of the foetal mortality in patients with organic heart disease, Teel (1935) states that the greatest foetal loss occurs in that rather small group of patients with severe heart lesions, most of whom, if they had sought medical attention, would have been advised that their heart reserve was too low to withstand the strain of pregnancy. On the other hand, patients with well-compensated heart disease and without history of past failures or embarrassment seem to tolerate pregnancy well, under strict medical regime; and for this group, the foetal mortality does not differ materially from that of normal subjects. In the borderline group whose past histories reveal no failures or loss of reserve and who fail for the first time in the middle of pregnancy, Teel (1935) states that their babies are extremely important, since their cardiac condition may deny them a future pregnancy and a decision may have to be made to terminate the pregnancy.

He estimates that, if the date of confinement is known, the thirty-sixth or if possible the thirty-seventh week offers a more certain prognosis than estimation of the size of the baby and after the thirty-fifth week of pregnancy the results are much brighter. Of fifty-five of his patients delivered in congestive heart failure, only five babies were still-born. He considers that pelvic delivery is the choice for the baby but adds that he hesitates to induce labour, since it may be long and tiring and a serious ordeal for the cardiac patient: on the other hand Caesarean section offers the poorest chance for the premature baby, but in those few cases which cannot wait until term it must be performed for the mother's sake. As far as full-term babies of compensated cardiacs are concerned, Teel (1935) says that normal delivery, low forceps and Caesarean section all offer an excellent prognosis, but in his series low forceps had the lowest foetal mortality with only two still-born babies and no neo-natal deaths in two hundred and ten deliveries.

Management of Labour.

In order to prepare for labour and to anticipate its outcome, the patient should be admitted to hospital at least one week before the estimated delivery date and have the further benefit of rest, reassurance and of accommodating herself to her surroundings.

The 1st Stage of Labour.

When no obstetric complication exists, she is allowed to go into labour spontaneously in full confidence of a successful outcome. Morphia is the drug "par excellence" for the cardiac patient and is given as soon as labour is established, being repeated as necessary and with such adequate sedation that the first stage of labour passes off uneventfully.

Many of the maternal exhaustions at this stage are due not to effort or straining but to an unnecessary worry about its progress and outcome and for this Morphia is of especial value. Uterine inertia and mental worry are both dangerous to the patient with heart disease and may of themselves cause an increase in pulse rate and breathlessness.

These anxieties of labour are best met and reconciled in the antenatal period by a brief and

simple explanation of how the baby is born and also by a few attendances at the physiotherapy clinic where the patient is taught how to leave her muscles relaxed, a practice which relieves the pains of labour and allows smoother advancement of the foetal head and promotes intelligent co-operation and a calm assurance.

Vaginal examinations are limited in order not to disturb the patient unduly and need be made only when the membranes rupture, and, if necessary, to confirm full dilatation of the cervix. As with normal labour the patient need not stay in bed at this stage unless she has been on a strict-bed routine prior to labour. Should the presentation be a vertex posterior one a longer and sometimes painful first stage of labour is experienced so that adequate nutrition with glucose drinks, bread and butter and sweet tea and milk puddings should maintain the patient in good condition: and pethidine gr 1/200 has been given towards the end of this stage with good results. The pethidine has not such a strong respiratory depressant action as morphia: but it has been noticed that doses of gr 1/100, cause difficulty in establishing respirations if the child is born within an hour or two of the injection and if the child is premature.

Mendelson and Pardee (1942) advised, as a means of anticipating or preventing cardiac failure, immediate digitalisation of the heart should the pulse rate rise above 110 or the respiratory rate above 24 during the first stage of labour; and they state that such rises in pulse and respiratory rates preceded each instance of cardiac failure amongst their patients, whilst no case of failure occurred in those patients with pulse and respiration rates below these levels during the first stage of labour, regardless of the severity of the cardiac condition as indicated by the American Heart Association Classification.

The 2nd Stage of Labour.

The accoucheur is present throughout the whole of the second stage and, should there be undue delay or straining, a forceps delivery can readily be performed.

The form of anaesthesia well suited to the patient with heart disease is the local pudendal block which is as a rule sufficient to obviate the use of any analgesic - although some patients find it comforting to hold a mask over their faces supply- a little gas and oxygen.

This local anaesthesia is as a rule sufficient

for low forceps application should this be deemed necessary - but may be supplemented by a little gas and oxygen.

The pudendal block technique, as used at Queen Charlotte's Hospital, is usually combined with a local infiltration of the perineum, as follows : the sterile-gloved finger is introduced into the vagina to palpate one of the ischal spines, the left index finger for the left spine, and a long fine needle is inserted through the skin of the perineum and up the post-lateral aspect of the vagina to the region of the ischal spine and the area of the pudendal nerve suffused with 10 c.c.'s of ½% novocaine; using the other index finger as a guide, the other pudendal nerve is anaesthetised; 5 c.c.'s of novocaine is then injected subcutaneously backwards from the posterior fourchette on either side of the midline to catch the sacral nerve supply; finally (and not always a practice) 5 c.c.'s of novocaine are injected over the inferior ramus of the pubis of either side, two inches from the midline, to catch the fibres of the ileo-inguinal nerve, the whole procedure taking only a few minutes.

In the patient with heart disease there is typically an easy effacement and dilatation of the of the cervix, followed by a quick descent of the

head without undue straining. This softness and succulence of the cervix, with a ready resiliency of the pelvic tissues may be due to an increased local congestion and may explain, too, how labour in those patients is often accomplished with surprising suddenness and absence of strain - and this fact is especially notable in the Group III and IV patients. It is common therefore to find the foetal head on the perineum at the beginning of the second stage of labour - or descending quickly after a few pains so that the admonition so often given that the 2nd stage of labour should be eliminated by forceps is not a practical issue. Any delay in delivery at this stage may be overcome by an episiotomy: or if there is no advance it may be eased gently over the perineum by forceps - the pudendal block anaesthesia being sufficient for the purpose.

It is unwise to eliminate the 2nd stage of labour, if that means the application of high forceps. This procedure can cause shock to any patient and those with a heart lesion stand shock badly: the head should, therefore, be given time to mould and descend if at all possible. Likewise, any form of accouchement force such as manual dilatation of the cervix may be attended by sudden and severe shock. In one patient (1002/1943), whose

record is reported (page 121) there was a fatal ending following this procedure.

The 3rd Stage of Labour.

The third stage of labour is conducted in the same way as for normal patients. Separation of the placenta is waited for and the patient allowed to push it out at the end of 20 minutes. Mismanagement of this stage such as associated with too early or repeated attempts to expel the placenta or vigorous Crede manoeuvres may give rise to dangerous shock, and may further lead to a partial separation of the placenta, contraction ring and haemorrhage. Should it thus become necessary to perform a manual removal of the placenta this operation may cause shock which may precipitate heart failure, as in one case 518/1946 of this series reported on page 118. The blood loss during this stage of labour has not been noted as differing from that in healthy patients.

It has not been the practice in this series of patients to place a sand-bag on the abdomen after delivery of the infant; or apply a tight pad and binder or induce a pneumoperitoneum. It is said that these procedures may prevent the sudden lowering of blood pressure which may follow the emptying of the uterus.

The more-or-less sudden decrease in intra-abdominal

pressure, coupled with the lowering of the diaphragm and abrupt change in the cardiac axis, the closure of the utero-placental circulation can be factors which may lead to distention and paralysis of the right side of the heart and cardiac failure. Venesection at this stage, when the patient is cyanosed, can give much relief to the embarrassed heart, and with cardiac stimulants and oxygen be a life saving measure.

The Place of Caesarean Section in Heart Disease.

The role of Caesarean section for the cardiac patient is a limited one. There was a time when the operation was performed more frequently and with results which greatly lowered the existing maternal mortality from heart disease, but this, states Jensen (1938), was due to the previous results being so bad, owing to the premature induction and interference which was then the treatment under the belief that the patient had to be rid of the pregnancy as soon as possible. Caesarean Section for the patient with heart disease is, however, yearly becoming fewer, and Greenhill (1946) considers that the operation is not justified where the sole indication is rheumatic heart disease.

In Fitzgerald's (1935) series of 126 women with severely damaged hearts no patient was delivered by Caesarean section because of the heart condition. No maternal death occurred in his series during pregnancy or labour, but there was one death 6 weeks post-partum from acute bacterial endocarditis.

Mendelson and Pardee (1942) report a series of two hundred cases of pregnancy complicated by rheumatic heart disease, in which only one patient was delivered by Caesarean Section and the remainder vaginally without a death in the entire series. In

another series of one hundred and sixty-two Group III and IV cases Mendelson (1944) reports a mortality of 12 per cent following abdominal delivery as compared with 0 per cent following vaginal delivery and all the abdominal operations were elective Caesarean sections without haemorrhage, while at the same time there was nothing in the histories of the abdominal group to make them appear as the most serious cases, yet there was no doubt about the severity of the vaginal group, which included thirty-four Group IV cases and seven with auricular fibrillation. While the data shew that Caesarean Section is performed less frequently in Groups III and IV patients there is no justification, however, Mendelson (1944) concludes, for the statement that Caesarean section should never be performed in the presence of heart disease.

TABLE XX.

Abdominal Delivery Because of Class III or Class IV Rheumatic Heart Disease. Mendelson (1944)

Year	Total Cases	Hysterotomy	Caesarean Section
1932	18	0	0
1933	101	0	7
1934	79	0	2
1935	106	0	1
1936	99	1	2
1937	116	0	4
1938	125	0	2
1939	78	0	2
1940	75	1	1
1941	95	1	0
1942	98	1	0
1943	99	1	0
	1089	5	21

McLennan (1933), too, says that the results of Caesarean section compare unfavourably with those of forceps delivery, and in his Group III patients there were twenty-two spontaneous deliveries with two deaths, and sixteen Caesarean sections with two deaths and, he adds, in commencing cardiac failure the shock of a surgical operation combined with a sudden emptying of a large abdominal viscus is considerable, so that the justification for the operation is open to question.

Caesarean sections were performed on twenty-one patients with Rheumatic heart disease in the present series, in the following groups: one in Group I, six

in Group II, ten in Group III, and four in Group IV. One death occurred, in a Group III patient, giving a maternal mortality rate of 4.8 per cent. In four of the above twenty-one patients, other complicating factors, causing dystocia, were present: there were no still-births in this series of Caesarean sections, but the neo-natal death rate was 4.8 per cent.

In the elective Caesarean sections, performed for disproportion on one hundred and eighty-six cases, over the same period of ten years at Queen Charlotte's Hospital, the maternal death rate was 1.2 per cent, the still-birth rate 0.6 per cent, and the neo-natal death rate 3.8 per cent. It is necessary, therefore, that the risk of Caesarean section 1 per se, should be borne in mind when advising this procedure.

In their review of cardiac fatalities, Hoffman and Jeffers (1942) found only one death from sepsis following vaginal delivery, as compared with six deaths from sepsis following Caesarean section.

There is, nevertheless, a very definite place for Caesarean section in the patient with heart disease. Where there is the possibility of prolonged or difficult labour Caesarean Section, with its known amount of strain, is to be preferred. Should there be a malpresentation, in which vaginal

delivery might be difficult or mean excessive trauma and shock, a Caesarean section is the treatment of choice. While a small foetus presenting by the breech may easily pass through the pelvis, especially if it is extended and the patient multigravid, the large breech free at the beginning of labour may well be associated with a delay and difficulty which the damaged heart does not well withstand and Caesarean section is advisable. In malpresentations such as a brow, shoulder or face it is also best to avoid the possible delay and the vaginal manipulations. The contracted pelvis which prevents engagement of the head before labour should indicate delivery by Caesarean section, and there is no place for trial of labour in the patient with heart disease.

Again there is the type of patient who, late in pregnancy, has reached an optimal pitch of improvement, or in whom improvement cannot be maintained and termination of the pregnancy is advisable; in such a case either rupture of the membranes or a Caesarean section can be performed. In the multigravid patient, with the cervix soft and already partially dilated, and perhaps the head well down, rupture of the membranes performed without an anaesthetic can be succeeded by a quick and easy labour. In other instances where for example, the cervix is long and

closed this procedure can be a serious risk, associated as it often is with prolonged labour, the patient anxious and distressed at the delay and her pulse rate increasing: and there is therefore the further possibility of adding to the risk of sepsis and shock by some form of interference. Lennie (1927) has shewn the disastrous results which follow induction of labour by bougies under a general anaesthetic. The maternal death rate in his series treated by this method was 44 per cent as compared with a mortality, in the same series, of 11 per cent following Caesarean section. In a series reported by Bramwell (1935), induction of labour performed in twenty-seven patients had a mortality rate of 14.8 per cent, while in the same series there were twelve Caesarean sections, with a mortality rate of 8.3 per cent. Both forms of treatment are, therefore, grave undertakings, with Caesarean section the lesser risk to the patient in most circumstances.

While, therefore, it may be said that vaginal delivery is best there is a definite place for Caesarean Section where some form of dystocia is anticipated, or in the rare instances, in the later weeks of pregnancy, where a premature delivery is

desirable and rupture of the membranes considered to carry the risk of a prolonged inertia of the uterus.

Sterilisation.

The question of sterilization is important, but plays no part in the decision to perform an abdominal section. Resection of an inch of each tube, burying the cut ends, is an easy addition to the operation of Caesarean section and should be fully discussed with the patient and her husband, should it be inadvisable for her to have more children. If she has two live children, or if there is a history of heart failure, signs of strain this additional operation should be added, if a Caesarean section is being done. Although other methods of preventing future pregnancies are not so certain as the resection of the tube, any abdominal section has a risk out of proportion to the usual methods of birth control. Sterilisation can also be performed at the end of the second week of the puerperium. Those who advocate this say that with the Fallopian tubes still easily accessible the operation can be conducted with minor disturbance under local anaesthesia. There can be little doubt however that the damaged heart needs its full time to recover and any additional interference adds to its burden. Hamilton and Thomson (1941) report that the only non-pregnant patient in this series who died

after tubal sterilisation was operated on twelve days after delivery: and they consider that her death, which was due to pulmonary embolism, might have been avoided had the operation been postponed until after the puerperium. Tubal sterilisation is therefore best done about six months after the vaginal delivery. Recently with the more accurate assessment, by means of daily recording of the patient's temperature, of ovulation time more reliance can be placed on contraceptive methods which have a high success with the more intelligent patient and advised also to the patient approaching the menopause.

The Anaesthesia in Caesarean Section.

The anaesthesia used for abdominal section in cardiac patients in this series has been gas and oxygen and light ether. Gas and oxygen well given does not cause the cyanosis and straining at times seen in inexpert hands and there is no doubt about the effect of ether as a cardiac stimulant. Greenhill (1946) and Stander (1935) prefer ether: and Hamilton (1944) states that he prefers Gas and oxygen or ether, considering ether an excellent anaesthetic for the cardiac patient and he adds local anaesthesia may be given at the same time, if indicated. MacLennan (1933) and Lewis (1933) state

that there is in their experience a greater risk of post operative bronchitis after ether. A local anaesthetic alone such as ½% Novocaine can be used, and is the safest anaesthetic for all Caesarean sections including those on patients with heart disease. It is true, however, that the patient must be mentally suited for this otherwise there may be a risk of precipitating heart failure. An epidural block and a spinal anaesthetic have the same disadvantages as a local anaesthesia, added to which the former is difficult and uncertain and the latter dangerous from its action in lowering the blood pressure no matter how this is counterbalanced by drugs. Continuous caudal anaesthesia usually required supplementing with a general anaesthetic: it is also uncertain in its results and attempts to use it may greatly disturb the patient. Vinethene and also cyclopropane have been used successfully; the latter given with Gas and Oxygen has been recently used with success. Reis and Frankenthal (1935) recommend Ethylene for heart patients.

In the choice of an anaesthetic it may therefore be said that if it is not advisable, owing to the patient being nervous, to use local anaesthesia, then the skill of the anaesthetist is of greater importance than the type of anaesthetic.

Modified from Jensen. "THE HEART IN PREGNANCY" P.263.

Source	Total Deliveries.	Total Caesarean Sections.	Percentage of Total Delivered by Caesarean Section.	Caesarean Section for Cardiac Indications.	Percentage of Total Number of Caesarean Sections for Cardiac Indications.	Fatal Caesarean Sections for Cardiac Indications.	Percentage of Caesarean Sections for Heart Disease which were fatal.	Rate of Cases, fatal from Caesarean Section for Heart Disease, per 100,000 obstetrical Admissions.
American	175,863	4,729	2.7	130	2.8	10	7.7	5.7
American	192,321	5,048	2.6	-	-	11	-	5.8
Maternal Mortality in New York	180,000	3,963	2.2	-	-	17	-	9.5
Continental	69,105	1,360	2.0	8	0.6	4	50.0	5.8
Edinburgh	-	436	-	41	9.3	4	9.8	-
Melbourne	40,183	486	1.2	9	1.8	1	11.1	2.5
Heynemann	68,947	1,004	1.45	9	0.9	3	33.3	4.4
Queen Charlotte's	29,713	434	1.46	23	5.3	1	4.3	3.4
Hoffstrom, (Sweden)	20,892	100	0.5	-	-	-	-	-

RESULTS IN THE PRESENT SERIES OF HEART DISEASE AND PREGNANCY

Of the total 225 cases presented there was some form of congenital heart disease in 13 and these are therefore considered separately.

The Rheumatic Heart Group.

In the remaining 212 cases, 194 or 93 per cent gave a definite history of rheumatic fever, but since in the remaining 18 cases acute rheumatism could not with certainty be excluded they have been considered together.

TABLE XXI.

Functional groupings and results.

	Group I	Group II	Group III	Group IV	Totals
No. of patients	86	72	37	17	212
Percentage in group	40.6	34.0	17.4	8.0	100.0
Spontaneous Delivery	73	50	16	2	141
Forceps Delivery	11	16	5	5	37
Patients delivered after 28th week, total percentage per vaginam	97.7	91.7	67.7	63.6	
Caesarean Section	1	6	10	4	21
Hysterotomy	1	0	6	5	12
Died undelivered	0	0	0	1*	1
Maternal deaths	0	1	3	3	7
Percentage maternal deaths	0	1.4	8.1	17.6	
Stillbirths	4	0	1	1	6
Neo-natal Deaths	0	2	1	1	4

*included in next column figure.

Analysing the figures in Table XXI, it will be seen that the majority of patients are in the more favourable groups I and II, and that the majority had a spontaneous delivery.

TABLE XXII.

Spontaneous Delivery Percentage:

GROUP	PRESENT SERIES	GILCHRIST (1931)	STANDER (1942)	McLENNAN (1933)
I	85.9	84.6	74.6	vast majority
II	69.4	77	59.9	69.5
III	51.6	55.6	31.6	44.5
IV	18.1	9	15.7	-

The forceps rate in the present series can be analysed as follows :-

Of the vaginal deliveries in each group, which ended in the use of forceps the rate in each group was as follows :-

TABLE XXIII.

Forceps Delivery Percentage:

Group I	13.1%
Group II	24.2%
Group III	23.3%
Group IV	71.4%

The Caesarean section rate has already been discussed. It is higher, I think, than need be, but the rate for the latter years is gradually becoming much lower.

There were twelve abdominal hysterotomies with one death (8.3 per cent), in a group IV patient. The hysterotomy performed in a group I case was on a patient with mitral stenosis and aortic regurgitation, who already had two healthy children.

TABLE XXIV.

Chart of type of Delivery in Heart Disease Complicating Pregnancy. Stander (1942)

Type of delivery	Group I		Group II		Group III		Group IV	
	No.	per cent	No.	per cent	No.	per cent	No.	per cent
Spontaneous	224	74.6	178	59.9	19	31.6	3	15.7
Operative (cardiac indication)	12	4.0	53	17.8	21	35.0	10	52.6
Operative (other indication)	50	16.6	30	10.1	1	1.6	0	0.0
Abortions (therapeutic and spontaneous)	14	4.6	36	12.1	19	31.6	6	31.6
Total	300	99.8	297	99.9	60	99.8	19	99.9

The still-birth rate in the series was 3 per cent, including four cases complicated by, 1 toxaemia: 1 vasa praevia: 1 accidental haemorrhage:

1 artificial rupture of the membranes. The corrected still-birth rate was, therefore, 2 out of 199 deliveries, or 1 per cent. The neo-natal death rate was 2.0 per cent. These figures compare well with the rates for all patients over a period of ten years, which were :- still-births 2.9 per cent, neo-natal deaths 1.6 per cent. The higher neo-natal death rate associated with the cardiac patient may, at least in part, have an explanation in the greater frequency of prematurity in these patients.

The average age of the patients in the series was 28.9 years; 131 were primigravidae, of whom 13, or 9.9%, were in group IV, and 81 were multigravidae, of whom only 4, or 4.9%, were in group IV. It is of interest to note, therefore, that there were more seriously affected hearts in primigravid patients and this is emphasised by the fact that all of the 7 patients in this series who died were primigravid; and in a group of cases reported by Bramwell (1935) there were 145 primigravidae with 13 deaths (9%), 155 multigravidae with only 4 deaths (2.6%). The average age of the patients, who shewed cardiac decompensation, was 29.8 years; and as regards the time of failure developing, 70% failed after the 24th week of pregnancy. These figures suggest that the condition of the heart is a more decisive factor

in prognosis than multiparity: and that it is in the latter half of pregnancy, as would be expected, that the greatest strain falls on the patient - a fact to be remembered where there is early decompensation and the question of abortion is being considered. The average duration of pregnancy for all patients preceding beyond the 28th week was 39 weeks, and in the group IV patients pregnancy lasted on an average 37.7 weeks; and in the pregnancies going beyond the twenty-eighth week and ending fatally, the average length of the pregnancy was 36.8 weeks, shewing the prematurity associated with cardiac decompensation.

Maternal Deaths.

The total maternal mortality in this rheumatic fever group of 212 patients was 7 deaths, or 3.29%.

A summary of the maternal deaths is given below:

1. 1946/518. Age. 25. Gravid 1: mitral presystolic, diastolic and aortic diastolic bruits. Patient reported as well, and was group II until 38th week of pregnancy, when she started to have acute breathlessness and bronchitis: admitted at 39th week as a group III case and with rest in bed improved so that two days later pulse 90, liver normal and lung bases clear, and

considered fit for normal delivery: improvement maintained with pulse 80 to 90, no evidence of cardiac embarrassment, no dyspnoea; slight oedema of vulva the only other abnormality. Labour started spontaneously a few days after expected date of delivery. There was a long first stage of labour, lasting 31 hours, 20 minutes, and although there was no dyspnoea or cardiac embarrassment the pains were so strong as to distress her: the pulse 96 strong, good volume and regular, and sedation with morphia and chloral. Towards the end of the first stage, however, the pulse had risen to 130 without any evidence of cyanosis or distress. Second stage, forceps delivery decided upon and was performed with ease: later the placenta did not separate, and although there was no bleeding, the pulse volume was noticed as not being good and her colour became poor. A manual removal of the placenta under 0.4 gr. of pentothal with continuous oxygen decided upon. This was just completed when the patient suddenly collapsed and died despite resuscitation methods.

2. 1946/2635. Age 25. Gravid 1. Mitral Stenosis. A well-compensated heart when first seen with pulse slow and regular, group II. Two months

later, at 22 weeks pregnancy, reported as being very breathless on the least exertion. Now a group III case, but with rest she improved, and was considered to be in group II four weeks later, there being only some oedema of the legs. At 37 weeks' pregnancy, there was again a relapse to group III and she was admitted to hospital. Her pulse was 100 but settled to 80 by the end of the week. The lungs cleared, but oedema of the legs was still present when the patient went into spontaneous labour at 38 weeks. The first stage of labour lasted 30 hours 13 minutes. The second stage lasted 1 hour 40 minutes, and she had some cyanosis at the end of this stage. Continuous oxygen was given: and spontaneous delivery was accomplished 10 minutes later after appearance of cyanosis, pulse 95, blood pressure 130/95. One hour after delivery patient became blue in colour, pulse regular and strong at 100. The following day, still cyanosed, and oxygen continued, pulse 88 to 112: given digitalis and mersalyl: on the second day of the puerperium, pulse 110, passed 67 oz. of urine, condition maintained. The third day, cyanosis increased, some dyspnoea, pulse rapid, regular, bronchitis

present and pneumonic patches in the lungs, temperature 100.4. Condition gradually deteriorated and she died on the 6th day of the puerperium. P.M. report: signs of congestive heart failure and chronic venous congestion of the lungs and acute dilatation of a previously well-compensated heart.

3. 1943/1002. Age 33. Gravid 1. Mitral stenosis. Patient in group II during the whole of pregnancy, and admitted at 38 weeks for pre-parturition rest: pulse good, normal rate and rhythm. Patient went into spontaneous labour as an L.O.P.: developed primary uterine inertia and after 126 hours in the first stage the cervix was still not fully dilated and there were signs of maternal and foetal distress: maternal pulse 120. Under general anaesthesia, forceps were applied inside the half-dilated cervix, which was gently slipped over the forceps, and a 9 lb. 1 oz. child delivered, which died a few hours later. Mother's condition became rapidly worse after delivery, pulse rising to 160 and the abdomen becoming extremely distended: a stomach tube was passed and 28 ozs. fluid was withdrawn, but despite further resuscitative efforts the patient died 12 hours later.

4. 1943/1157. Age 31. Gravid 1. Mitral stenosis. First seen at 14 weeks as a group IV case and admitted for rest. She had a sudden attack of breathlessness with cyanosis and coughing watery, frothy, blood-stained sputum, and the liver was 1 fingerbreadth below the costal margin. With continued rest and digitalis, condition greatly improved and pulse 80 to 90. Hysterotomy and sterilization performed at 18 weeks, but patient never fully recovered and had attacks of cyanosis and breathlessness and died 3 weeks after the operation.

5. 1938/1598. Age 31. Gravid 1. Mitral stenosis. First seen at 32 weeks in group IV, and admitted to hospital. A few hours after admission, had an attack of acute abdominal pain and dyspnoea, the patient stating that she often had similar attacks. The attack was relieved by oxygen. One hour later, developed sudden severe dyspnoea and cyanosis with moist sounds all over the chest, heart regular and slow. In spite of treatment died 25 minutes later, undelivered. P.M. shewed advanced mitral stenosis with pulmonary oedema and enlargement and passive congestion of liver.

6. 1938/2143. Age 23. Gravid 1. Mitral stenosis. Condition one of moderate decompensation, a group III case when first seen. Four weeks' rest in bed at 30 weeks completely relieved her symptoms. Classical Caesarean section was performed at term with delivery of living child. Post-operative condition satisfactory until 3rd day, when dyspnoea and fibrillation commenced. This was, however, controlled by digitalis, but 17 days after the operation the patient had a sudden attack of dyspnoea and cyanosis with moist sounds all over chest and died shortly afterwards.

7. 1946/1632. Age 25. Gravid 1. Mitral stenosis. Group II when first seen at 8 weeks, with cardiologist's report of no evidence of failure or other abnormality except anaemia and the mitral stenosis. Admitted to hospital at 36 weeks, with history of 2 recent attacks of heart failure, and now showing marked oedema of the ankles, colour pale, dyspnoeic at rest, with crepitations at both bases, pulse 120, in group IV. Put on digitalis. Four days later, went into spontaneous labour, cyanosed but not breathless, pulse regular and volume good, and

with morphia had an easy labour, first stage 19½ hours, second stage ½ hour. Spontaneous delivery, baby 5 lb. 5½ oz. Pulse continued with good volume and regular, but put on digoxin which brought the pulse down from 105 to 78, per min., by the third day of the puerperium. The afternoon of the third day, however, patient had a sudden attack of dyspnoea and cyanosis and started coughing up blood-stained sputum, pulse 100 and fibrillating. The dyspnoea increased and despite further medical measures, the patient became worse with gross bubbling all over chest and she died the evening of the third day of the puerperium.

There are several factors of interest and importance in the records of the seven fatalities of this series. Four of the patients were in functional group II when first seen: one died in this group, whilst the others became worse as pregnancy advanced, two changing to group III, and one to group IV. Changes from group II to group III are not uncommon, especially about the twenty-eighth week of pregnancy, but in the large majority of cases, a week's good rest in bed restores them to their original group:

it is, however, disconcerting to find, as already stated, that some of these group II patients become worse and develop cardiac failure, which may have a fatal ending, thus supporting the dictum of Hamilton and Thompson (1941) that, "any cardiac may fail at any time." Additional considerations may, therefore, be required in the assessment of some patients, which would serve to anticipate this deterioration with the strain of pregnancy and labour, and it might be advisable for each heart case to be treated as an individual problem, using, besides the functional classification, all those factors discussed in the assessment of heart cases. It is noted, too, that even the group II patient does not stand interference well, and that any form of accouchement forcé, however carefully performed, may have a fatal termination: in one group II case of the present series there is such a fatality, when, after an inertia of a hundred and twenty-six hours, there was maternal and foetal distress and the cervix was manually dilated and delivery completed by forceps. In another patient, in group III, the shock of a manual removal of the placenta was immediately followed by collapse and death.

Hunt (1926) correlated the tendency to premature labour with the presence of congestive failure, and,

from observation of the present series, this has been noted to occur in the group IV cases more than in the other groups. This is an unfortunate feature, since it often does not give the patient time to benefit from her medical treatment before meeting the added strain of labour. The average length of pregnancy in the fatal cases of this series, who went beyond the twenty-eighth week, was 36.8 weeks, and among the rest of the group IV patients the average length was 37.7 weeks.

The risk of a prolonged labour is an obvious one, and, in three of the fatalities described, the first stage of labour lasted for an average of twenty-seven hours, and in one case for a hundred and twenty-six hours. It was noted, too, that three out of four who went into labour and who died had a pulse of over 112 during the first stage of labour; and one recalls again Mendelson and Pardee's (1942) assertion that, in all their cases of inter-partum or post-partum heart failure, the pulse was elevated to 110 or over, either alone, or with an increase in respiration of 24 a minute, during the first stage of labour; and that they found no serious heart failure, irrespective of functional classification, provided the pulse and respiration

rate remained below these critical levels throughout the first stage of labour.

Time of Death.

The time of death in the present series varied. Two patients died soon after premature labour, two died in the puerperium on the third and sixth day after spontaneous delivery at thirty-six and thirty-seven weeks respectively; one died undelivered at the thirty-second week, one three weeks after hysterotomy at the eighteenth week of pregnancy, and one the third day after a Caesarean section. Jensen, (1938) commenting on the time of death in heart cases, said that the commonest time was between the fifth and twenty-eighth day after delivery, and gives the following figures from a series of cases:-

During Pregnancy,	68.
In labour,	46.
In first twenty-four hours,	75.
First to fourth days,	71.
Fifth to twenty-eighth day,	131.

Hoffman and Jeffers, in their review of sixtyone fatalities, found that 75 per cent of deaths in group IV patients occurred in twenty-four hours after labour and were due to decompensation.

Sheehan and Sutherland (1940) give a number of tables illustrating the time of death in patients with heart disease as found from their investigation.

TABLE XXV.

Relation of Time of Cardiac death to duration of Pregnancy - Sheehan and Sutherland 1940.

Time of death	Duration of pregnancy in weeks. To 29	30 - 35	36 and over
During pregnancy	2	4	3
During labour or within 4 hours after	1	3	6
During puerperium	3	6	14

TABLE XXVI.

Time of death in relation to Delivery (compared with non-cardiac patients) - Sheehan and Sutherland 1940.

	Time of Death						
	During Pregnancy	During Labour	After delivery. 0-4 hrs.	5-24 hrs.	1-4 days	5-10 days	over 10 days
Not decompensated							
Non-cardiac death	3	1	4	5	3	6	6
Cardiac death	2	0	1	1	3	2	1
Slight decompensation							
Non-cardiac death	0	1	0	1	0	2	0
Cardiac death	5	2	3	0	0	0	4
Severe decompensation							
Cardiac death	2	1	2	2	1	2	4
Control obstetric patients without any valve lesions	71	56	155	144	70	143	69

It might in theory be expected state Sheehan and Sutherland (1941) that the physical task of labour and delivery would cause severe decompensation of the heart and that the patient would die either during labour or within a few hours afterwards. In practise this is not usually the case. From the above table it will be seen that deaths during labour or the first day after delivery are relatively less frequent among the patients with heart disease than among the controls.

TABLE XXVII.

Cardiac deaths in relation to delivery.
Sheehan and Sutherland (1940)

	Time of Death						
	During Pregnancy	During Labour	After delivery.				
Type of death			0-4 hrs.	5-24 hrs.	1-4 days	5-10 days	over 10 days
Acute heart failure	4	1	3	2	2	1	1
Anaesthesia	0	2	2	0	0	0	1
Terminal decompensation	3	0	0	1	0	1	2
Progressive decompensation	1	0	1	0	1	1	2
Cardiac lung complications	1	0	0	0	0	1	2
Cerebrol Embolism	0	0	0	0	1	0	1

From the above detailed grouping of the cardiac deaths Sheehan and Sutherland state that most of the fatalities during labour or the first day after

delivery are due to acute heart failure, or occur under anaesthesia: the patients with progressive or terminal decompensation do not tend to die at this time.

The actual time of onset of acute heart failure in relation to delivery is given and it is noted that the tendency to acute failure during labour is not related to the degree of decompensation of the heart.

TABLE XXVIII.

Relation of Type of Death to delivery
Sheehan and Sutherland (1940).

	Time of Death				
	During Pregnancy.	During Labour	After delivery		
			First 24 hours	1-4 days	5-28 days
Jensen (1938)					
Cardiac deaths in 10,000 obstetric patients with chronic valve lesions	68	40	75	71	131
Sheehan & Sutherland (1940.)					
Deaths in 10,000 obstetric patients with chronic valve lesions.					
Cardiac death	92	38	77	46	131
Non-cardiac deaths	23	15	77	23	108
Sheehan & Sutherland (1940).					
Non-cardiac deaths in 10,000 control obstetric patients with normal valves	9	7	38	9	27

During pregnancy, labour, or after the first day of the puerperium the cardiac deaths are from 5 to 9 times as many as the controls: and during the first day of the puerperium such deaths are absolutely the most frequent. The non-cardiac deaths are twice as common as in the controls during pregnancy labour or the early puerperium, illustrating the bad influence of even a symptomless valve lesion on the ordinary obstetric complications.

Cause of Death.

TABLE XXIX.

Manner of Deaths in patients with Heart Disease.
Hoffman and Jeffers (1942).

Cardiac decompensation, including pulmonary oedema	.. 64 per cent
Sudden death from embolism	.. 16 per cent
Puerperal sepsis and terminal cardiac failure	.. 15 per cent
The remaining three cases (subacute bacterial endocarditis, acute antepartum endocarditis, and one uncertain).	.. 5 per cent

The cause of death in heart disease and pregnancy, Jensen (1938) states, is congestive failure in at least 70 per cent of the cases, and a large number of the remainder die from pulmonary causes, including pulmonary oedema, and occasionally from cardiac exhaustion, sudden collapse and embolus.

Jensen says, too, that the incidence of death from sepsis is also above what should be expected (fatal sub-acute bacterial endocarditis has been seen in the puerperium, although not in this series). Of the fatalities recorded in this series, 71.4 per cent were due to congestive failure; and sudden collapse during labour accounted for two cases.

Sheehan and Sutherland (1940) state that the valve lesions which lead to acute heart failure are not significantly different from those in other kinds of death: and the problem therefore remains, they contend, of why one patient should develop congestive failure while another with an apparently identical valvular lesion does not. The valvular lesion present for a long time can be only one of the factors, they consider, and asking whether the death is the result of an acute myocarditis or an acute endocarditis go on to give it as their opinion that nearly all of their obstetric patients, whose hearts had been decompensated or who died cardiac deaths had recurrent endocarditis (i.e. fresh vegetations on the valves).

TABLE XXX.

Incidence of Recurrent Endocarditis on Chronic Valve Lesions.

Obstetric patients.

Type of Death	Age Groups 16-25	26-35	36-45	Recurrence per cent
Not decompensated. Died from obstetric accidents	1/1	1/2	0/1	50
Not decompensated. Died from various diseases	2/2	9/12	4/9	65
Any decompensation or cardiac death	9/9	18/20	9/11	90
recurrence per cent	100	82	62	79

Non-pregnant women.

Type of Death	Age Groups 16-25	26-35	36-45	Recurrence per cent
Not decompensated. Died from obstetric accidents.	0/3	0/2	0/2	0
Not decompensated. Died from various diseases.	2/5	4/10	6/23	32
Any decompensation or cardiac death	27/32	23/34	16/43	61
	72	59	32	51

(In the above table the upper figures in each fraction shows the number of patients with recurrent endocarditis and the lower figure shows the total number with chronic lesions.)

This recurrence of fresh vegetations on the heart valves as shown at autopsy is presumably an indication of some active lesion in the heart valve, but Aschoff nodules were not found in any of the cases examined microscopically by Sheehan and Sutherland (1940). Also since, they state, the recurrence is usually before delivery it is not due to puerperal infection. The importance of recurrent endocarditis is that it is thought to be associated with a myocarditis, which leads to congestive failure. Sheehan and Sutherland (1940) state that the fact that the vegetations may be found in patients whose hearts are not decompensated proves that, at any rate in their early stages, they do not necessarily cause congestive failure - but their almost invariable occurrence in decompensated patients points to some definite association. It is not clear however whether the recurrent endocarditis causes or is caused by the decompensation, or whether both are the results of a third factor: and there is not any satisfactory evidence that it is associated with a pancarditis. The immediate significance of the recurrent endocarditis is not yet elucidated. And Jensen (1938) could not find any convincing evidence that pregnancy shortens the life of patients with chronic valvular disease - and such a shortening might be expected if the recurrent endocarditis so commonly associated

with pregnancy led to subsequent further scarring of heart valves.

The clinical diagnosis of recurrent endocarditis Sheehan and Sutherland (1940) state cannot be based on variations of pulse and temperature though it can reasonably be inferred from the age of the patient and the cardiac function.

While such evidence is produced by Sheehan and Sutherland (1940) both Coombs (1924) and French and Hicks (1906) denied any predisposition to recurrent endocarditis during pregnancy.

Bramwell and Jones (1944) report two cases of acute pulmonary oedema leading to death, about half way through pregnancy and, discussing the mechanism of production of this complication, attribute it to acute left auricular failure. They state that of the two main causative hypotheses - toxic or mechanical - there now appears to be little satisfactory evidence to support the toxic theory, and add that, even in cases of renal disease with hypertension, pulmonary oedema is usually ascribed to left ventricular failure: and in left ventricular failure there is no clear dividing line between pulmonary congestion and acute pulmonary oedema. Similarly, they say, in mitral stenosis, pulmonary congestion and its rarer sequel pulmonary oedema differ only in degree and are produced by the same mechanism of which there are

three stages - (1) stage of faultless compensation, (II) stage of failure of left side of heart, (III) stage of failure of right side of heart, with systemic venous engorgement. Both their patients died in mid-pregnancy and it seems probable, they conclude, that the increasing blood volume and rising heart output at this stage of pregnancy were factors in precipitating heart failure.

THE PUERPERIUM.

It is unsafe to predict the outcome of pregnancy until the puerperium is safely passed. The heart, it would seem, able to withstand the strain of pregnancy and labour, may suddenly begin to fail in the puerperium and in fact as shewn previously (Pages 127 to 131) this is the commonest time of death. The appearance of the patient and the physical signs are typically those of congestive heart failure as in case 2635/46 reported in page 119. The patient becomes breathless and cyanosed, her lungs full of moist sounds and she coughs up an excess of sputum often blood-stained and there is a spreading oedema and an anuria and a heart which is fibrillating: sometimes, too, there is evidence of thrombi from the heart valves reaching the brain. It is of great importance therefore to have evidence from Mendelson and Pardee (1942) that no patient of theirs developed heart failure in the puerperium where the pulse during the first stage of labour was below 110 per minute and if it were so increased failure did not follow in the puerperium if immediate digitalisation was instituted.

The danger of puerperal infection is greater where there is a damaged heart valve to provide a nidus for organisms gaining access to the circulation.

Subacute bacterial endocarditis may thus occur and prove fatal. In order to avoid this catastrophy Mendelson (1944) recommends the routine use of sulpha drugs in the puerperium for all patients with valvular disease of the heart. No case of subacute bacterial endocarditis was encountered in the present series and it is doubtful if this extreme in prophylaxis would be justified or even effective. Penicillin has a more curative action, but even it does not succeed with the less penicillin sensitive strains of the streptococcus viridans.

A wise precaution however is to place all patients with heart disease on a four-hourly pulse and temperature chart during the puerperium so that any deviation from the normal can instantly be noted. From a study of the charts in the above series it is seen that the highest period in the pulse rate is during the 4th and 5th day. There is no corresponding temperature rise so that this is not likely to be due to congestion of the breasts and whatever significance can be attached to this peak in pulse rate it is advisable to ensure the maximum rest and sleep for the patient during the early days of the puerperium. Sedatives are therefore freely given and the patient does not indulge in the routine of physiotherapeutic exercises encouraged in other patients.

The routine in the present series ensures a full fourteen days rest in bed after delivery, and the patient is then allowed out of bed and slowly to increase the up-period, being fit for discharge, if her progress has been satisfactory, by the end of the third week.

The majority of patients are able to breast feed their babies, but for the Group III and Group IV patient it is a hazardous task and bottle feeding should be established.

PROGNOSIS IN RHEUMATIC HEART DISEASE.

Immediate Prognosis.

The large majority of women with heart disease, in whom physical limitations are absent or only slight, will go through pregnancy without undue risk. It is equally true, that the strain of pregnancy may be fraught with extreme danger and may even prove fatal to some hitherto enjoying an active life.

Carr and Hamilton (1933) advocate that weekly visits and examinations of the patient should be the rule whether apparently needed or not, in order to lessen the risk of heart failure which they insist may occur even between the weekly visits. All observers agree that it is the development and extension of antenatal care, coinciding with the great drop in maternal mortality from heart disease, which has proved itself of cardinal importance. Frequent examination of the patient can do much, for instance, to prevent the development of heart failure. Each visit by the patient should entail the following:-

1. Enquiry into the daily routine of the patient, ensuring that she is working well within the limits of her cardiac condition and avoiding tiredness and breathlessness.
2. Ascertaining that there is no new emotional upset, prolonged mental strain, or financial worry.

3. Watching for other causes of precipitating heart failure such as anaemia, upper respiratory tract infection, or other inter-current disease.
4. Instruction should be given to the patient to report any new illness, such as colds, and she should immediately stay in bed at their appearance until she is better.
5. Haemoptysis or cough may mean a failing heart and the attention of the examiner must be drawn to them by the patient.
6. The earliest sign of failure such as persistent rales at the lung bases can be diagnosed at this examination before the condition has been present for any length of time.
7. Hospital conditions until delivery should be the rule in all cases of heart failure, where the pregnancy is continuing. The general appearance of the patient, her pulse rate and her colour, frequently suggest that pregnancy is becoming a dangerous burden to her before actual failure appears.

The history of previous decompensation is of great value in that, as Gilchrist (1931) said, this complication must be watched for in succeeding pregnancies. Gorenberg and McGleary (1941) give

the following table on this aspect.

TABLE XXXI.

PROGNOSTIC VALUE OF HISTORY OF DECOMPENSATION.

Gorenberg and McGleary (1941)

Cardiac failure previously but not in this pregnancy.	11
Cardiac failure previously and again in this pregnancy.	33
Cardiac failure for the first time in this pregnancy.	44
Previous cardiac failures	44
Previous cardiac failures and again in this pregnancy	33-75%

Treatment of the patient, when the first signs of failure show themselves, is almost always successful, whereas if the patient is not seen until the heart failure is well advanced the danger of a fatal issue is greatly increased. Bramwell (1935) stated that in his series of cases, of 13 cases admitted directly to the wards as emergencies, there were 9 deaths, whereas out of 278 patients who had been attending the antenatal clinic there were only 8 deaths. Bramwell (1935) further says that in heart failure, if the heart's liabilities can be reduced

by an increased restriction of the patient's activities, the outlook is favourable; but when heart failure persists in spite of confinement to bed, the outlook is much more serious. In his series of 300 cases, 21 patients had signs of congestive heart failure when they first came under observation, and of these 6 died prior to or soon after confinement, 5 were known to have died in the next four years, and 4 others were detrimentally affected. These figures show, states Bramwell, the extreme gravity from the prognostic point of view of established heart failure in pregnant women.

The question of therapeutic abortion has been fully discussed on page 83, and it undoubtedly plays an important part in the reduction of maternal mortality. Auricular fibrillation, p.63, is a complication of grave prognostic significance, but both Gilchrist (1931) and Bramwell (1935) assert that it is surprising how well some of these patients do provided they have adequate treatment commencing at an early stage of pregnancy. Haemoptysis is always alarming to the patient and is generally considered to be of grave significance in prognosis. Valvular lesions, as previously mentioned, (Page 61) do not afford any help in prognosis. Mitral stenosis was looked upon as the more serious valvular disease since, as in this series too, this was a valve commonly found affected at death; it is, however,

the seat of the heart lesion in the large majority of patients with rheumatic carditis.

Enlargement of the heart is taken as a rough index of the severity of the heart condition, the bigger the heart the smaller its reserve; it is, however, only one of the cardiac factors in prognosis and its difficulty in assessment, as described in page 60, limits its usefulness.

The mortality rate in the more favourable groups I and II in this series was 0.62 per cent, while in the presence of definite cardiac decompensation of group III and IV the rate was 11.1 per cent. The effect of age and parity on decompensation of the heart are fully discussed in pages 68 and 73; age being considered by most observers to be associated with a decrease in the heart's resiliency and response to strain, and parity carrying at least an increase in the immediate risks to the patient with each succeeding pregnancy. The emphasis on obtaining if possible a spontaneous vaginal delivery has been another step forward in the management of the cardiac patient. It is certainly not always possible to predict whether a patient with heart disease will have an easy labour or not; and there is an essential place, therefore, for the Caesarean Section operation in lowering the mortality rate

in such cases who are believed to be bad prospects for a long labour or where dystocia is anticipated.

The foetal prognosis shows no additional loss from spontaneous abortion in patients with heart disease and the total stillbirth rate is not raised. The stillbirth rate in the combined groups I and II of this series was 2.5 per cent and was more favourable than in the combined groups III and IV in which it was 4.76 per cent; all the stillbirths being, as previously mentioned, associated with an additional complicating factor. The neonatal death rate in groups I and II was 0.63 per cent and in groups III and IV 4.76 per cent. The higher neonatal death rate in the latter groups is associated with the premature labours and will doubtless be improved upon with better facilities for treating the premature baby.

Teel (1935) also says that the greatest foetal loss occurs in that small group of patients with severe heart lesions most of whom, had they sought competent medical attention, could have been advised that their heart reserve was too low to withstand the strain of pregnancy: and he considers that, in the larger group of patients, with the well compensated heart, the foetal mortality does not materially differ from that of normal subjects.

Distant Prognosis.

Reviews of this nature have been undertaken by a number of authors such as, French and Hicks (190), Tunis (1933), Gilchrist and Murray-Lyon (1933), Herrick (1933), Lamb (1937), Boyer and Nadas (1944).

A study of this kind is known, however, to be full of many difficulties since there are numerous factors other than pregnancy which can influence the health of patients with rheumatic heart disease, and because a follow-up of all cases from the commencement of the disease is not possible the study must necessarily be incomplete. Difficulties due to a moving population and a failure of the patient to report have also to be contended with: and comparative groups such as males and nulliparae have certain imponderable features peculiar to themselves.

Nevertheless it is possible to obtain a fairly accurate clinical impression from the assessment of a patient's condition during pregnancy and at a later follow-up and to formulate some views on the subject. Looked upon as a separate entity a study of this nature was made in the present series and of those asked to report 37 did so and 23 answered a questionnaire from which a knowledge of their grouping, according to the American Heart Association, could be obtained.

Questionnaire.

Can you do your own housework.

Do you rest at frequent intervals.

Do you climb stairs.

1. without stopping
2. resting at the top
3. without stopping or resting.

Can you walk back from shopping without a rest.

Is there any swelling of the ankles.

Have you a persistent cough.

Do you spend extra time in bed.

Have you an extra pillow at night.

Are you confined to bed.

Have you had further pregnancies.

Were you well during them.

PRESENT SERIES.

A REVIEW AND FOLLOW-UP OF 60 PATIENTS WITH HEART DISEASE WHO ATTENDED QUEEN CHARLOTTE'S MATERNITY HOSPITAL - FEBRUARY 1948.

The hospital number of each patient is given with the year of confinement, and followed by a brief resume of the history and assessment.

No. 1548/42. Age: 24 years. Gravid.2

Heart lesion: Mitral and aortic stenosis.

Previous History:

Rheumatic fever at the age of 8 years.

Obstetrical History:

Normal delivery one year ago 6¾ lb. baby. In good health throughout pregnancy.

Course of Pregnancy:

Patient well with heart well compensated when first seen at 20 weeks pregnancy but at the 28th week of pregnancy became decompensated with dyspnoea, rapid pulse and haemoptysis: was admitted to hospital for 4 weeks with improvement and was discharged from hospital. Two weeks after discharge condition broke down again and was therefore readmitted. No cyanosis or oedema but pulse 114 and breathlessness. There was quick and satisfactory improvement with rest, and a cough which was troublesome settled and pulse 90. X-ray of chest showed a thickening in the region of the hilum of the lungs: enlargement of the right ventricle and marked dilatation of right side of heart.

Delivery:

Caesarean Section and sterilisation August 1942 under

gas oxygen and ether at 37th week of pregnancy. Child 6 lbs. 8 oz.

Puerperium:

Convalescence satisfactory, pulse 80 - 90, discharged at the end of 3 weeks condition fairly good.

Postnatal Clinic:

No breathlessness nor cyanosis, occasional swelling of ankles, pulse 86 normal rhythm.

Follow up:

Patient's general condition fair. Unable to work without stopping at frequent intervals and resting half way up a flight of stairs: persistent cough present: requires to spend extra time in bed. Pulse 80 slow fibrillation controlled by digitalis which patient has been taking for two years: haemoptysis repeated small attacks still, congestion present at left base. Liver normal in size, slight cyanosis. X-ray of heart showed a well marked cardiac enlargement with T.D., 155 mm.: pulm. conus +, L.A.+: generalised pulm. vascular congestion of slight degree.

Comment.

Patient showed a change of group during her pregnancy developing decompensation about the 25th week. Rest in bed was shown to produce marked improvement and to bring patient back into her original group.

Cardiac breakdown occurred shortly after the patient was discharged suggesting, therefore, that she should have stayed in hospital until delivered. Condition at Follow up Examination showed deterioration.

No. 533/43. Age: 23 years. Gravid. 2

Heart lesion: Mitral stenosis.

Previous History:

Rheumatic fever in childhood.

Obstetrical History:

Normal delivery of 5 lb. child in 1941. Pregnancy normal.

Course of Pregnancy:

Slightly breathless at 16 weeks and advised to rest. Heart x-ray slight cardiac enlargement. With rest at home patient remained in group II with no sign of decompensation at any period. There was slight anaemia Hb. 65%. Patient admitted for rest at 28th week and one week before term. Pulse varied from 80 to 90. No oedema and no cyanosis.

Delivery:

Normal delivery at 40 weeks, labour 3 hours 50 mins. child 5 lbs. 13 oz.

Puerperium:

Normal. Pulse 70 - 90.

Postnatal Clinic:

General condition good but slight cyanosis.

Pulse 85. No undue breathlessness.

Follow up:

Patient had 2 further pregnancies, one in which she went to term and had breathlessness during it; the second pregnancy ended in hysterotomy and sterilisation at 18 weeks for early decompensation of the heart. Now resting at frequent intervals, can do moderate work without stopping, has a persistent cough and requires extra pillow at night. Is unable to walk back from shopping without a rest.

Comment.

General condition has deteriorated.

No. 1001/43. Age: 27 years. Gravid. 1

Heart lesion: Mitral stenosis.

Previous History:

Chorea at the age of 10 years.

Course of Pregnancy:

Condition fairly good until the 22nd week of pregnancy when there was slight oedema of the ankles and shortness of breath with effort: slight haemoptysis at 26 weeks: x-ray of chest, lungs N.A.D. Heart enlarged with prominent pulm. conus. Condition improved with rest at home but developed a mild toxaemia of pregnancy and was admitted one week before term for termination of the pregnancy for toxaemia. A.R.M. performed.

Delivery:

A.R.M. for toxaemia at 39th week: patient distressed with the pains but spontaneous delivery after 12 hours 5 minutes labour: child 6 lbs. 9 oz.

Puerperium:

Normal: pulse 70 - 80,

Postnatal Clinic:

General condition good. Toxaemia clear., no undue breathlessness.

Follow up:

Able to do own housework resting only at end of effort. There is, however, a persistent cough and slight swelling of the ankles and patient needs an extra pillow at night.

Comment.

General condition now only fair: pregnancy had shown a complication of both toxaemia and haemoptysis.

No. 1212/43. Age: 32 years. Gravid 2.

Heart lesion: Mitral stenosis.

Previous History:

No definite history of rheumatism, has never been breathless.

Obstetrical History:

Normal delivery in 1940 of an 8 lb. 6 oz. baby. Pregnancy normal.

Course of Pregnancy:

General condition good and no breathlessness . At 37th week of pregnancy developed mild toxaemia, twin pregnancy found. Admitted to hospital. Albumin and slight oedema disappeared with rest.

Delivery:

Went into spontaneous labour at 39th week, labour 7 hours 20 minutes, twins 6 lbs. 15 oz. and 6 lbs. 13 oz. healthy.

Puerperium:

Normal, pulse between 70 and 80.

Postnatal Clinic:

No sign of toxaemia, no signs of decompensation.

Follow up:

Patient has no undue breathlessness with effort: unable to do a full day's work without stopping. There is no cough or swelling of the ankles.

Comment.

A group I patient who developed toxaemia during pregnancy: made complete recovery from the toxaemia and still in group I as regards heart condition.

No.1509/43. Age: 39 years. Gravid. 5

Heart lesion: Mitral stenosis and incompetence.

Previous History:

Chorea in childhood.

Obstetrical History:

Four spontaneous deliveries all premature between 7 - 8 month with weight of children all about 4 lbs. Had a heart attack with the third pregnancy in 1935.

Course of Pregnancy:

During the 5th pregnancy patient was in group II during the early months but became slightly breathless as pregnancy advanced. By the 37th week of pregnancy had occasional attacks of gallop rhythm and was breathless on slight exertion. She was admitted to hospital and examination showed moist

sounds at the lung bases, pulse varying between 95 and 110, liver two fingers below the costal margin. With rest the chest cleared and general condition greatly improved so that there was complete compensation again.

Delivery:

Spontaneous at term, child 5 lbs. 2¼ oz., labour 50 minutes.

Puerperium:

Remained 26 days in hospital, the pulse varying between 90 and 100.

Follow up:

Unable to do work without frequent rests: has to stop several times while climbing stairs and cannot walk back from shops without a rest. There is a persistent cough and patient needs an extra pillow at night. There is no swelling of the ankles. Pulse 90, auricular fibrillation, slight cyanosis present, liver normal in size. X-ray generalised cardiac enlargement. Giant left auricle, T.D., 186 mm.

Comment.

Patient gave a history of cardiac breakdown with 3rd pregnancy and with 5th. Present condition shows deterioration. The rest in bed with the last pregnancy produced only immediate improvement.

Patient stated that she had to work hard attending to her children.

No.1512/43. Age: 25 years. Gravid. 1

Heart lesion: Mitral stenosis.

Previous History:

Scarlet fever in childhood. Chorea at 8 years. Rheumatic fever at 17, 21 and 23 years with periods of 2 months in bed on each occasion.

Course of Pregnancy:

Well until the 18th week of pregnancy when slight breathlessness and slight oedema of the ankles appeared. Condition remained the same until the 36th week when breathlessness increased so that patient could not lie down in bed, pulse 112. Patient also complained of pains in the knees and sweating. Temperature was normal. Condition greatly improved with rest in bed.

Delivery:

A.R.M. at 39 weeks for small gynaecoid pelvis.
Labour 29 hours 50 minutes, condition good.
Child 7 lbs. 5 oz.

Puerperium:

Pulse 80 to 90, puerperium normal. General condition good on discharge. Sinus arrhythmia.

Postnatal Clinic:

Some dyspnoea only on excessive exertion.

Follow up:

No disability as regards daily work, does a full days work without breathlessness.

Comment.

Patient showed evidence of changing her group during the pregnancy and showed the possible recrudescence of the acute rheumatism but the sedimentation rate was 22 and the condition subsided without treatment.

No.1813/43. Age: 25 years. Gravid.1.

Heart lesion: Mitral stenosis and incompetence.

Previous History:

Rheumatic fever at 15 years.

Course of Pregnancy:

Patient was breathless only at end of effort and with rest no signs of failure developed and she remained in group II. There was just a brief period at her 26th week when she said she was a little more breathless than usual.

Delivery:

Spontaneous labour at the 37th week, extended breech delivery, labour 27 hours 45 minutes, no undue distress, child 5 lbs. 4¼ oz.

Puerperium:

Pulse between 70 and 90, discharged in good condition.

Follow up:

General condition good, pulse 65, lungs clear, no oedema, able to do a full days work without stopping, had one miscarriage at the 3rd month one year ago.

Comment.

Heart appears well compensated.

No.1790/43. Age: 21 years. Gravid. 1

Heart lesion: Mitral stenosis and incompetence.

Previous History:

A definite history of rheumatic fever at 10 years.

Course of Pregnancy:

Patient went straight through her pregnancy as a group 1 case. Did not complain of breathlessness and there was no cough or cyanosis.

Delivery:

At 40 weeks spontaneous, labour 17 hours, child 8 lbs. 1¼ oz.

Puerperium.

Normal. Pulse 80 - 90.

Postnatal Clinic:

General condition good. No breathlessness.

Follow up:

History of further pregnancy with delivery of full term child 4½ months ago. Was slightly breathless during the pregnancy. Now has to rest at the end of effort and has to spend extra time in bed. Pulse 96. X-ray of heart - no cardiac enlargement, no left ventricular hypertrophy. T.D. 122 mm.

Comment.

Patient appeared to have gone through her first pregnancy with ease, was slightly breathless with the 2nd pregnancy and has not yet returned to her pre-pregnancy condition.

No. 197/44. Age: 32 years. Gravid. 1

Heart lesion: Mitral stenosis and incompetence.

Previous History:

Rheumatic fever 1935.

Course of Pregnancy:

Heart fully compensated throughout pregnancy. No undue breathlessness shown.

Delivery:

40 weeks spontaneous, labour 39 hours 30 minutes, child 7 lbs. 10 oz.

Puerperium:

Pulse 80 - 85 normal.

Follow up:

No breathlessness with physical effort but patient states she does not overdo things.

Comment:

A group I case, no signs of decompensation.

No. 1111/44. Age: 20 years. Gravid. 1

Heart lesion: Mitral stenosis and aortic incompetence.

Previous History:

Chorea at 6 years and again at 14 years.

Course of Pregnancy:

Patient remained in group I throughout her pregnancy with no sign of decompensation.

Delivery:

Spontaneous at 40 weeks, labour 9 hours 10 minutes, child 7 lbs. 8 oz.

Puerperium:

Normal, pulse 70 - 80.

Postnatal Clinic:

No breathlessness, condition good.

Follow up:

Patient has to rest at end of effort but able to do a full day's work: pulse 80, no cyanosis, no pulm. congestion. X-ray - slight left ventricular hypertrophy, left auricle not obviously enlarged.

T.D. 133 mm.

Comment:

There would appear to be no physical deterioration in the patient's condition, withstood the strain of pregnancy well.

No.1173/44. Age: 30 years. Gravid. 1

Heart lesion: Mitral stenosis

Previous History:

Rheumatic fever at 16 years.

Course of Pregnancy:

Breathlessness only on undue effort, no signs of decompensation during the pregnancy.

Delivery:

Spontaneous labour at 40 weeks, length 2 hours 40 minutes, child 5 lbs. 8 oz.

Puerperium:

Normal, pulse 70 - 80.

Postnatal Clinic:

Condition satisfactory, no undue breathlessness.

Follow up:

Had a further pregnancy one year ago and stated that she was fairly well during it. Now able to do work but rests at the end of effort. Pulse 80. No evidence ofcyanosis, lungs clear. X-ray - no cardiac enlargement, L.A. not apparently enlarged.

Comment:

Patient has withstood the strain of two pregnancies well.

No. 1245/44. Age: 27 years. Gravid.1.

Heart lesion: Mitral stenosis.

Previous History:

No definite history of rheumatic fever.

Course of Pregnancy:

Patient remained in group II until the 28th week of pregnancy when she was complaining of more breathlessness. Pulse 90 - 110. Admitted for rest. No oedema and lungs clear. Heart considerably enlarged to left. After two weeks rest in bed condition greatly improved, pulse 70. Discharged from hospital. Readmitted for rest two weeks before term, compensation good.

Delivery:

Forceps delivery for maternal distress after 1 hour 15 minutes in the second stage of labour - total length of labour 9 hours 35 minutes.

Puerperium:

Patient in hospital for 19 days, pulse 80 - 100 settled to 80 - 90.

Follow up:

Patient able to do her own housework but rests at frequent intervals and at the end of effort, and has to have extra pillow at night-time.

Comment:

Pregnancy showed the common development of breathlessness at the 28th week, which responds well to effort.

No. 474/44. Age: 34 years. Gravid. 5

Heart lesion: Mitral stenosis.

Previous History:

Scarlet fever in childhood. A definite history of rheumatic fever in adolescence.

Previous Obstetrical History:

Toxaemia with each pregnancy requiring premature induction of labour and A. P. H. with one pregnancy. Children all S.B. except one which died at 3 weeks.

Course of Pregnancy:

General condition good, no undue breathlessness throughout pregnancy but developed pre-eclamptic toxaemia about 28th week of pregnancy. With rest in bed pregnancy continued to her 34th week when the toxaemia not improving the pregnancy was terminated.

Delivery:

A.R.M. at 34 weeks, labour 3 hours, child 4 lbs. 1 oz alive and well.

Puerperium:

Normal, toxaemia subsided, pulse 80.

Postnatal Clinic:

No sign of toxaemia, no breathlessness.

Follow up:

Patient rests at frequent intervals during effort and states that walking makes her tired. Her weight has increased by 1 stone. Pulse 78. Slight cyanosis. X-ray - no enlargement of heart as a whole, minimal L.A. enlargement only. T.D. 118 mm.

Comment.

Toxaemia of pregnancy associated with mitral stenosis, general condition fair with increase in weight adding to her tiredness.

No. 545/44. Age: 23 years. Gravid. 1.

Heart lesion: Mitral stenosis

Previous History:

Rheumatic fever in childhood.

Course of Pregnancy:

No breathlessness throughout pregnancy, stayed in group 1.

Delivery:

Spontaneous at 40 weeks, labour 28 hours 25 minutes, child 8 lbs.

Puerperium:

Normal.

Follow up:

Patient able to do complete days work without any undue breathlessness or rest. There was a further pregnancy one year ago without any decompensation developing. Examination showed lungs and liver normal, no cyanosis, pulse 60. X-ray - heart size and shape was within normal limits.
T.D., 114 mm.

Comment.

Heart able to withstand the strain of both pregnancies without ill effect.

No. 2520/45. Age: 25 years. Gravid. 1.

Heart lesion: Mitral stenosis.

Previous History:

Rheumatic fever at 9 years.

Course of Pregnancy:

Breathless only at the end of effort and patient remained in group II throughout pregnancy, resting duly as advised.

Delivery:

Spontaneous labour at 40 weeks, easy forceps delivery for slight maternal distress after the second stage lasting 2 hours 10 minutes. Child 7 lbs. 15 oz.

Puerperium:

Pulse slightly raised between 90 and 100 the first week, settled to 70 before discharge.

Follow up:

Pulse 86 regular. No oedema or cyanosis, able to do own housework but rests at the end of effort:

Comment.

Patient withstood strain of pregnancy well but 2nd stage of labour would appear to have been unduly prolonged, heart however still well compensated.

No. 1508/45. Age: 24 years. Gravid. 2

Heart lesion: Mitral stenosis and complete heart block.

Previous History:

Rheumatic fever at 15 years, two attacks.

Scarlet fever at 17 years.

Prevous Obstetrical History:

Low forceps delivery of 5 lb. 10 oz. child.

Exercise tolerance during the pregnancy good.

Course of Pregnancy:

No breathlessness throughout. Patient remained in group 1. Pulse 40.

Delivery:

Spontaneous delivery at 40 weeks. Labour 11 hours 5 minutes, child 5 lbs. 15 oz. Delivery easy.

Puerperium:

Normal. Pulse 60 - 70, 65 on discharge.

Postnatal Clinic:

General condition good, no decompensation.

Follow up:

Had a further pregnancy 1½ years ago and was very well during it. No undue breathlessness with effort.

Comment.

The complete heart block appeared to have no bad influence and the patient came through her pregnancies without any decompensation.

No.846/45. Age: 31 years. Gravid. 3

Heart lesion: Mitral stenosis.

Previous History:

Rheumatic fever at 7 years and 20 years.

Obstetrical History:

In 1944 instrumental delivery of 7 lb. 14 oz. baby, in 1942 spontaneous delivery of 8 lb. baby.

Course of Pregnancy:

In early pregnancy there was slight breathlessness at the end of effort. This became more marked by the 20th week with patient having to rest at frequent intervals. About the 28th week there was slight swelling of the ankles with resting pulse 100. The chest was clear although patient had a slight cough. Advised to rest at home, the pulse stayed in the region of 100 to 104 and the patient was eventually admitted to hospital at the 38th week of pregnancy when the breathlessness had slightly increased. Rest in bed produced marked improvement, the breathlessness and slight oedema clearing up.

Delivery:

Spontaneous 40 weeks, six hours labour, child 8 lbs. 14 oz.

Puerperium:

Pulse remained at 80, the patient's general condition good.

Postnatal Clinic:

Breathlessness on moderate exertion but no oedema or cyanosis.

Follow up:

Patient resting at frequent intervals, having occasional swelling of the ankles and sometimes resting on the way back from shopping. Spends extra time in bed and has an extra pillow at night.

X-ray - no L.A. enlargement apparent. T.D. 127 mm.

Comment.

Patient showed a change of group during her pregnancy from II to III, and then back again to II with rest. The strain of pregnancy first showed itself in early 20's and becoming obvious about the 28th week.

No. 998/45. Age: 34 years. Gravid. 3.

Heart lesion: Mitral stenosis.

Previous History:

Tonsillitis several times,with definite history of rheumatic fever at 15 years.

Obstetrical History:

1939 Normal delivery 7 lb. 4 oz. baby. Pregnancy normal. 1943 Miscarriage at 3 months.

Course of Pregnancy:

In group 2 the early part of pregnancy, became slightly breathless on exertion and developed a troublesome cough at the 28th week. At 38 weeks slight oedema of legs appeared and there were moist sounds at the bases. Patient was admitted to hospital for rest and stayed in bed until delivery with marked improvement in her condition.

Delivery:

Normal labour at 40 weeks lasting 2 hours 35 minutes. Child 6 lbs. 8 oz.

Puerperium:

Pulse 80 - 90 the first week, settled to 80. Well on discharge.

Follow up.

The patient states that she was able to do her own housework, has no shortness of breath and does not have to rest unduly.

Comment.

History shows a changing of the groups from II to III during pregnancy and again the development at the 28th week of the breathlessness. Rest in bed restored the patient to her original group.

No.1705/45. Age: 22 years. Gravid. 1

Heart lesion: Mitral stenosis.

Previous History:

Scarlet fever in childhood. Rheumatic fever at 12 years with illness for 2 years.

Course of Pregnancy:

The patient was slightly breathless after exertion but with rest remained in good condition and in the same group.

Delivery:

Normal labour at 40 weeks lasting 6 hours 25 minutes, child 7 lbs. 14 oz.

Puerperium:

Normal, pulse 80 - 90.

Postnatal Clinic:

General condition good, no undue breathlessness.

Follow up:

Able to do housework resting only at end of effort. Occasional swelling of the ankles. Has had one miscarriage since last pregnancy, no change in condition.

Comment.

Patient maintaining her pre-pregnancy condition.

No.1839/45. Age: 31 years. Gravid. 1.

Heart lesion: Mitral stenosis.

Previous History:

Rheumatic fever in childhood.

Course of Pregnancy:

Patient was occasionally breathless on effort during pregnancy, but there was no cough, no oedema or cyanosis.

Delivery:

Caesarean Section and sterilisation at 39th week. Child 7 lbs. 4½ oz.

Puerperium:

Convalescence good, pulse 75 - 80.

Follow up:

Able to do own housework and climb stairs without stopping. No physical limitations beyond the normal.

Comment.

As regards the pregnancy there would appear to be no special reason for the Caesarean Section. The patient's present condition is, however, good.

No. 550/46. Age: 24 years. Gravid. 1

Heart lesion: Mitral stenosis and incompetence.

Previous History:

No definite history of rheumatic fever.

Course of Pregnancy:

Group II during early pregnancy but at 30 weeks developed slight oedema of the ankles and was more breathless on exertion. With rest at home the condition improved and at 37 weeks breathless only at end of exertion. Admitted for rest, however, and the slight oedema of the ankles cleared up.

Developed mild toxaemia of pregnancy.

Delivery:

Forceps delivery for maternal distress after 2nd stage of 1 hour 5 minutes. Patient's pulse was 120. Labour lasted 25 hours 30 minutes. Child 7 lbs. 2 oz.

Puerperium:

Toxaemia subsided and pulse 80 - 90 satisfactory.

Postnatal Clinic:

No albumin, blood pressure normal. No cyanosis or breathlessness.

Follow up:

Pulse 80 regular. No cough, no oedema of ankles. Patient has to rest after climbing a flight of stairs and at intervals during housework. X-ray of heart - no abnormality in size or shape, T.D. 114 mm.

Comment.

The toxaemia of pregnancy is noted but it responded rapidly to treatment. It is noted too that the patient tended to change her group from II to III during pregnancy and this too responded to rest. Her heart condition has been maintained as before pregnancy.

No. 1001/42. Age: 27 years. Gravid. 1

Heart lesion: Mitral stenosis.

Previous History:

Scarlet fever at 19 years; rheumatic fever at 23 years, ill for 5 months. Auricular fibrillation in May 1941, in bed 3 months.

Course of Pregnancy:

Patient had a persistent cough and was slightly breathless on exertion. Slight oedema of the ankles appeared towards the end of pregnancy. There were no signs of congestion of the lungs. She was admitted at the 38th week of pregnancy forrest. Pulse stayed regular throughout pregnancy.

Delivery:

A.R.M. was performed when the patient was thought to be 10 days overdue, forceps delivery was required for maternal and foetal distress, the second stage of labour lasting 55 minutes. Total length of labour 27 hours 40 minutes. Child 7 lbs. S.B.

Puerperium:

Pulse ranged from 80 to 100.

Follow up:

Patient resting at frequent intervals during her housework and unable to climb hills or walk back from shopping without a rest. There is slight swelling of the ankles, a persistent cough and the patient needs an extra pillow at night.

Comment.

Patient gives a history of heart failure before pregnancy and her present condition shows that there has been a deterioration. The length of labour following the A.R.M. was long and it is possible the forceps might have been applied earlier with advantage. Further pregnancies would be a great risk.

No. 760/42. Age: 32 years. Gravid. 1

Heart lesion: Mitral stenosis.

Previous History:

No definite history of rheumatic fever.

Course of Pregnancy:

No antenatal care. Admitted in labour with severe cardiac failure.

Delivery:

Forceps delivery of a child 5 lbs. 5 oz. full term. Patient distressed during delivery. Condition poor.

Puerperium:

Pulse 130 gradually settling to 100 by the end of the 9th day, and to between 70 and 80 by the end of the 3rd week. Placed on digitaline, grains 1/600, two four-hourly for five days, then one four-hourly for five days. Condition fair on discharge.

Postnatal Clinic:

Occasional breathlessness and slight cyanosis present.

Follow up:

Condition has gradually become worse over the last 3 years, patient becoming more breathless and spent 3 months in bed last year, and just up at present after another spell in bed. Unable to do own housework and cannot walk on the level without being breathless. There is a persistent cough, patient needs an extra pillow at night.

Comment.

The absence of antenatal care is noted and patient arrives in labour with severe cardiac failure. Her condition is deteriorating.

No. 2616/46. Age: 26 years. Gravid. 1

Heart lesion: Mitral stenosis.

Previous History:

Chorea and acute rheumatic fever in childhood.

Course of Pregnancy:

There were no symptoms from the heart during the pregnancy and no abnormal signs. The heart appeared fully compensated and patient remained in group I throughout.

Delivery:

Spontaneous labour at 40 weeks lasting 12 hours 35 minutes. Child 7 lbs.

Puerperium:

Pulse 80 - 90.

Follow up:

Patient able to do her own housework, resting only at end of effort. General condition satisfactory.

Comment.

The patient went through her pregnancy well and her condition has in no way retrogressed.

No. 2419/46. Age: 28 years. Gravid. 2

Heart lesion: Mitral stenosis.

Previous History:

Rheumatic fever and nephritis at 12 years.

Obstetrical History:

Spontaneous delivery in 1942 of 6 lb. 11 oz. child, pregnancy normal.

Course of Pregnancy:

Slight breathlessness at the end of exertion in early pregnancy. Slight oedema of ankles developed at the 22nd week but this cleared up with rest. Condition maintained. Admitted two weeks before delivery for rest.

Delivery:

A.R.M. because patient one week overdue. There were short attacks of breathlessness in labour which ended in a forceps delivery under caudal anaesthesia. Length of labour 4 hours 10 minutes, second stage 25 minutes. Weight of child 9 lbs.

Puerperium:

Pulse between 70 and 80, normal.

Postnatal Clinic:

General condition good. Occasional breathlessness only after exertion.

Follow up:

Pulse 80. No oedema or cyanosis, lungs clear. Patient resting at frequent intervals, spends extra time in bed and requires an extra pillow at night. X-ray - heart appreciably smaller than during pregnancy in 1946. T.D. 130 mm.

Comment.

The x-ray finding is noted, suggesting that the heart is less enlarged than during the pregnancy. The A.R.M. for one week post mature is of doubtful obstetrical value and it was noted that there were short attacks of breathlessness in labour.

No. 2262/46. Age: 31 years. Gravid. 1

Heart lesion: Mitral stenosis and incompetence.

Previous History:

Rheumatic fever at 10 and 14 years.

Course of Pregnancy:

Patient in group II during early pregnancy, slight breathlessness showing itself about 20 weeks. There was no increase of dyspnoea until one week before term. The patient was, therefore, admitted for rest, her pulse at the time varying between 100 to 120.

Delivery:

Spontaneous labour at 40 weeks ending in forceps for maternal distress after 2nd stage of labour 1 hour 50 minutes. Total length of labour 11 hours 45 minutes. Condition, however, satisfactory after delivery.

Puerperium:

Pulse between 100 to 110 during the first week, settled to 80.

Postnatal Clinic:

There was slight cyanosis and breathlessness at the end of effort.

Follow up:

Pulse 72. No oedema or cyanosis, lungs clear. Patient able to do her own housework without having to rest unduly. X-ray - well-marked enlargement of heart, L.A.+, L.V.+., T.D. 156 mm.

Comment.

Patient has made a good recovery as regards her symptoms but the x-ray finding of marked cardiac enlargement might suggest that a further pregnancy may again be associated with an increase in pulse rate and the risk of maternal distress on effort.

No. 2127/46. Age: 29 years. Gravid. 1

Heart lesion: Mitral stenosis and incompetence.

Previous History:

Rheumatism in hands and shoulders all her life.

Course of Pregnancy:

A slight increase of breathlessness appeared about the 18th - 20th week. This persisted though not unduly marked: at 36th week had slight haemoptysis and admitted for a complete rest. The blood stained sputum continued for two weeks and then cleared up. General condition fairly good - no oedema or congestion of the lungs.by full term.

Delivery:

Normal delivery at 40 weeks, labour lasting 6 hours 40 minutes. Child 7 lbs. 11 oz.

Puerperium:

Pulse 80 - 100, settling by the end of the 3rd week.

Follow up:

Patient now 9 weeks pregnant again, pulse 76, no cyanosis, able to do own housework. No undue shortness of breadth. X-ray - some enlargement of L.A. to right. T.D. 134 mm.

Comment:

In view of haemoptysis during previous pregnancy patient will require close watching during this one.

No.2087/46. Age: 31 years. Gravid. 1

Heart lesion: Mitral stenosis.

Previous History:

Rheumatic fever and chorea at 10 years.

Course of Pregnancy:

There has been dyspnoea on moderate exertion for three years, worse since the pregnancy. At 22 weeks no oedema, slight cough. At 28 weeks slight increase in breathlessness, lungs clear. Admitted for rest at 36th week because of increase in breathlessness. Dyspnoea improved considerably with rest. Malpresentation , breech converted to vertex but foetus hyper-extended and presenting as a brow.

Delivery:

Classical Caesarean Section for brow presentation with normal pelvis. Child 7 lbs. 9 oz.

Puerperium:

Convalescence good. Pulse settling from 80 to 100, to 70 to 80.

Follow up:

Pulse 80 regular, no cyanosis or oedema or congestion of lungs. Patient able to do housework without undue rest and is fit for a full day's work.
X-ray - Pulm. conus and L.A. prominent. Slight L.V. likely. T.D. 137 mm. Mitral stenosis with some reason for minor L.V. enlargement.

Comment.

Patient started to have pregnancy in group II, her condition deteriorated slightly into group III but again with rest was brought back into group II. The Caesarean Section in the presence of an abnormal presentation was a wise decision. The follow up shows the patient in a satisfactory condition.

No. 1806/46 Age: 28 years. Gravid. 1

Heart lesion: Mitral stenosis.

Previous History:

Rheumatic fever at 5 years, ill for six months. Some restrictions at school. Three heart attacks in 1944.

Course of Pregnancy:

No undue breathlessness at any time, in group I throughout.

Delivery:

Forceps delivery for delay in the second stage. Labour lasting 7 hours, second stage 45 minutes. Child 6 lbs. 13 oz.

Puerperium:

Pulse 90 in the first week, settling to 75.

Postnatal Clinic:

General condition good, no breathlessness.

Follow up:

Pulse 72. No oedema, no congestion of lungs, able to do full day's work without resting. No cough, no swelling of the ankles. X-ray - no shape abnormality of the heart, T.D. 119 mm.

Comment.

It would appear that pregnancy has had no adverse effect on the patient.

No. 1787/46. Age: 31 years. Gravid. 2

Heart lesion: Mitral stenosis.

Previous History:

Rheumatic fever at 10 years, restrictions at school.

Obstetrical History:

One miscarriage at 3 months.

Course of Pregnancy:

Patient well compensated throughout but there was a slight increase of breathlessness noted at the 28th week of pregnancy, responding to rest. Admitted for rest in bed two weeks before delivery.

Delivery:

Normal spontaneous delivery after a labour of 18 hours 25 minutes. Child 6 lbs. 12½ oz.

Puerperium:

Pulse 70 - 90, settling during second week.

Postnatal Clinic:

General condition satisfactory, no undue breathlessness.

Follow up:

Pulse 64. No oedema and no cyanosis. Chest clear. Able to do own housework but resting at the end of effort. X-ray - no cardiac enlargement, T.D. 112 mm. Doubtful if L.A. enlarged.

Comment.

Pregnancies have not apparently had any adverse effect.

No. 1751/46. Age: 33 years. Gravid. 2

Heart lesion: Mitral stenosis.

Previous History:

Rheumatic fever in childhood, with games restriction at school.

Obstetrical History:

Normal delivery 1943 of 7 lb. 2 oz. child.

Pregnancy normal.

Course of Pregnancy:

Patient in group II throughout, slight breathlessness if she hurries unduly or at the end of effort.

Rested one week before labour.

Delivery:

Normal delivery at term, labour 6 hours 50 minutes, child 8 lbs. 14 oz.

Puerperium.

Pulse 75 - 95 first week, settling to 70.

Follow up:

Patient able to do her own housework but has to rest at the end of effort and states that she becomes very tired. Has occasional slight swelling of the ankles.

Comment.

Condition maintained as in the pre-pregnant state.

No. 1697/46. Age: 27 years. Gravid. 4

Heart lesion: Mitral stenosis and incompetence.

Previous History:

Rheumatic fever in childhood, chorea three times.

Obstetrical History:

Two miscarriages and one full term child 8 lbs. 8 oz. No undue breathlessness.

Course of Pregnancy:

Group II in early pregnancy but at the 35th week developed congestion of the lungs and oedema of ankles. Admitted to hospital for rest. Condition improved with rest in bed and congestion at bases

cleared.

Delivery:

Normal labour at term lasting 8 hours 5 minutes.

Child 7 lbs. 13 oz.

Puerperium:

Pulse 80 - 100 first week, settled to 70.

Postnatal Clinic:

Slight breathlessness at the end of effort. General condition fairly good.

Follow up:

Able to do own housework and resting only at the end of effort. Occasional swelling of the ankles. No cough, cyanosis or oedema; lungs clear. Pulse 74.

X-ray - appreciable cardiac enlargement,

L.A. enlarged.

Comment.

Patients remark that she feels as good now as before pregnancy, does not cover the fact that during the pregnancy there was cardiac decompensation.

No. 1444/46. Age: 23 years. Gravid. 1

Heart lesion: Mitral stenosis.

Previous History:

Rheumatic fever in childhood.

Course of Pregnancy:

Well all through pregnancy, continuing in group I with the cardiac lesion well compensated.

Delivery:

Forceps delivery for delay in advance of head after 40 minutes in second stage. Total length of labour 19 hours 30 minutes. No maternal or foetal distress. Child 7 lbs. 6 oz.

Puerperium:

Pulse 75 - 95, settling to 70.

Postnatal Clinic:

No sign of decompensation. General condition good.

Follow-up:

No complaints. Able to do a full day's work without resting.

Comment.

Pregnancy would appear to have had no adverse effect on the heart.

No. 1345/46. Age: 30 years. Gravid. 2.

Heartlesion: Mitral stenosis.

Previous History:

Chorea, 4 attacks 11 to 17 years; rheumatic fever at 12 years.

Obstetrical History:

One normal pregnancy and labour with child 6 lbs.8 oz.

Course of Pregnancy:

Group II in early pregnancy but at 32nd week there was slight distress on effort like climbing stairs. This continued despite rest at home and the patient was admitted at 38th week with a few basal crepitations. Rest in bed restored her condition back to group II, the lungs clearing up and the breathlessness decreasing.

Delivery:

Normal delivery at term, labour 14 hours 35 minutes, child 6 lbs. 8 oz.

Puerperium:

Pulse 80 - 90, settling to 75.

Follow up:

Able to do own housework without undue breathlessness has occasional swelling of the ankles. Pulse 88. No cyanosis, lungs clear. X-ray - moderate enlargement of heart, L.V.+, L.A.+, T.D. 145 mm.

Comment.

Patient's condition now good but history during pregnancy shows that it was a strain on her heart. Her condition deteriorated slightly but was restored again with rest.

No. 893/46. Age: 28 years. Gravid. 1

Heart lesion: Mitral stenosis.

Previous History:

Rheumatic fever at age of 24, ill for 3 months.

Course of Pregnancy:

There was slight dyspnoea on exertion which persisted throughout pregnancy. The pulse varied between 70 and 100. Patient was admitted for rest and the pulse settled to average about 80.

Delivery:

Forceps delivery for slight maternal distress, the presentation was L.O.P. and labour lasted 46 hours the first stage, and 40 minutes the second stage. Child 7 lbs. 8 oz.

Puerperium:

Pulse 85 - 108, settling towards end of second week.

Postnatal Clinic:

Slight breathlessness at end of exertion. No cyanosis. Pulse 80.

Follow up:

Patient states that she took things very carefully for two to three months and then felt better. She is able now to do a full days work and can climb stairs with only occasionally resting at top.
X-ray - no enlargement as a whole, pulm. conus prominent. T.D. 119 mm.

Comment.

Pregnancy was obviously a slight strain on patient but her condition is now back to the pre-pregnant state.

No. 695/46. Age: 31 years. Gravid. 1

Heart lesion: Mitral stenosis.

Previous History:

Rheumatic fever at 11 and 20 years.

Course of Pregnancy:

Heart well compensated, breathless only at the end of effort and not worse with the pregnancy. No cyanosis, no oedema.

Delivery:

Normal delivery at 40 weeks, labour lasting 8 hours. Child 8 lbs. 2 oz.

Puerperium:

Pulse 80 - 90 first week, settling 70 - 80.

Follow up:

no physical signs except heart lesion. Able to do own housework and no undue breathlessness. X-ray dominant enlargement to right probably mainly L.A. T.D. 142 mm.

Comment.

Patient's condition as in pre-pregnant state.

No. 646/46. Age: 39 years. Gravid. 1

Heart lesion: Mitral stenosis and aortic regurgitation.

Previous History:

Rheumatic fever at 19 years.

Course of Pregnancy:

The patient had had only slight breathlessness at the end of effort during pregnancy except in the latter weeks when there was an increase in dyspnoea with an increase in pulse rate. No signs of cardiac insufficiency developed.

Delivery:

Lower segment Caesarean Section and sterilisation at 38 weeks, child 6 lbs. 13 oz.

Puerperium:

Convalescence good, pulse settling from 80 to 100, to 70 to 80.

Postnatal Clinic:

No undue breathlessness, general condition good.

Follow up:

Pulse 80, no congestion of lungs, no oedema. Able to do housework and no need to rest after ordinary effort. X-ray slight cardiac enlargement, L.A. not apparently +, T.D. 136 mm.

Comment:

Patient came through her pregnancy well, and condition as good as pre-pregnancy state.

No. 126/46. Age: 29 years. Gravid. 1

Heart lesion: Mitral stenosis.

Previous History:

Rheumatic fever and scarlet fever in childhood.

Course of Pregnancy:

Group II in early pregnancy and remained in good condition until about the 30th week when she complained of some increase in breathlessness. No signs of decompensation were found however, but pulse rate was slightly raised. She also had some degree of hydramnios and was admitted for rest in bed.
A pyelitis was also found and was treated. Patient's pulse was persistently 100 - 115and she was given digitalis folia grains 2 three times a day for three days, and then grain 1 daily which was gradually reduced. Pulse settled to 70 - 80 before delivery.

Delivery:

Prolonged first stage of 83 hours 30 minutes, patient agitated with labour and there was some foetal distress. Forceps delivery at the beginning of the second stage. Child 7 lbs. 12 oz.

Puerperium:

Pulse 110 - 125 for the first week, settled to 80 by the second week. Patient discharged in good condition.

Follow up:

Patient spends extra time in bed and has to rest at frequent intervals and sometimes during effort.

Has swelling of the ankles and a persistent cough. Pulse 118 - 120 fast regular, lungs clear, no oedema, no cyanosis. X-ray - doubtful whether L.A. enlarged, T.D. 130 mm.

Comment.

The main feature about this case has been the increase in pulse rate which was present during pregnancy and has persisted, and to which has now been added a persistent cough and swelling of the ankles.

No. 50/46. Age: 24 years. Gravid.1

Heart lesion: Mitral stenosis.

Previous History:

Scarlet fever in childhood. Rheumatic fever two years ago.

Course of Pregnancy:

No sign of decompensation in early pregnancy. At 28 weeks however, there was some breathlessness at the end of effort. With rest, however, her condition was maintained in group I.

Delivery:

Normal delivery at term, labour lasting 19 hours 15 minutes. Child 6 lbs. 14 oz.

Puerperium:

Pulse 100 for the first day, then settled.

Discharged well.

Postnatal Clinic:

No complaints, general condition good, no undue breathlessness.

Follow up:

Patient is able to do her own housework but tires very quickly and has to rest at intervals during effort. There is no cough however, and no swelling of the ankles.

Comment.

Patient has not quite regained her pre-pregnancy state but this she thinks is due to the amount of work she has to do in her home.

No. 17/46. Age: 34 years. Gravid. 2

Heart lesion: Mitral stenosis.

Previous History:

Rheumatic fever 10 years ago.

Previous Obstetrical History:

1942 instrumental delivery of a 10 lb. 1 oz. child, normal pregnancy.

Course of Pregnancy:

Group II in early pregnancy with slight breathlessness at the end of effort at the 8th week of pregnancy. By the 30th week of pregnancy had developed a cough and was breathless after slight exertion and required 3 pillows. There was no oedema or cyanosis however, and the lung bases were said to be clear. Patient was brought in to rest from the 34th to the 36th week of pregnancy, then discharged with condition much improved. At 37th week, however, was breathless even at rest or lying down and had to maintain a sitting position. There was no cyanosis or cough and no oedema, but crepitations were present at the bases. With rest condition greatly improved, the breathlessness disappeared when at rest but crepitations persisted at the bases.

Delivery:

A spontaneous easy labour, slightly overdue, lasting 6 hours and weight of child 9 lbs. 4 oz. No distress during labour.

Puerperium:

Condition improved, pulse settling to 80 but patient still slightly breathless at the end of short exercise on discharge from hospital.

Post-natal Clinic:

No undue breathlessness. General condition fairly good, lungs clear.

Follow up:

Patient can't do very much and tires more quickly than before the last pregnancy. She has to rest at frequent intervals during effort. There is a persistent cough and slight swelling of the ankles. She has to spend extra time in bed and needs an extra pillow in bed at night-time. Pulse 100, no cyanosis.Chest clear.

Comment.

Patient is definitely worse than before her last pregnancy which appeared to cause a great deal of strain. During the pregnancy patient started in group II and her condition deteriorated into Group IV. Rest however, bringing her into Group III again. The decompensation apparently appearing after the 30th week.

No. 2635/46. Age: 25 years. Gravid. 1

Heart lesion: Mitral stenosis.

Previous History:

Rheumatic fever at 12 years.

Course of Pregnancy:

Symptom-less in early pregnancy with pulse slow and regular. At the 24th week of pregnancy however, was very breathless on the least exertion and two weeks later developed some oedema of the legs. With rest

in bed however, condition improved rapidly and the patient was up again soon. Condition maintained except for oedema which re-appeared and for which patient was admitted 14 days before labour.
Breathless only at the end of effort. Lungs clear.

Delivery:

Normal delivery one week before term, labour lasting 32 hours 25 minutes. Patient slightly cyanosed at end of second stage and given oxygen. Pulse 90 regular.

Puerperium:

Normal, pulse settled and no cyanosis.

Follow-up:

Has complained of increased tiredness since pregnancy, pulse 100, chest clear, no cyanosis. Patient states that she has to rest at frequent intervals. Has slight swelling of the ankles.

Comment.

History again shows patient may change her group during pregnancy and her condition is not so good as before pregnancy. The first stage of labour was prolonged and the second stage had lasted 1 hour 40 mins: This stage might have been terminated with benefit to patient probably one hour earlier, and this would emphasize the need for a doctor to be present for the whole of the second stage of labour in all heart cases.

No. 2705/46. Age: 44 years. Gravid. 2

Heart lesion: Mitral stenosis.

Previous History:

No definite history of rheumatic fever.

Obstetrical History:

Normal delivery of a 7 lb. 4 oz. baby seven years previously.

Course of Pregnancy:

This is fully described in page 286 .

Follow up:

Patient's general condition surprisingly good after the history of severe cardiac failure during last pregnancy. No cyanosis, no oedema present, lungs clear. Apparently after resting for five weeks following the birth of her baby, patient made quick recovery and is now able to do a full day's housework and able to climb hills without resting even at the top. X-ray - heart slightly enlarged but much less than in November '46 when she was admitted to hospital in heart failure. L.A. slightly +, T.D. 137 mm.

Comment.

History again showed how pregnancy may affect the heart and how patient's condition may change from group II to group IV during pregnancy, and how a vaginal delivery can be successful in the group IV patient.

No.
2523/46. Age: 29 years. Gravid. 3

Heart lesion: Mitral stenosis.

Previous History:

Rheumatic fever at 7 years.

Obstetrical History:

1940 normal delivery 8 lbs. 8 oz. baby, 1943 normal delivery 7 lbs. 8 oz. baby. Did not notice any breathlessness with these two pregnancies.

Course of Pregnancy:

In group II in early pregnancy and remained well until the 28th week when breathlessness increased. Condition improved with rest at home and was maintained until term.

Delivery:

Normal labour lasting 7 hours 50 minutes at term, child 7 lbs. 8½ oz.

Puerperium:

Normal, pulse 60 - 80, well on discharge with no breathlessness.

Postnatal Clinic:

No complaints, no undue breathlessness.

Follow up:

Fit for housework but occasionally resting during effort. No cough, no swelling of ankles but has to have an extra pillow at night.

Comment:

Patient succeeded in passing through the first two pregnancies without undue breathlessness but felt the strain of the third pregnancy more.

No.1719/43. Age: 21 years. Gravid. 1

Heart lesion: Mitral stenosis.

Previous History: No definite history of rheumatic fever.

Course of Pregnancy.

Patient remained in group I throughout her pregnancy.

Delivery:

Normal delivery at term, labour lasting 16 hours 15 minutes. Child 8 lbs. 1½ oz.

Puerperium:

Pulse between 80 and 90, satisfactory.

Postnatal Clinic:

General condition good, no signs of decompensation.

Follow up:

Patient fit for full day's work without breathlessness. No limitation of her physical work. Has had one further pregnancy a year ago and was perfectly well during it.

Comment:

This patient has stood well up to the strain of two pregnancies.

No. 16/42. Age: 27 years. Gravid. 1

Heart lesion: Mitral stenosis.

Previous History:

A definite history of rheumatic fever at 17 years.

Course of Pregnancy:

In group I throughout pregnancy and without limitation of activities. Developed a mild toxaemia of pregnancy at the 38th week.

Delivery:

Rupture of membranes at term for toxaemia of pregnancy. Labour lasted 28 hours 55 minutes. Child 7 lbs. 9 oz.

Puerperium:

Normal, good condition on discharge.

Follow up:

Patient had one further pregnancy two years ago, and was well throughout. She is fit to do a full day's work without breathlessness. There is no cough and she sleeps well.

Comment.

Toxaemia of pregnancy treated by A.R.M. with delay in labour noted. No apparent change in patient's condition since before her pregnancies.

No. 138/42. Age: 27 years. Gravid. 1

Heart lesion: Mitral stenosis and aortic incompetence.

Previous History:

Rheumatic fever at 9, 21 and 25 years.

Course of Pregnancy:

First seen at 14 weeks pregnancy, slight tachycardia, no oedema, no cyanosis, rather breathless on exertion, no liver enlargement, chest clear. Continued to rest at home throughout pregnancy until the 28th week when she was admitted to hospital. General condition then fair. Pulse 100, a few creptitations at the bases, no liver enlargement, no oedema. Patient developed a right sided pyelitis which responded to treatment. Patient discharged from hospital, condition fair, breathless if undue effort, chest clear, no cyanosis. Re-admitted in labour at the 37th week.

Delivery:

The first stage was 6 hours 45 minutes but patient was distressed by the end of that stage and forceps delivery was performed after 25 minutes in second stage. The patient's pulse 104 - regular. Respirations rapid. Child 5 lb. 3 oz.

Puerperium:

Patient's condition improved, pulse settled from 110 to 84, slight breathlessness on effort.

Postnatal Clinic:

Condition fair, pulse 100, dyspnoeic when lying down. Has a cough with few crepitations at lung bases. No oedema.

Follow up:

Patient died June '44.

Comment:

Patient's condition throughout pregnancy had been poor. It is noted however that the early weeks of pregnancy were associated with marked disability, and termination of the pregnancy might have been considered but it is doubtful whether it would have altered the further course. Patient too was anxious to have a child.

No. 223/42. Age: 25 years. Gravid. 1

Heart lesion: Mitral stenosis and incompetence.

Previous History:

Rheumatic fever at 5, 7 and 14 years.

Course of Pregnancy:

Patient in group II with well compensated heart lesion when first seen at 14 weeks. At 18 weeks became decompensated with moist sounds at left base, slight oedema and an increase in breathlessness. With rest at home condition improved but one month later cough and orthopnoea. Admitted for rest at 26th week of

pregnancy. At this stage patient in group IV. Moisture at both bases, breathless lying in bed and therefore propped up to sleep, no oedema, slight cyanosis. With ten days rest condition greatly improved. Cough less troublesome and patient less breathless. By 30 weeks patient was discharged, greatly improved with chest clear and breathless only at end of effort. She maintained this improved condition and was re-admitted for rest two weeks before full term.

Delivery:

Normal at term, length of labour ten hours thirtyfive minutes. Child 7 lbs. 3 oz. The patient did not show any signs of distress.

Puerperium:

Good, pulse 80 - 90, settled with rest.

Postnatal Clinic:

General condition fairly good but states that she has a slight increase in breathlessness and occasional pain in the chest. No oedema, chest clear, Pulse 80.

Follow up:

Patient able to do her own housework resting only at the end of effort. She states however that she has a persistent cough.

Comment.

The case illustrates again that the patient can change her group throughout pregnancy and that pregnancy is a strain on the heart. It also shows the

importance of rest during pregnancy.

No. 1320/43. Age: 34 years. Gravid. 1

Heart lesion: Mitral stenosis.

Previous History:

Scarlet fever in childhood. A definite history of rheumatic fever at 10 and 15 years.

Course of Pregnancy:

Patient in group II throughout pregnancy, breathless at the end of exertion only.

Delivery:

Normal delivery at term, labour lasting 23 hours 35 mimutes. Child 7 lbs. 13 oz.

Puerperium:

Pulse normal throughout, discharged well.

Postnatal Clinic:

No dyspnoea, no cough, general condition good.

Follow up:

Able to do a full day's work with some breathlessness at the end of effort. There is however a persistent cough. Pulse 62 - regular, no oedema, chest clear. X-ray - No cardiac enlargement, T.D. 126 mm. Heart slightly over to left no apparent cause.

Comment.

Patient stood up to the strain of pregnancy well, but her cough, which she says she has every year, has been slightly more persistent.

No. 1236/45. Age: 30 years. Gravid.2

Heart lesion: Mitral stenosis.

Previous History:

Rheumatic fever in childhood.

Course of Pregnancy:

Patient was well throughout the whole of her pregnancy and had no undue restrictions to her activities. Group I throughout.

Delivery:

Spontaneous delivery at term of a 6 lb. 8 oz. baby after labour of 17 hours.

Puerperium:

Normal.

Postnatal Clinic:

Patient enjoying normal health and activity.

Follow-up:

Patient at present 36 weeks pregnant and has been in group I up to date.

Comment.

Patient appears to maintain her pre-pregnancy condition satisfactorily.

No. 1003/42. Age: 24 years. Gravid. 1

Heart lesion: Mitral stenosis.

Previous History:

Acute rheumatism at 21 and 22 years. Ill for one month each time.

Course of Pregnancy:

Patient well throughout pregnancy apart from anaemia which was treated with iron. Breathlessness at the end of exertion.

Delivery:

Normal delivery at term, labour 13¾ hours.

Child 7 lbs. 6 oz.

Puerperium:

Normal. Pulse 80 - 95.

Postnatal Clinic:

No undue breathlessness. General condition satisfactory. Pulse normal.

Follow up:

Had another pregnancy two years ago, and has been more tired since. States that she felt fine after first pregnancy. Now resting at frequent intervals but able to do own housework. No oedema or cough.

Comment.

Apparently stood up to the strain of the first pregnancy better than the second. Some increase in physical limitations now.

No. 532/43. Age: 35 years. Gravid. 3

Heart lesion: Mitral stenosis.

Previous History:

Chorea 9 years, rheumatic fever 3 times in childhood.

Obstetrical History:

Two normal deliveries in 1930 and 1934. Pregnancies normal.

Course of Pregnancy:

Condition good until the 22nd week when some breathlessness appeared and increased. There was no oedema at first but it developed by the 28th week. A cough appeared and at the 29th week there were signs of decompensation with cyanosis and breathlessness. Pulse 100, liver not enlarged. Admitted to hospital. Had sudden severe pain in the left arm when in hospital but the condition did not suggest coronary disease. Her condition improved with rest, cyanosis gradually disappearing.

Patient remained in hospital however until delivery.

Delivery:

Patient dyspnoeic and cyanosed during pains and slightly dyspnoeic between pains. Pulse 80, volume good. Continuous oxygen given and $\frac{1}{200}$th of a grain strophanthin injected. First stage 17 hours 33 mins., second stage forceps delivery at the beginning of the second stage. General condition good, not breathless or cyanosed after labour.

Puerperium:

Normal, pulse 70 - 80.

Postnatal Clinic:

No undue breathlessness, no oedema, pulse 80.

Follow up:

Patient has to rest at frequent intervals, having to spend extra time in bed. Cannot climb stairs without stopping. Has slight oedema of the ankles and a persistent cough. Pulse 100, lungs clear.

X-ray - appreciable cardiac enlargement, T.D.144 mm.

Comment.

Patient apparently changed from group II to group III during pregnancy, the change in condition showing itself at the beginning of the second half of pregnancy. The third pregnancy appeared to have made in-roads into her cardiac reserve and there is an increase in the physical limitations.

No. 484/44. Age: 32 years. Gravid. 2.

Heart lesion: Mitral stenosis.

Previous History:

Rheumatic fever 10 years ago.

Obstetrical History:

Spontaneous delivery at term following normal pregnancy, child S.B. (cord round neck)

Course of Pregnancy:

Admitted 10 days before term as an emergency with cyanosis, pulse 96, breathless on moving about and unable to sleep in normal position without respiratory distress. No oedema, some congestion at bases. The patient states well until 26th week when ankles and legs began to swell. Her doctor sent her to bed and the swelling cleared up after 3 weeks. Since then she has remained in bed with no recurrence of the swelling but dyspnoea on exertion. Condition improved with rest. Pulse 110 to 90.

Delivery:

Two weeks overdue from dates, labour lasting 3 hours 40 minutes with a second stage of 10 minutes. Spontaneous delivery without distress.

Child 6 lbs. 14 oz.

Puerperium:

Appeared satisfactory with pulse between 80 and 90 except for the third day when it was 100, and then on the 6th day pulse increased to 110 and temperature rose to 100°. Oedema appeared in both legs and moist sounds were heard at the lung bases. Condition became worse with the liver enlarging and the neck veins dilating and there was consolidation of the right lung base with temperature 104°, pulse 130, respiration 36 and purulent sputum.

Digitalis grains 1 given three times a day and sulphathiazole for the pneumonia. On the 12th day there was slight improvement in the condition which gradually cleared up and she was discharged on the 28th day, the pulse settling to between 80 and 90 and patient slightly breathless only after exertion.

Postnatal Clinic:

No cyanosis or oedema, occasional cough, slight breathlessness on exertion. Pulse 80.

Follow up:

Patient has to rest at frequent intervals and cannot climb stairs without stopping nor walk back from the shops without a rest. There is no cough or oedema of the ankles but she spends some extra time in bed.

Comment.

Patient apparently came through her first pregnancy without trouble but developed cardiac failure during the second. The decompensation again appearing about the 26th week. Her present condition shows that she has not recovered her pre-pregnancy state.

No.
615/44. Age: 47 years. Gravid. 11

Heart lesion: Mitral stenosis.

Previous History:

No note made of previous illnesses.

Obstetrical History:

Ten normal deliveries between 1923 and 1941. Weight of child 6¾ lbs. to 8½ lbs. Patient stated she was well during her pregnancies.

Course of Pregnancy:

First seen at 28th week of pregnancy when she complained of breathlessness on exertion. No oedema or cyanosis present: lungs clear. General condition poor, anaemic. Did not report further until 36th week of pregnancy. No oedema present, lungs clear, frequent extrasystoles: advised to come into hospital for rest although no sign of failure. Patient did not come in to hospital.

Delivery:

Patient admitted in labour, pulse 84, general condition fair. Labour lasted 6½ hours with a second stage of 10 minutes. Child 7 lbs. 11 oz. born spontaneously. No distress during delivery.

Puerperium:

Normal, pulse 70 - 85, discharged fairly well no breathlessness.

Follow up:

Patient died 3½ years later after a stroke. This was the second stroke in less than 12 months.

Comment.

Considering the mitral stenosis lesion and the age of the patient at death, the pregnancies do not seem to have affected the course of the disease.

No. 641/44. Age: 19 years. Gravid. 1

Heart lesion: Paroxysmal tachycardia, heart sounds clear.

Previous History:

Attending heart hospital since age of 4 years. Breathlessness on exertion in childhood.

Course of Pregnancy:

Early pregnancy condition good apart from slight anaemia. At 32 weeks first complained of some breathlessness and occasional swelling of the ankles. Tachycardia persistent, heart sounds clear. At 33 weeks complained of palpitations and occasional fainting. Tachycardia regular, pulse 120. Admitted at 39 weeks for rest. Condition good, no breathlessness, no oedema, no tachycardia.

Delivery:

Spontaneous normal labour lasting 12 hours 45 mins.

Puerperium:

Uneventful, pulse 80 - 92. Discharged 10th day well.

Postnatal Clinic:

General condition good. No tachycardia, no breathlessness.

Comment.

The condition of tachycardia was not affected by the pregnancy.

No. 213/45. Age: 23 years. Gravid. 1

Heart lesion: Mitral stenosis

Previous History:

Rheumatic fever at 12 and 18 years.

Course of Pregnancy:

First seen at 18 weeks when there was slight breathlessness on exertion. By the 26th week there was breathlessness after slight exertion only. Pulse normal and regular rhythm, lungs clear. By the 34th week there was more breathlessness, oedema of ankles and pulse 118. Patient admitted at 36th week for rest. Her breathlessness increased and she had changed from group II to group III. After one week's rest in hospital the breathlessness cleared up and she was restored to group II and remained in this group until delivery.

Delivery:

At 40 weeks, spontaneous, labour lasting 10 hours 40 minutes, normal. Child 7 lbs. 11 oz.

Puerperium:

Normal, pulse 94 settled to 84 on discharge.

Post-natal Clinic:

No cardiac symptoms, general condition satisfactory.

Follow up:

Patient able to do her own housework, resting at the end of effort and at times during effort. Spends extra time in bed and has an extra pillow at night. Pulse 78. No cyanosis. X-ray, moderate cardiac enlargement, L.A.+, conus and L.A. moderately enlarged, T.D. 149 mm.

Comment.

Patient again showed the change of group during pregnancy, coming on after the 24th week. The pregnancy was obviously a strain and left her with increased physical limitation.

No. 473/45. Age: 25 years. Gravid. 1

Heart lesion: Mitral stenosis, mitral incompetence and aortic incompetence.

Previous History:

Rheumatic fever at 8 and 11 years.

Course of Pregnancy:

First seen at 8 weeks, group II. At 16 weeks group III and at 24 weeks patient showed cyanosis, enlarged liver and complained of an increase in breathlessness and was admitted to hospital. Oedema of legs spread up to sacrum, lungs were clear and pulse regular. At 33 weeks there was little change in the patient's condition, improvement appeared to be stationary and termination of the pregnancy decided upon.

Delivery:

Classical Caesarean Section at 33 weeks, child 4 lbs. 4 oz.

Puerperium:

Convalescence satisfactory. Pulse settling from 112 to 80 on discharge: oedema clearing up and breathlessness not present on moderate exertion.

Follow up:

Patient unable to climb stairs without stopping and cannot walk back from shops without a rest. No cough or swelling of the ankles but requires extra pillow at night time.

Comment.

Pregnancy appeared to have been a strain on the heart and her condition has deteriorated.

No. 1614/46. Age: 25 years. Gravid. 1

Heart lesion: Mitral stenosis.

Previous History:

Chorea at 9 years, in hospital for six weeks and again in hospital at 14 years.

Course of Pregnancy:

First seen at 9 weeks, no oedema, always slightly breathless on exertion. At 16 weeks no dyspnoea or any other sign of failure. Patient's condition continued unchanged until the 36th week, when she complained of an increase in breathlessness. At 38th week there was also slight oedema of the ankles. Admitted at 39 weeks with an increase in breathlessness, pulse 108, slight oedema of ankles, chest clear. Went into labour next day.

Delivery:

Forceps delivery at 39th week after the 2nd stage had lasted 30 minutes and no advance. Patient's condition was satisfactory throughout. Child 7 lbs. 13 oz.

Puerperium:

Rise in pulse the 4th and 5th day to 120 but gradually settled to 80. Condition satisfactory on discharge.

Post-natal Clinic:

Heart well compensated. No undue breathlessness.

Follow up:

Is able to do housework resting only at the end of effort. No oedema, no cyanosis, pulse 80, regular, no congestion of the lungs. Has to have an extra pillow at night. X-ray, heart enlarged slightly, T.D. 144 mm.

Comment:

Patient has made a good recovery from her pregnancy which appeared to cause some strain on heart.

No. 1/44. Age: 32 years. Gravid. 2

Heart lesion: Mitral stenosis.

Previous History:

A definite history of rheumatic fever and tonsilitis.

Obstetrical History:

Normal delivery after a normal pregnancy, two years ago.

Course of Pregnancy:

First seen at 28th week of pregnancy, slight breathlessness at the end of exertion: no cyanosis or oedema. At 31st week slight oedema of the ankles, moisture at left base, tachycardia, pulse 100. Admitted for rest. Patient's condition rapidly improved with complete rest, the pulse rate dropping to 84 and the chest became clear. She was discharged after 9 days rest with no sign of decompensation. Re-admitted in premature labour.

Delivery:

At 38th week normal, labour lasting 6 hours 10 mins. Patient's condition good.

Puerperium:

Normal, pulse settling from 96 to 78. Condition good.

Postnatal Clinic:

Compensated mitral stenosis, no undue breathlessness.

Follow up:

Since pregnancy in 1944 patient has had occasional attacks of haemoptysis and one year ago had an attack of heart failure: has been on digitalis since then. Pulse, slow fibrillation present. Unable to do work without frequent rest. No oedema, no cough.

Comment.

Patient's condition has deteriorated. The history of the last pregnancy showed that it was a definite strain on the heart although it responded to rest in bed.

No. 753/42. Age: 35 years. Gravid. 2

Heart lesion: Myocarditis, heart sounds normal.

Previous History:

Rheumatic fever at 15 years and 18 years of age.

Obstetrical History:

First pregnancy, normal delivery at term, well during pregnancy.

Course of Pregnancy:

First seen at 20 weeks with some breathlessness, patient 17 stone in weight, blood pressure 140/80. Condition maintained in group II, but patient admitted for rest because of increased breathlessness

at 32 weeks, when there was also slight oedema of ankles and legs and blood pressure 150/90. Improved with rest and pulse settled from 120 to 96, and blood pressure dropped to 100/60. Discharged well.
Re-admitted for rest during the last 3 weeks of the pregnancy. Patient maintained in group II.

Delivery:

At term, normal, labour lasting 11½ hours. No maternal distress.

Puerperium:

Pulse settled from 100 to 78. The patient discharged in a satisfactory condition.

Postnatal Clinic:

Breathless only after exertion, no signs of cardiac decompensation, pulse 80.

Follow up:

Patient states that she is not so active since the last pregnancy and has to rest at intervals. She has a persistent cough, slight swelling of the ankles and requires an extra pillow at night. Is still much over weight. X-ray, T.D. 155 mm. difficult to say if appreciable enlargement of heart because of weight of patient.

Comment.

Patient's excessive weight adds to her breathlessness, but added burden of pregnancy is a strain on her, and her condition is not as good as before the last pregnancy.

No.538/42. Age: 28 years Gravid.1

Heart lesion: Mitral stenosis.

Previous History:

A definite history of rheumatic fever at 9 years.

Course of Pregnancy.

Patient noticed a shortness of breath from time to time which had increased slightly by the 28th week of pregnancy. She was admitted at that time for a pyelitis of pregnancy which responded to treatment. At that time it was noted that the lungs were clear, liver normal and no oedema. The pulse which had been rapid with the pyelitis fever settled to 105 but was still less on discharge. The patient maintained her condition as a group II case and was re-admitted in labour at the 34th week of pregnancy.

Delivery:

Labour commenced with premature rupture of the membranes and lasted 7 hours 10 minutes, the child being 4 lbs. 14 oz. in weight. The patient's general condition was noted as being fairly good during labour and there was no signs of decompensation of the heart.

Puerperium:

During the third day of the puerperium patient felt faint during breast feeding and the pulse became poor in quality and remained so for five minutes. The following day she developed a slight cough which

increased and at the fifth day crepitations were present at both lung bases especially the left base. By the sixth day there was an obvious increase in breathlessness with the slightest effort in bed and breast feeding had to be stopped. The pulse which had hitherto been 95 - 100 rose on the 6th day to 120 and stayed between 110 - 120 for five days and then gradually settled to 95. There was no rise in temperature. The patient's general condition improved gradually, the chest clearing up and the breathlessness becoming less. There was never any oedema nor marked cyanosis. The patient was discharged two weeks later, the breathlessness having cleared up and not appearing with moderate effort: pulse, however, was still slightly rapid at 95. She did not attend the postnatal clinic.

Follow up:

Patient was never well after the birth of this baby and had to spend most of the time in hospital until she died in June 1947. The enquiry reveals that the ankles kept swelling and that the oedema gradually spread up her legs to become generalised.

Comment.

It would seem that the acute infection in the form of pyelitis which the patient had during her pregnancy

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It would seem that the acute infection in the form of pyelitis which the patient had during her pregnancy

was associated with a rise in pulse rate which persisted unduly. This was further seen during the puerperium when the pulse was abnormally high in the early days, and was associated with a heart attack. The pregnancy had evidently an adverse effect on the heart condition and the patient died of heartfailure five years later.

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From a study of the history and follow-up of the 60 patients in this series several features of interest are noted and a number of questions can be considered.

The importance of antenatal care for the patient with heart disease is shown, since the only patient 760/42 who had no pre-natal care was admitted in labour with severe heart failure. The dilatation of the heart during pregnancy as a result of failure is seen in the records of case 2705/46, with a recession of its size when the strain of pregnancy is removed.

Of the three patients who died, one 615/44 with mitral stenosis lived to the age of 50½ years and had two strokes, presumably cerebral emboli, in the last year of life; and had borne 11 children the last, 3½ years before death, without any of the pregnancies causing any undue distress. The second, 538/42, showed a progressive heart failure and died at the age of 33 years. The congestive heart failure was evident during the puerperium of her pregnancy at the age of 28 years. The third, 138/42, showed signs of congestive heart failure when first seen in the early weeks of pregnancy, and died at the age of 29 years. The first patient was in Group II throughout her pregnancy the second changed to Group IV during the puerperium, and the third was in Group III early in her pregnancy.

Lamb (1937) who had followed up a series of 78 women for a period extending from one month to nine years after delivery, found that six of them had died as follows:-

1. died suddenly following a recurrence of rheumatic carditis nine months following pregnancy.
2. died 5 months after hysterotomy and sterilisation, death due to cardiac decompensation.
3. died 2½ years after last pregnancy with congestive heart failure.
4. died after living as a chronic invalid.
5. died 7 years 11 months after the last pregnancy with a recurrence of rheumatic carditis and cardiac failure.
6. died from lobar pneumonia 6 years and 5 months following her gestation.

Three of the patients in the present series had haemoptysis; one during the pregnancy, one both during the pregnancy and following it, and one since the pregnancy. There were two patients with auricular fibrillation, in one this complication had appeared since the pregnancy.

TABLE XXXII.

The Functional Heart Grouping of the present follow-up series at follow-up examination.

Group	No.	Per cent.
I	23	38.3
II	21	35.0
III	11	18.3
IV	5	8.3

TABLE XXXIII.

The Functional Heart Group during their pregnancy of the follow-up series.

Group	No.	per cent.
I	24	40
II	22	36.6
III	10	16.6
IV	4	6.6

TABLE XXXIV.

A comparison between the functional heart group of the patients during pregnancy and follow-up.

FOLLOW UP

Pregnancy Group	No.	Group I No.	Group I per cent	Group II No.	Group II per cent	Group III No.	Group III per cent	Group IV No.	Group IV per cent	Av. no.of years
I	24	22	91.7	2	8.3					3.6
II	22	1	4.5	15	68.3	4	18.1	2	9.1	3.6
III	10	0		3	30	6	60	1	10	3.1
IV	4	0		1	25	1	25	2	50	4
		23		21		11		5		

From the above chart it was seen that of patients In group I during pregnancy, 91.7 per cent were found to be still in this group at the follow-up, while 8.3 per cent changed to group II. Of the pregnancy group II patient 72.8 per cent maintained their group or improved so that 27.2 per cent showed a deterioration in their condition and one patient died.

Jones (1944) reported that over 90 per cent of his group I cases (i.e. those of slight severity without definite impairment of exercise tolerance before pregnancy, and only slight radiological cardiac enlargement) there was no appreciable deterioration

in exercise tolerance immediately following the pregnancy. There is therefore no reason to believe that the pregnancy will materially affect the subsequent course of the disease in these cases. In Jones' group II cases (i.e. those of moderate severity, with impairment of exercise tolerance before pregnancy, or moderate cardiac enlargement, or pulmonary congestion, but no heart failure at any time, and regular rhythm), about one-third of his patients were appreciably less well following the pregnancy, though in some cases this deterioration may be only temporary he thought.

Of the pregnancy group III in this series 30 per cent were improved and 10 per cent (1 patient) died. One of the 4 group IV patients died and two others shewed improvement. In Jones' group III patients (i.e. his most severely affected group) two patients died and in all his remaining traced cases pregnancy led to deterioration in their cardiac condition.

TABLE XXXV.

The relationship of Age of the patient to functional heart group at Follow-up.

	AGE									
	21-25		26-30		31-35		36-40		over 40	
Group	No.	per cent	No.	per cent	No.	per cent	No.	per cent	No.	per cent
I	3	75	9	42.8	6	30	4	40	1	20
II	1	25	8	38.1	10	50	1	10	1	20
III	0		3	14.3	15	15	4	40	1	20
IV	0		1	4.8	5	5	1	10	2	40
Totals	4	100	21	100	20	100	10	100	5	100

It can be seen from the above table that the percentage of patients in groups III and IV is greater as age increases.

In patients above 35 years of age there is a steep rise in the numbers in groups III and IV. While in the 25 years and below groups there were none in group III or IV. This may be considered consistent with the progressive course of rheumatic heart disease.

TABLE XXXVI.

Parity in relation to follow-up group.

	Parity									
	1		2		3		4		over 4	
Group	No.	per cent	No.	per cent	No.	per cent	No.	per cent	No.	per cent
I	18	50	3	20	2	50	0	0	0	0
II	13	36.1	6	40	0	0	1	100	1	25
III	2	5.5	6	40	2	50	0	0	1	25
IV	3	8.3	0	0	0	0	0	0	2	50
Totals	36		15		4		1		4	

It is not possible to say from the above table that parity has any relationship to the group of the patient.

TABLE XXXVII.

Parity of the patient in relation to change of Group during pregnancy. Present series.

Gravid.	No change		change	
	No.	per cent	No.	percent
1	24	63.2	14	36.8
2	8	57.1	6	42.9
3	1	25	3	75
4 & over	2	50	2	50

The change in condition during pregnancy would seem to fall heaviest on patients gravid 3.

It would seem that the patient during her first pregnancy has a distinctly less chance of changing her group to a worse one during pregnancy: but the risk is appreciable and is much the same for her during her second pregnancy. During her third pregnancy there is a markedly higher risk of her condition deteriorating. The fact that this is not so marked with the fourth pregnancy, although distinctly greater than the first and second, may be due to the fact that only the patients in good condition as Gilchrist says (1931) continue to have children.

Age at death.

The question arises does pregnancy tend to shorten the span of life in women with Rheumatic heart disease.

Gilchrist and Murray Lyon (1933) studying a series of 40 males, 28 nulliparous women and 41 parous women found, after eliminating deaths before a marriageable age had been reached, that the average age at death was as follows - Males 39.3 years, nulliparae 42.1 years and parous women 42.0. They were unable to establish therefore that nulliparous women live longer than those who have borne children.

Boyer and Nadas (1944) in their investigation found the average age at death of women who had borne children to be higher than in nulliparous women. Their figures were:-

TABLE XXXVIII.

		No. of cases	Average age at death.
18 years of age or over	Parous	103	43.7
	Nulliparous	49	39.0

It is true as Gilchrist and Murray Lyon say (1933) that it is the patients with the less severe cardiac lesion who are selected for marriage, and Boyer and Nadas further found that of the Nulliparous patients who died in 37 per cent of them death occurred between the ages of 18 years and 29 years.

Considering therefore only women who had reached 40 years - in order to eliminate severe illness or the severe rheumatic heart patients - Boyer and Nadas (1944) found the ages at death of each group almost identical.

TABLE XXXIV.

		No. of cases.	Average age at death.
40 years of age or over	Parous	68	51.5
	Nulliparous	22	49.7

Boyer and Nadas discussing the influence of multiple pregnancies on the age at death give the following comparative tables.

TABLE XXXV.

		No. of cases	Average age at death
18 years or older	Nulliparous	49	39.0
	Para IV or more	46	46.2
	Males	137	39.8
40 years or older	Nulliparous	22	49.7
	Para IV or more	34	49.8
	Males	66	50.1

This may shew that a patient who is fit to withstand pregnancy may bear several children without decreasing the expectation of life.

It cannot be doubted but that the heavy household duties of women who have borne more children should not result in as arduous a life as the nulliparous woman enjoys. It would seem reasonable to suppose, therefore, that it is the severity of the disease and its effect on the heart which is the decisive factor in the length of life of the patient with rheumatic heart disease.

This, of course, does not mean that pregnancy does not carry a definite immediate risk, but Boyer and Nadas (1944) ask if the patients who died from congestive heart failure during pregnancy would shew, had they lived any tendency towards delayed unfavourable effects. It can be shewn from the present investigations that patients who change their groups during pregnancy tend to remain in a less favourable group as compared with patients who did not fail during pregnancy.

Relation of change of group during pregnancy to distant prognosis.

TABLE XXXVI.

Patients who changed their group to a less favourable one during pregnancy, their condition at the follow-up in relation to their pre-pregnancy group was assessed.

		per cent	Average no. of years since preg.
No. of patients in same group as pre-pregnancy.	13	52.0	2.6
No. of patients in a worse group than pre-pregnancy.	12	48.0	3.7

TABLE XXXVII.

Of patients who did not change their group during pregnancy - condition at the follow-up in relation to their pre-pregnancy group.

		per cent	Average no. of years since preg.
No. of patients in same group as pre-pregnancy.	27	77.1	3.0
No. of patients in a worse group than pre-pregnancy.	8	22.8	4.1

The fact that a patient changed her group during pregnancy suggests that pregnancy has caused an increased strain; and the adverse effect on the heart would seem to have a sequel in the follow-up investigation, which shows a higher percentage of patients in a less favourable group - compared with patients who did not change their group during pregnancy.

Does the length of labour influence the follow-up group of the patient. Present series.

TABLE XXXVIII.

Average length of labour in patients who were in a worse group at follow-up as compared with their pregnancy group	11.00 hrs.
Average length of labour in patients who were in the same group at follow-up as compared with their pregnancy group	12.45 hrs.

It would appear, therefore, that the change of condition into a less favourable group at follow-up as compared with their pregnancy group is not conditioned by the duration of labour.

The duration of the rheumatic disease has an obvious bearing on the condition of the heart, but Gilchrist and Murray-Lyon (1933) did not find any significant difference in this between multiparous and nulliparous. With the high percentage of deaths in nulliparae under thirty years of age (page 237) it would seem that it is the severity of the action of the rheumatic disease on the heart which is of more serious prognostic import: and it is probably the case that as the slightest over-strain or infection form the final precipitating cause in nulliparae, so it is that pregnancy may be just incidental or take the place of over-strain in women who will succumb at this time because of the rapid downhill progress of the rheumatic disease.

Scott and Henderson (1934) take the view that the deaths at an early age are due to the rheumatic disease, per se, but the immediate mortality in this series of cases, 3.1 per cent, included some patients with well compensated hearts who, although they might have withstood the strain of normal labour, showed that they could not survive the stress of a sudden or a prolonged strain, which they would not have experienced had they not become pregnant.

The expectation of life of the patient in the presence of a debilitating heart lesion must also depend on whether she works within the limit of causing undue strain on her heart. The outlook in the case of the mother, who has a heavy engagement of household duties to contend with single handed, must be very different from that of the woman with a similar cardiac lesion but with someone to help lighten the burden of the daily task. This aspect of after-care of the patient must loom large in social medicine and sufficient help be provided, as it already is in some centres, by Local Councils to remove the financial difficulty in securing the benefits of this aid. The Home Help Scheme, inaugurated at Oxford by a voluntary organisation during the war, has done much good work of this nature and has paved the way for the State to take over this essential duty of care of the sick. Health visitors are a further check on the progress of the patient, the extent and nature of her daily work, and her observance of medical instruction and attendance at a Cardiac Clinic, and they are a constant source of sympathetic aid in the patients troubles.

THE FUNCTIONAL HEART AND PREGNANCY.

From time to time, at the antenatal clinic, is seen the patient with many of the symptoms of heart disease, but in whom no cardiac lesion can be discovered. She is anxious, breathless and has submammary pain and occasionally a thumping in the region of the heart, following a missed beat; usually the anxiety and marked breathlessness, which she displays even at rest stand out in such strong distinction to the lack of physical signs in the heart, that a careful enquiry into her medical history is immediately sought. There is usually a story of mental anxiety for some reason, associated either with the fear of pregnancy or some accident in a previous one, or there is temperamental instability of some kind. The typical case is one, whose previous pregnancy had been a painful, unrelieved labour ending in a difficult forceps delivery and still-birth and the patient is now in fear of the outcome in the present confinement.

These patients reveal in an exaggerated fashion the effort syndrome, described by Jones (1944), of left submammary pain, breathlessness and palpitations, and difficulty in breathing which sometimes starts with the patient at rest, particularly if she is

startled or worried: such dyspnoea, he states, varies from day to day, depending on the patient's mental state - whereas the dyspnoea of heart disease begins during exertion and is relieved by rest. The submammary pain, too, tends to be more troublesome when the patient's mind is unoccupied and to disappear when she has an interesting task to perform. Jones (1944) adds that such pain is not likely to be confused with true anginal pain, which is exceedingly rare in pregnancy and is always related to exertion and relieved by rest: and, he concludes, as regards the irregularity of the pulse, it is more likely, in those patients with sinus arrhythmia, or premature beats, which usually disappear with effort, whereas the irregularity of an auricular fibrillation becomes more obvious on exertion.

Treatment by sedatives, a period of rest, and full reassurance on the conduct and outcome of the pregnancy are, as a rule, successful in alleviating the anxiety and the dyspnoea.

TOXAEMIA OF PREGNANCY IN ASSOCIATION WITH HEART DISEASE.

Toxaemia of pregnancy is stated by some authors to be more common in heart disease than in normal patients. Corwin (1927) found an incidence of 19.4 per cent in his cardiac group as compared with 6 per cent in the non-cardiac patients. Reis and Frankenthal (1935), in their series, found that 24.5 per cent of patients with heart disease shewed definite evidence of toxaemia when their general clinic incidence was 7 per cent. The toxaemia rate in the present series was 6.6 per cent against the general rate of 4.4 per cent for all patients over the same period.

The treatment of the toxaemia is the same as for patients without heart disease and if termination of the pregnancy is indicated as a treatment of the toxaemia then it should be done. Labour is induced by artificial rupture of the membranes if the cervix is soft and partly effaced admitting a finger its state in most parous women and an early start to labour anticipated. In other instances when the cervix is firm and long, closed and not taken up the risk of a delay may be deleterious to both the cardiac condition and to a progressing toxaemia, and a Caesarean section should be considered.

In the 15 cases of this series, however, rupture of the membranes was undertaken in 7 instances without untoward result for the patient, and the others went into spontaneous or medically induced labour.

OTHER HEART LESIONS.

SUB-ACUTE BACTERIAL ENDOCARDITIS.

Subacute endocarditis is a rare complication of pregnancy and only nineteen authenticated cases have been reported in the literature. Until the introduction of penicillin, subacute endocarditis was always looked upon as a fatal disease and, occurring during pregnancy, the prognosis was so hopeless that all the patients died by the 6th post-partum month.

The patient shews the cardinal signs of the disease, the damaged heart, emboli to various parts of the body, often seen in the terminal vessels of the fingers and revealed as settling in the kidney by the presence of blood in the urine; the occasional spike of temperature and blood culture positive for the streptococcus viridans. It is not easy to diagnose the condition, since it is so rare and unexpected and the signs often difficult to find, without often prolonged and repeated observation and examination. The patient's listlessness and breathlessness and severe anaemia serve to indicate that something more than the heart lesion itself is present, and her urgent admission to hospital for blood examinations at the time of maximal temperature may lead to

success in making a diagnosis. Typical of the condition are also anorexia, night sweats, headaches, mental slowing, insomnia, loss of weight pain in the renal region from embolic infarction and a cafe-au-lait appearance of the skin.

The use of penicillin for this fell disease, has brought a more hopeful outlook for the patient. This is especially true if it is found that the infesting organism is penicillin sensitive to a high degree. In a recent case 2016/47 (outside the limits of this series) a patient aged 32 Gravid 4, with a mitral stenosis developed an irregular pyrexia and pain in the right renal angle at the 32nd week of pregnancy with some blood and a moderate growth of B. coli in the urine. The temperature did not subside under the usual treatment for pyelitis nor with lavage of the kidney pelves but at the time the condition was considered to be a pyelitis of pregnancy, which was resistant to the ordinary treatment and was therefore treated by rupture of the membranes at the 36th week to terminate the pregnancy and secure better drainage for the kidneys. The irregular pyrexia persisted into the puerperium when a blood culture was taken to reveal the streptococcus viridans. Blood culture repeated the following day was found to be similar. This patient had some septic teeth removed at the

31st week of pregnancy a few days before her renal pain, which was later considered to be due to emboli, since too, there was recurrence of a similar pain in the other kidney with blood cells, but no organisms in the urine just before penicillin treatment was instituted. The organism proved highly penicillin sensitive in vitro and the patient responded well to one million units penicillin a day, 125,000 units 3 hourly for one month, the temperature settling after a few days and remaining normal, except for a brief interval of three days when she had pains in all her joints and sweating, which subsided with salicylates the blood culture being sterile. No positive blood culture was obtained after the 2nd day of treatment. The penicillin was stopped after a month and the patient allowed home one week later. She was slightly breathless with effort but was improving. Seen 4 months later there was no evidence of recurrence and no further treatment had been given and the dyspnoea was less.

An additional case of subacute bacterial endocarditis complicated by pregnancy, not included in Table XXXIX , is reported by Dobson (1946). The patient aged 24, Gravid II was 12 weeks pregnant when she reported with a history of rheumatic endocarditis, Temperature 100° F, Pulse 100, Hg 61 per

cent, backache, emboli in Rt. foot: and the streptococcus viridans was recovered from the blood stream, and found to have good penicillin sensitivity. She was treated with 200,000 Oxford units of penicillin daily and the blood cultures were sterile after 60 hours. After 5 weeks of penicillin treatment her progress was not considered satisfactory and she continued to have new petechiae although the blood cultures remained negative. Compared with other cases of subacute endocarditis with a similarly sensitive strain of streptococcus viridans treated at the author's clinic her response was not thought to be favourable and as the only added feature about this case was the pregnancy this was considered to be responsible. Hysterectomy, because of several associated uterine fibroids, was performed removing an 18 week pregnancy. The patient's response to this was good and penicillin treatment was stopped 2½ weeks after operation. (Foetal cultures were negative). Patient was discharged nine days after penicillin had been discontinued. Her cardiac reserve was good and she was considered to have recovered from subacute bacterial endocarditis. The author asks how much her improvement can be considered to have been due to the termination of the pregnancy.

Another interesting feature reported by Dobson's case was the group of new petechiae which were

noticed five days after discontinuation of the penicillin and the patient's temperature was 99.8° F. sedimentation rates remained normal and repeated blood cultures were negative and the condition was taken to be a "sterile episode."

As regards the obstetrical treatment it may be considered that if the patients response to penicillin were good the pregnancy be treated according to the reserve of the heart muscle and the patient's functional group as defined by the American Heart Association classification. Unsatisfactory response to penicillin, in the presence of an organism sensitive to it in vitro may suggest, in the light of Dobson's (1946) case, that the pregnancy should be terminated.

Since recurrence of subacute endocarditis is possible some months after treatment and since, too, deterioration of the heart condition may occur and prove fatal, the prognosis as to the future must be guarded.

THE OBSTETRIC ASPECTS OF ALL REPORTED CASES OF STREPTOCOCCUS VIRIDANS ENDOCARDITIS IN PREGNANCY. TABLE XXXIX.

AUTHOR	AGE	PARA	PAST HEART HISTORY.	ONSET OF SYMPTOMS	TIME OF DELIVERY.	TIME OF DEATH POST PARTUM	PREGNANCY TERMINATED BY	STR. VIRIDANS RECOVERED IN	FOETUS
Freund(1913)	30	2	Rheumatic heart disease	1 month	unde-livered	Ante partum	Undelivered	Heart blood at autopsy	Non-viable.
Findley(1921)	30	2	Not stated	"Early"	4 months	3 weeks	vaginal hysterotomy	maternal blood	Non-viable.
Walser(1928)	23	1	chorea and rheumatic fever	6 months	8½ months	1 month	spont. labour and delivery.	maternal blood foetal blood	living, culture negative in 1 week
Walser(1928)	24	0	not stated	6 months	7½ months	"several months"	spont. labour & delivery	maternal blood foetal blood	died after 17 hours
Kobacker(1930)	18	0	rheumatic heart disease	3 months	9 months	4 days	Caesarean sections on moribund patient	maternal blood foetal blood	living, culture negative in 2 weeks
Reid (1930)	?	5	rheumatic heart disease	?	5 months	5 months	spontaneous abortion	maternal blood	Non-viable
Mengert (1933)	21	0	"rheumatism"	6 months	8 months	6 months	spontaneous labour breech delivery	maternal blood	living
Mengert (1933)	27	4	negative	8 months	9 months	7 weeks	spontaneous labour breech delivery	maternal blood	living
Terwilliger(1934)	21	1	negative	7½ months	8½ months	not stated	spontaneous labour and delivery	maternal blood and urine	living, negative cultures
Bradford (1934)	24	1	"heart disease"	7 months	9 months	4 weeks	spontaneous labour and delivery	maternal blood	living
Lieberman (1934)	22	0	"rheumatism"	post partum	8 months	6 weeks	spontaneous labour and delivery	maternal blood	living
MacRae (1937)	27	0	negative	6 months	unde-livered	Ante partum	undelivered	maternal blood	undelivered neg. cultures at autopsy
MacRae (1937)	27	4	chorea	"near term"	"near term"	day of delivery	Caesarean section for Abruptio Placentae	heart valves at autopsy	Still-born (died from abruptio-placentae)
Felsen et al. (1937)	25	?	negative	7½ months	9 months	16 days	spontaneous labour and delivery	maternal blood throat, urine breast milk	living, negative cultures
Jensen (1938)	27	2	rheumatic fever	7 months	8 months	2 months	spontaneous labour and delivery	heart valves at autopsy	living
Page & Campbell (1939)	35	2	rheumatic heart disease	5 months	6 months	1 week	spontaneous labour and delivery	maternal blood	died in 1 hour
Page & Campbell (1939)	30	2	rheumatic heart disease	2 months	3 months	day of abortion	spontaneous abortion	maternal blood	non-viable
Page & Campbell (1939)	20	0	negative	7 months	8 months	1 month	spontaneous labour and delivery	maternal blood	living, cultures negative
Niehaus (1939)	30	?	scarlet fever with endo-carditis.	7 months	8 months	11 days	spontaneous labour and delivery	maternal blood	living

THE HYPERTENSIVE HEART.

Hypertensive heart disease of a severe degree may antedate pregnancy and already have encroached on the cardiac reserve. With the added burden of pregnancy the hypertensive disease may worsen and precipitate heart failure. It is however in the patient past childbearing age that hypertension is found to be of the malignant type with severe heart damage and it is rare to find it complicating the heart of the pregnant woman. In those chronic illnesses there appears to be, too, a high infertility rate. Sodeman (1940) states that pregnancy confers an explosive character, the etiology of which is obscure, upon the hypertensive heart. The results of work on animals, he further states, may have a bearing on this phenomenon. This work was done by Dill and Erickson (1938), who found that dogs, which had been made hypertensive by Goldblatt method, shewed a marked exacerbation of the process when they became pregnant. Whereas the control animals shewed only slight lesions, the pregnant animals developed widespread hepatic lesions similar to those of eclampsia, haemorrhage and infarction of the myocardium and evidence of terminal acute cardiac failure and pulmonary oedema.

Hamilton and Carr (1933) in a series of 500

cases found 14 patients whose systolic blood pressure was over 140 systolic. Bramwell and Longson (1938) state that hypertension in pregnant women is usually secondary either to a toxaemia of pregnancy or to a lesion in the aortic valve.

It is a rare thing to find a patient with hypertension, who shews any signs of cardiac insufficiency despite the fact that the blood pressure is high enough to be considered malignant. In the present investigation no heart lesion attributable to hypertension was found.

HYPERTHYROIDISM AND PREGNANCY.

Hyperthyroidism is a rare complication of pregnancy. Kibel (1944) reports an incidence of 0.075 per cent. Hamilton and Thomson (1941) state that in a series of 50,000 deliveries a diagnosis of hyperthyroidism was reasonably established in only 28 cases.

There is a lowered fertility rate in thyrotoxicosis, but this is fortunate since the condition places a strain on the heart and circulation and may cause auricular fibrillation. Bramwell and Longson (1938) say that in all forms of hyperthyroidism the heart is overactive, its beats unduly forcible, its rate quickened and made more susceptible to emotional and other disturbances. They record a series of 350 patients, in whom 5 had heart disease attributable to thyrotoxicosis and in all the patients, with one exception, the thyrotoxic manifestations increased during the pregnancy: and they add that those patients are very liable to abort at the times corresponding to the suppressed menstrual period and should be kept in bed for a few days at this time. Naish, (1937) on the other hand, found that only one out of the thirteen patients she reported with thyrotoxicosis during pregnancy experienced any cardiac decompensation: and Jensen (1938) states that the cases of

hyperthyroidism, which he has seen associated with pregnancy, did not present any important cardiac features.

Kibel (1944) records seven cases, in which therapeutic abortion was performed in two cases, 16.6 per cent and toxaemia of pregnancy occurred in 55.5 per cent of patients reaching the 28th week of pregnancy. A subtotal thyroidectomy was done in 33.3 per cent of Kibel's cases and, he states, if any operative treatment is being considered, it is the thyrotoxicosis not the pregnancy which should be interrupted.

Clute and Daniels (1930) state that thyroidectomy for primary hyperthyroidism can be undertaken during pregnancy with safety to both mother and child.

Hamilton and Thomson (1941) say that the occasional association of hyperthyroidism and pregnancy has been frequent enough to establish the following well known rules, to which they heartily ascribe:-

1. "If hyperthyroidism occurs in pregnant women the hyperthyroidism should be treated surgically as usual, without waiting for the pregnancy to terminate:
2. The interruption of pregnancy because of hyperthyroidism is not indicated if there are dangerous symptoms of thyroid toxicity. The

sicker the patient the more dangerous it would be to attempt to aid the patient by therapeutic abortion."

Hamilton and Thomson (1941) admit however that surgical clinics have reported a higher percentage of operative treatment among their patients who had hyperthyroidism complicated by pregnancy than is found if the patient has stayed at an obstetrical clinic - the latter group being satisfactorily treated by physicians.

Patients seen apart from the present series of heart lesions have proceeded to term without accident and it would seem that where mild thyrotoxicosis is present the patient can be kept under strict medical supervision and the condition controlled, allowing the pregnancy to go to term. In severe thyrotoxicosis, however, including cases in which auricular fibrillation is present, full medical treatment should be instituted immediately. In the early months of pregnancy it would, therefore, be advisable to empty the uterus in order to avoid the further emotional strain which pregnancy may bring and then, if indicated, a subtotal thyroidectomy can be performed with greater chance of restoring any abnormal cardiac rhythm, which may be present.

THE ARRHYTHMIAS.

PAROXYSMAL TACHYCARDIA.

True paroxysmal tachycardia Jensen (1938) states has certain characteristics without which the diagnosis should not be made; and only 19 cases, occurring in pregnant women, have been recorded, in which the diagnosis was beyond reasonable doubt. In eight cases the attacks occurred in women with valvular disease, and in only three of those the attacks had been present before pregnancy; and in all these cases the attacks did not interfere with cardiac compensation, except in one case reported by Hamilton (1928), which ended in fatal congestive failure. In the eleven patients with otherwise normal hearts, Jensen (1938) states that five had suffered attacks before becoming pregnant. Two of these patients died, suggesting therefore, that the condition cannot be ignored as held by Eastman (1933).

Weyler and Duslin (1942) reporting the case of a patient, who had runs of tachycardia in four pregnancies, state that the arrhythmia is due to ectopic beats, which vary in their point of origin, some definitely auricular and arising near the sino-auricular node, others followed by a ventricular complex wholly unlike the normal. They state that the

irregularity in no way interfered with the patient's normal activity during her pregnancies. Their attempts to control the tachycardia was unsuccessful and digitalis had no affect on its rate or rhythm. An E.C.G., made six weeks after the last delivery, shewed no runs of tachycardia in their case.

Borwning and Clark (1941) report two cases of paroxysmal auricular tachycardia complicating pregnancy and state that this complication occurs in less than 1 per cent of known cardiacs and much less frequently in otherwise normal persons. Perfectly regular rhythm differentiates it from paroxysmal ventricular tachycardia, auricular flutter and fibrillation. Quinidine, they state, is an effective drug in stopping attacks and may be used in pregnancy without fear of precipitating premature labour.

Bramwell and Longson (1938) state that paroxysmal tachycardia can be distinguished from simple nervous tachycardia by the abrupt onset and termination of the attacks, within a single heart cycle, and for no apparent cause changing from 70 to 200 beats per minute. If tachycardia be prolonged, dilatation of the heart and signs of congestive failure may appear, to clear up with restoration of the normal rhythm. Vagal stimulation by direct pressure on the carotid

sinus of the neck, or, Campbell (1926) suggests, by forced deep inspiration may abort or shorten an attack. Gilchrist (1931) says that paroxysmal tachycardia, especially if accompanied by precordial pain and exhaustion, may in itself be an indication for the interruption of pregnancy.

Three patients with paroxysmal tachycardia were found in the present series of which one was noted as being functional. There are no electrocardiographic records available, of the cases none of which shewed any decompensation during her pregnancy.

HEART BLOCK.

Jensen (1938) states that, since only about 10 per cent of cases of heart block occur in young persons, it is rare during pregnancy and only 17 cases have been described in the literature. The etiology of the cases described was obscure, four cases being associated with rheumatic lesions, one with syphilis and the others variously attributed to infections or, as Jensen (1938) says, considered congenital lesions without convincing evidence: in four patients the heart block was partical. Since a complete heart block, however, is compatible with efficient circulation, even in the presence of pregnancy Jensen (1938) holds that it should not constitute a contra-indication to child bearing.

Greenhill (1933), on the other hand, comments that heart block is rare and serious, and of the 12 cases reported in the literature, including a case of his own, there were two fatalities, a death rate of 17 per cent: and he further states that it is not very uncommon to observe heart block during pregnancy, labour or the puerperium as a temporary condition, in women who do not have heart block at any other time. As regards treatment Greenhill (1933) says that in women with perfect compensation and without untoward symptoms, delivery through the natural passages should yield good results for

mother and child; the second stage of labour should, however, be shortened in primigravida by means of low forceps under direct infiltration anaesthesia of the perineum and vagina. In women with evidence of decompensation, however, Greenhill (1933) considers a lower segment caesarean section under local the safest procedure.

Two patients were seen in the present series - one a primigravid, aged 26, with a history of rheumatic fever 4 years previously. Her pulse rate was 32 and she was breathless and tired easily on exertion. Labour was assisted by forceps in the second stage, because the patient became distressed. The pulse during labour varied from 32 to 40 in the second stage. During her stay in hospital the pulse range was 32-44, but both she and her baby were discharged well.

The other patient, Gravid 2 aet.24, first pregnancy, low forceps, no trouble with confinement. Previous history of heart trouble since rheumatic fever at age of 16 years. Jones (1944) reported on her electrocardiogram, which shewed a complete heart block and he considered it possible for the condition to have arisen from the rheumatic attack although, he added, it is perhaps more probable that it is of congenital origin. She also had a persistent

leucocyte count of 15,000 to 20,000 with a normal differential count. Present pregnancy was normal, without any undue breathlessness; labour lasted eleven hours and ended spontaneously the pulse being 40, and the puerperium was normal.

CONGENITAL HEART DISEASE.

The incidence of congenital heart disease is given by Pardee and Mendelson (1941) to be 1.89, by Abbott (1936) as 1.2 per cent, Bramwell and Longson (1938) 4 per cent, and McIlroy and Rendel (1931) 3 per cent of all organic heart disease cases. In the present series of 225 cases, the condition was found in 13 patients, a percentage of 5.8.

The most serious of these lesions is congenital coarctation of the aorta and a patient with this lesion is advised against pregnancy. Should she become pregnant, however, therapeutic abortion should be procured, and if seen later in pregnancy, Caesarean section is advisable in order to avoid the strain of labour which may cause a sudden catastrophe such as rupture of the aorta or cerebral haemorrhage. There is less risk to the patients with other types of lesion, but Mendelson and Pardee (1941) state that widely patent foramen ovale, interventricular septal defects and patent ductus arteriosis all form a special category by virtue of veno-arterial shunt possibility, although this is a rare accident and may be prevented by avoiding strain during labour and by the early use of forceps during the second stage of labour.

Owing to the difficulty in diagnosing with exactness the type of cardiac lesion, the generic term "congenital" has been applied to some of the cases: it may be possible, too, that in some of the cases the term "congenital" may have been applied as being nearest to the diagnosis. If these cases, therefore, were excluded, the corrected number would be 7, or 3.1 per cent of all cardiac cases in this series with a maternal mortality - 0 per cent and foetal mortality - 0 per cent.

TABLE OF CONGENITAL HEART DISEASE AND PREGNANCY IN PRESENT SERIES.

Age	Gravid	Mat.	Lesion	Group	Labour	Mother	Child	Remarks
29	2	40	"Congenital"	2	Spontaneous delivery	A	A	
29	2	40	"	1	" "	A	A	
28	2	38	"	1	" "	A	A	
24	1	40	"	1	" "	A	A	
39	2	42	Patent Interventricular septum	1	" "	A	A	
21	1	34	"Congenital"	4	Caesarean Section	A	A D	Also pulm. TB and Twins
27	4	40	"	2	Spontaneous Breech del.	A	A	
24	1	40	Congenital pulm. stenosis	2	Forceps	A	A	
24	1	38	Patent interventricular septum	4	Spontaneous delivery	A	A	
33	3	40	Coarctation of the Aorta	1	" "	A	A	
25	2	38	Patent Ductus Arteriosus	2	" "	A	A	
25	1	38	Inter-auricular septal defect	1	Caesarean Section	A	A	
31	2	40	Inter-auricular septal defect	1	Spontaneous delivery	A	A	

MATERNAL DEATHS - 0. FOETAL DEATHS - 1.

PATENT DUCTUS ARTERIOSUS

One patient, aged 25, gravid 2, with this lesion is reported in the present series. She gave a history of being under the care of a heart hospital in childhood and of attending a special school until the age of 17 years; there was no history of an infective condition.

Obstetrical history shewed that the patient had been admitted to hospital two weeks before full term with her previous pregnancy, and rested in bed for two months after parturition.

During the present pregnancy, there was no cyanosis or dyspnoea, and the pulse was normal in rate and rhythm. She was uncertain about the date of the last menstrual period, but the membranes were ruptured for a lateral placenta praevia and she had a spontaneous labour with a live child weighing 6 lb. 9 oz., the first stage of labour lasting 1 hour 10 minutes, and the second for 10 minutes. The pulse remained at 80 and no breathlessness or oedema were noted, and the patient was discharged on the 14th day. No tachycardia or dyspnoea, and only occasional palpitations were found at the post-natal examination.

There are 33 other cases of patent ductus arteriosus complicating pregnancy reported in the literature, including two by Jensen (1938) who quotes quotes Couderc (1912), who states that the average age of the patient is 27.2 years, and the average parity 2.2. Six deaths are recorded, only two of which, Pardee and Mendelson (1941) say, can be attributed to the lesion; one, they state reported by Schulez (1928), dying of congestive failure at the 5th month of pregnancy and the other, reported by Tunis (1929), dying from congestive failure following Caesarean section at term. The other patients, like the one recorded above, had a normal pregnancy without mishap.

PULMONARY STENOSIS.

One patient with congenital pulmonary stenosis was seen in the series.

Jensen (1938) states that this disease is rare in pregnancy, since if at all marked it causes cyanosis and the patient does not reach maturity. It is commonly combined with other heart lesions such as dextraposition of the aorta, patent interventricular septum and right ventricular hypertrophy providing the so-called tetralogy of Fallot. Jensen (1938) states that the average age of the patient is 22.9 years and the average parity 1.9.

The patient in this series was aged 24, gravid 1 - had nothing abnormal apart from the heart lesion and enjoyed a normal pregnancy and labour. Labour was spontaneous at term and lasted in the first stage for 6 hours and in the second stage for 1 hour 10 minutes and a live child, weighing 8 lb. 14 oz., was born: the patient's pulse was 76 throughout, and she was discharged on the 10th day after a normal puerperium.

Another case noted under "conginetal disease" was thought to be either a pulmonary stenosis or a simple septal defect. A primigravid aged 21, with bilateral active pulmonary tuberculosis: there was also a high malar flush and cyanosis of lips and

fingers, with the pulse 110. A Caesarean section was performed at the 35th week of pregnancy and twins were delivered, one of whom died in the puerperium. Patient was discharged for further treatment of the tuberculosis.

Jensen (1938) reports that no deaths were attributable to pulmonary stenosis associated with pregnancy, but there was one fatal case with tuberculosis as a cause of death.

PATENT INTERAURICULAR SEPTUM.

Nineteen cases are reported of patent foramen ovale. In eight there was no trouble, and one had a therapeutic abortion; but Jensen (1938) states that there are ten recorded deaths, of which six can be attributed to the lesion: the average age is stated to be 32.5 years and the average parity 6.6.

Two cases were seen in the present series: one a patient aged 31, gravid 2, who went through pregnancy and labour without shewing any abnormal signs apart from the heart lesion; she had a normal spontaneous delivery of an 8 lb. 8 oz., baby at term, after a labour lasting 7 hours 20 minutes; pulse 80, regular, no enlargement of the heart and the puerperium normal. The previous pregnancy was also normal in every way. The other was a patient, Gravid 1, aged 25, with Lutenbacker's syndrome and in Group III at ten weeks. She was admitted for rest during pregnancy because of breathlessness, cyanosis and oedema of the legs. Her lungs were clear but her liver at one stage was felt just below the costal margin; her pulse was 80-90, irregular from multiple premature contractions. The cyanosis cleared with rest and the liver returned to normal size. A Caesarean section and sterilisation were performed at the 38th week, and her convalescence was normal, with pulse rate of 80.

LUTEMBACHER

PATENT INTERVENTRICULAR SEPTUM.

There are 36 cases in the literature reporting the complication of interventricular septal defect and pregnancy. Jensen (1938) found the average age to be 28.5 years and the average parity 2.2; and two of the recorded deaths were due to congestive heart failure at the sixth month of pregnancy. Of the four cases reported by Pardee and Mendelson (1941) none had cyanosis or polycythemia and their care did not present any problems.

In the present series, there were two cases: one a patient aged 24, gravid 1, in a severe degree of decompensation (group IV) who went into spontaneous labour at the 38th week of pregnancy and delivered herself normally of a live child. The other case was that of a patient, aged 39, gravid 2, who shewed no decompensation throughout pregnancy or labour (group I), and whose previous pregnancy and labour were also normal. The condition is one, therefore, which may give rise to serious decompensation of the heart.

COARCTATION OF THE AORTA.

Coarctation of the aorta has frequently caused such sudden and dramatic fatality apart from pregnancy that it is looked upon as a serious condition. Abbott (1936) states that it threatens individuals in the full flood of adolescence or in the prime of early middle life; and Mendelson (1940) asserts graphically that the sword of Damocles hangs constantly over the heads of these unfortunate patients. The danger is that the strain of pregnancy and labour with the increase in cardiac, superimposed on coarctation may produce too great a tension on the arterial system proximal to the stricture and cause rupture of the aorta or of the cerebral vessels.

Pregnancy and labour, therefore, states Mendelson (1940) present such definite risks to these patients that pregnancy should be avoided, or if the patient is seen in early pregnancy therapeutic abortion should be performed: while those presenting themselves for the first time late in pregnancy should avoid the strain of labour and have an elective Caesarean section. Sterilization is advisable if an abdominal operation is performed.

The three cases reported by Mendelson (1940) are of interest. All, he states, experienced dyspnoea about the 4th month of pregnancy and it

increased with the progress of gestation, except for slight but definite relief prior to term. In one case, elective Caesarean section at term was associated with the disappearance of dyspnoea within the first 24 hours. In the second case, there was a spontaneous delivery after a four-hour labour with the pulse and respirations at physiological levels: but postpartum the dyspnoea became more severe and the general condition markedly worse. Definite cardiac and vascular changes were indicated by the development of a diastolic murmur to the left of the sternum. The third case had a Caesarean section shortly after labour began, because the pulse had already risen to 120 and respiration to 30. With the use of digitalis and the avoidance of further strain, the patient's condition was stated to be satisfactory two days later.

Mendelson (1940) found 29 other cases reported in the literature of coarctation of the aorta complicating pregnancy: the average age was 34 and the average parity 3. Five deaths were attributable to the lesion, two from rupture of the aorta, one cerebral haemorrhage, one cardiac failure, and one endocarditis. At the same time, in over half the cases, he states, pregnancy and labour had a delecterious effect upon the condition and, he adds, a satisfactory

clinical condition, when the patient is first seen, does not preclude a sudden and fatal accident.

Kenny, Sylvester and Levine (1945) reported another case of coarctation of the aorta complicating pregnancy and added an additional 9 cases from the literature.

Walker (1943), too, reports a case, in which the patient went into labour and had a dramatic and alarming second stage with sudden acute breathlessness and distress, severe headache, pulse 128 and with expansile pulsation at the root of the neck: delivery was, however, affected with forceps without further accident. Commenting on the condition he thought Caesarean section would have been advisable if he had seen the patient before labour commenced.

Bramwell and Longson (1938), and also Strayhorn (1935), report cases with dyspnoea and cardiac decompensation during pregnancy successfully delivered by Caesarean section.

Baber and Daly (1947) describe a case which came under their care and was delivered by Caesarean section and add a further two cases both of which had normal vaginal deliveries. These observers are not so gloomy in their outlook regarding this disease as Mendelson (1940) and give it as their opinion, from a study of the literature, that

pregnancy is not contraindicated in the majority of these patients, though adequate spacing and avoidance of late childbearing, they add, seems a wise precaution.

A patient 1904/43, was seen in the present series and reported by the Cardiologist to be a coarctation of the aorta. The patient, aged 33, Gravid 4, had no rheumatic history. Previous pregnancies were : 1929, instrumental delivery, 7¾ lbs.; 1935, premature breech delivery 3 lbs., still-birth; and 1 miscarriage. Her general health was good and she was accustomed to a lot of walking without breathlessness.

The Cardiologist's (Dr. Jenner Hoskins) report was :-

"Pulse 84 regular: the radial arteries were thickened. B.P. in the arm was 150/90.

The oscillometer readings were :-

Pressure	Arms	Legs
175	5	0
150	15	0
125	25	2
100	25	15
75	15	5

There was visible pulsation in the intercostal arteries at the back, and no pulsation palpable in the femoral arteries.

The heart was enlarged to the left, the impulse strong and systolic murmurs were heard at the mitral and aortic areas.

The X-ray picture shows notching of the under-surface of the ribs and an absent aortic knob and is pathognomonic of coarctation of the aorta.

On screening, coarctation of the aorta is seen just beyond the aortic arch.

The E.C.G. (below) shews : Right axis deviation: rate 90, regular. Large notched S_1 and R_3 ; T_3 is inverted. Record shews a right bundle branch lesion."

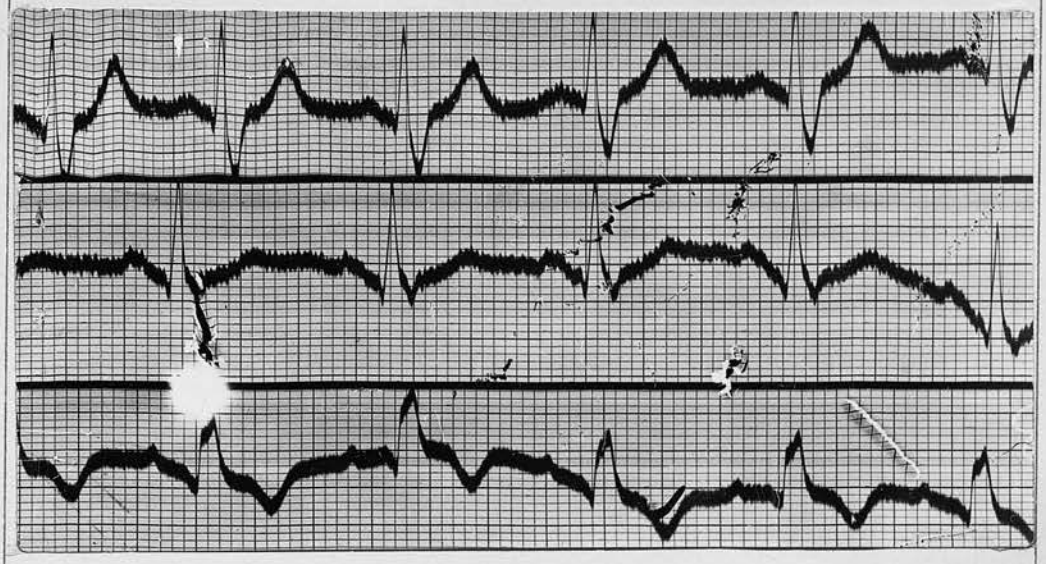
X-RAY REPORT ON CHEST.

Multiple rib notching present (Roester's sign) on both sides, better seen on right.

This is virtually pathognomonic of coarctation of aorta, but the aortic shadow is more prominent to the left than usual. The isthmic narrowing is probably more distal than the common site of co-arctation.

Minor left ventricular hypertrophy seen.

17 Rt Bundle Branch Block in
ELECTOCARDIOGRAPH OF COARCTATION OF THE AORTA.



She remained in Grade I throughout pregnancy, and labour at term was normal, the first stage lasting 13 hours and the second 10 minutes, with a live 6 lbs. 8 oz. child. The pulse remained steady in the region of 80 per minute, and the puerperium was uneventful.

It may finally be said that recent successful operative treatment for coarctation of the aorta, removing the stenosis, may ultimately change the outlook in this dread condition enabling the patient to go through life and pregnancy as a normal individual.

CORONARY OCCLUSION.

Coronary occlusion is a rare occurrence during pregnancy. White et al. (1937) consider, however, that it may not be so very rare even in the younger patients and record a case of acute myocardial infarction in a patient aged 22. The condition appeared as early as the 2nd month of pregnancy but settled down quickly to be followed by no harmful effects and the remainder of the pregnancy, labour, and the puerperium were normal. Electrocardiographic evidence is, they think essential, and recovery is the rule.

Reis and Frankenthal (1935) also describe a case of coronary occlusion which occurred in a patient aged 45, with marked hypertension and cardiac decompensation. With complete rest in bed the patient's condition improved and she was delivered vaginally at term without accident.

Routine electrocardiograms of patients who develop sudden decompensation of the heart may bring forth more frequent evidence of coronary thrombosis. It is of interest to reflect on the scarring of the heart muscle following this complication and of its power of recovery.

THE DISCUSSION

With the numerous dramatic and revolutionary advances of modern medicine we tend to lose sight of many gradual, less conspicuous but none the less significant changes, which are brought about rather by better understanding of the problems than by modern discoveries of new facts. Heart disease in pregnancy is a case in point. Until the latter part of the last century, an attitude of extreme pessimism persisted and pregnancy was considered a most dreaded complication of heart disease. This was due to failure to appreciate fully the heart's action and the warning signs of its distress, and to the fact that usually the patient was not seen until the pregnancy was far advanced or until labour had started, when as a rule, the heart was already in a severe state of decompensation. No antenatal care was given if the heart was compensated and, if decompensation was found, the situation was considered already so desperate that quick relief by prompt termination of the pregnancy seemed to be the only remedy, with results that were only too often disastrous.

This state of affairs is now greatly changed. We have learnt much about the manner in which pregnancy makes increasing demands on the cardiovascular

system, and the outstanding work of MacKenzie has served to place the treatment of these patients on a more rational basis; and the increasing antenatal care has enabled a close watch to be kept and prompt treatment to be initiated.

The incidence of heart disease in the series presented, was 225 (0.8 per cent) out of the 29,713 patients seen during the 10 year period 1937-1946. The incidence given by other authors has been higher but Fitzgerald (1935) considers that, unless checked by the patient's attendance at a cardiological clinic this rate is bound to vary, and quotes his own as 0.66 per cent, and Daley and Strouse 0.25 per cent, Fitzgibbon 0.16 per cent and Rees and Frankenthal 1.33 per cent: while Stander, as mentioned, finds the rate to be between 2 per cent and 3 per cent of all pregnancies.

Rheumatic fever is responsible for the large majority of cases, varying from 87 per cent to 95 per cent and frequently there is the additional history of sore throats, scarlet fever and chorea: and the mitral valve is the seat of the lesion in all but a few .

Jensen (1938) states that the mortality rate has come down in recent years from 8 - 10 per cent to about 2 per cent: and he reports a series of cases

seen between the years 1930 and 1938 with a death-rate of 5.6 per cent. At Queen Charlotte's hospital the mortality rate has fallen from 5 per cent for the years 1928-1930, as reported by Gibberd (1947) to 3.1 per cent in the series reported.

Heart disease forms, too, an important and significant part of the maternal death-rate from all causes, accounting for 11.6 per cent of the total maternal deaths during the 10 years reviewed in this series. Various authors such as Stander (1941), and Munro Kerr and MacLennan (1932) have reported similar percentages to stress the growing importance of heart disease as a cause of maternal death. The progressive lowering of the death-rate because of better obstetrics and control of sepsis has served to bring out in relief this cardiac mortality associated in pregnancy. Hoffman and Jeffers (1942) do not, however, admit of this improvement in the mortality figure in their review of 61 fatalities.

In an attempt to lower the high mortality in heart disease complicated by pregnancy the New York Heart Association in 1922 recommended grouping these patients into distinct functional groups. This classification is based, as Jensen (1938) says, essentially on the work of MacKenzie (1921) who postulated that the response of the heart will vary according to the degree of its reserve force: and,

that although the field of response be limited, the patient is safe so long as that limit is not reached or exceeded. Pregnancy and labour place certain burdens on the heart. The enlarging uterus and its contents require nourishment and an increase in their circulatory needs; and there are circulatory changes to allow for a greatly increased maternal blood flow through the placenta; metabolic processes are accelerated and new endocrine relationships appear - all of which would, finally, appear to cause an increase in the work of the heart, with possible hypertrophy which, however, is not grossly apparent and which experimental studies have failed to shew. The effect of pregnancy and labour on the heart, requires, therefore, a knowledge of the heart's response to effort and according to this functional classification, the patient's response to the daily routine of work, places her in its various groups, according to which she is then treated. Many authors, including Pardee (1937), Stander (1941), Mendelson (1944) in America and Gilchrist (1931), McIlroy and Rendel (1931) and MacLennan (1933) in this country, have reported favourably on the classification: and Lamb (1937) has suggested that certain additional factors, which have been described (page 59) should be taken into consideration.

Apart, however, from the generally admitted seriousness of auricular fibrillation and the awareness that rheumatic disease is progressive and may, rarely, become active and darken the outlook for the patient, these additional factors are limited in the help they give. Harris (1937) states, for instance, that no difference in prognosis can be deduced from the type of lesion, whether mitral stenosis alone, or those of aortic regurgitation, or both together: and in Hunt's series of 156 cases reported by Campbell (1926), aortic lesions were found to be no more unfavourable than mitral: and Rae Gilchrist and Murray Lyon (1933) similarly found that the combination of multiple valvular defects had little influence on the production of earlier death. The help gained also from the cardio-thoracic ratio as suggested by Lamb (1934) is equivocal since there is displacement of the heart, which makes interpretation of the sign of doubtful value, and since in all events Gilchrist (1931) warns that a small heart may fail and a large one stand up to the strain of repeated pregnancies. Scott and Henderson (1934) commenting on this aspect of the problem state that, since some patients go through repeated pregnancies without mishap and no apparent ill effects, it must be assumed that the cardiac pathology in these patients is stationary and without myocardial damage, since, too, the cardiac

efficiency is dependant upon the condition of the myocardium rather than upon the valvular lesion.

It is also true that patients may change their group to a less favourable one as pregnancy advances and it may be advisable, as Jones (1944) suggests, to classify the patient according as to how she responded to effort before she became pregnant and then to assess her present condition; and should her grouping be one less favourable as a result of the pregnancy or the progress of the disease, a close and more frequent watch should be maintained for further deterioration throughout the remainder of the pregnancy, advising longer periods of rest.

A patient, 2705/46, gives an example of this uncertainty of behaviour not uncommon to the damaged heart. This patient, age 24, gravid 2, with a normal delivery of a 7 lb. 4 oz. baby seven years previously, was first seen at the tenth week of pregnancy and found to be in the functional group II. She continued in this group until the thirty-fifth week of pregnancy, when she reported with a history of sudden dyspnoea and oedema of the legs, which had appeared four days previously; an examination shewed that she was breathless at rest, with cyanosis, a pulse of 120 per minute but regular, moist sounds at both bases and oedema spreading up from the legs to the abdominal wall and back, and a liver three inches

below the costal margin - a fully-fledged group IV case. The heart lesion was a mitral stenosis, and the X-ray picture shewed a considerable cardiac enlargement. She went into premature labour at thirty-seven weeks, with the breech presenting while still in group IV. Abdominal section in such a state was considered inadvisable, and labour was allowed to proceed; with good sedation in the first stage and a pudendal block in the second, the patient had a fairly easy labour, which lasted in the first stage, four hours, and in the second, twenty minutes, and she was delivered of a live 6 lb. 2 oz. baby, without any additional distress or cyanosis. With continued medical treatment, her condition did not deteriorate, but it was the fourteenth day before she suddenly improved, began to sleep better and without drugs, and gradually losing her oedema and cyanosis, so that she was able to go home at the end of the fourth week. The postnatal report a month later still shewed, however, only a fair general condition with marked dyspnoea on exertion. Although she survived the pregnancy, she was undoubtedly much worse after it.

Weekly examination of this patient might have revealed earlier some warning signs of impending decompensation. Although Carr and Hamilton say (1933) that cardiac failure may occur in between the

weekly visit - apart from some precipitating cause such as an intercurrent infected heart failure develops more gradually and a weekly examination is sufficient. Where there is a history of previous heart failure, however, a close watch is essential and it is doubtful if the patient should be allowed to be away from hospital if the failure occurred earlier in the pregnancy.

MacKenzie (1921) and later Carr and Hamilton (1933) emphasised that it is the appearance and persistance of crepitations at the lung bases which are the harbingers of cardiac decompensation, and if there are none and there is no breathlessness, the possibility of heart failure can be dismissed until the next weekly visit.

To avoid cardiac decompensation and to lower the death-rate in the Group II patient is a problem of serious moment. It may be that owing to her more favourable condition earlier in pregnancy she is not receiving the frequent attention, which might reveal earlier, any signs of decompensation. It may be that if Groups I and II cases were included in this weekly examination, any change in their condition could be observed and treated at an earlier date and progressive decompensation could be avoided.

Again, it is possible that patients in Groups I and II are not sufficiently impressed about the limitations of their condition and tend to do too much. The daily routine of work should, therefore, be outlined and discussed with each patient, and the dangers of exceeding these bounds should be impressed upon her. Fitzgerald (1935) advises a social service in which patients are visited in their own homes to see if they are keeping within the prescribed limits of their ability, and he advocates free hospital care if necessary to relieve them of financial worry. He further admits patients to hospital if they are unable to do light house-work without breathlessness, if they have a persistent cough, or if their pulse is over 100, or if they need an extra pillow to sleep at night. They should also be warned about the risks of minor ailments, such as coughs and colds, and told to stay in bed immediately on their appearance.

Stander (1940) stresses the seriousness of both anaemia and upper respiratory tract infection, regarding the latter as especially ominous, and stating that it may be the first signal of a break in compensation. Jones (1944), too, states that bronchitis and influenza are often the precipitating cause of heart failure in pregnancy: and, since in his series, twenty per cent of the patients developed upper

respiratory infections, it is wise to warn the patient of this, and to confine her to bed at once until she is better.

As Carr and Hamilton (1933) have said, a stubborn medical control should be maintained on the patient's progress through the whole of her pregnancy: and the daily regimen organised so that she expends the same amount of energy each day, being warned against new duties such as changing house, or doing too much during holidays; and in this country it can be added standing in long queues to return with heavy shopping. It is further suggested towards this end that the cardiac patient should be able to obtain assistance from such a body as the Home Help Service, during her pregnancy as well as after it. Should she be over thirty years of age, some authors consider that the control should be even more strict and she should keep well within the limits of straining her heart, resting more frequently and taking only moderate exercise. The appearance of new murmurs during pregnancy may be noted with routine examination of the heart at each visit. Changes in the cardiac tones are not uncommon in pregnancy, although the rough gestatory murmur, described in page 26, is rare: these changes are not of any special significance.

Admission to hospital is recommended for all heart patients about the 28th week for rest and assessment. During her stay in bed observation is kept on the condition of her lungs and the state of her breathing, the pulse rate in sleeping and waking and its tension, her colour, the presence of oedema, and the urinary output. Examination is again made after the patient is up and performing some ward duties and her condition compared with that of the resting state and an idea thus formed of the capacity of her heart to respond to effort.

The detailed antenatal care of the patient with heart disease is time consuming and particular to a degree, but its careful pursuit should guide the majority of patients to labour, with a heart able to withstand the strain demanded of it. Pardee states (1937) that the treatment of decompensation of the heart is to prevent it, for the outlook is altered immediately this condition sets in. Hoffman and Jeffers (1942) conclude their review of cardiac fatalities by stating, that if there is to be a significant decrease in the maternal death-rate among cardiac patients, who are permitted to bear children, it must come through a prevention of congestive heart failure. The seriousness of this latter complication is brought out in the present series in which there was a mortality rate of 11.1 per cent in the more serious Groups III and IV.

The problem of therapeutic abortion is one which should be first dealt with at the cardiac clinic. Patients should there be advised whether or not pregnancy would be a grave risk. Advice to avoid pregnancy could be extended to Group III and IV cases, to those with a history of previous heart failure, and to patients who already have two children and whose circumstances are such as to deny them proper rest during the pregnancy.

In early pregnancy patients in functional groups III and IV who do not improve with two or three weeks of rest should be advised to have the pregnancy terminated. This it is safer to do by the vaginal route, since hysterotomy carries a definite mortality rate 8.3 per cent in this series and hence the decision should, if possible, be taken before the end of the third month.

The history of a previous breakdown in cardiac compensation, whether or not associated with a past pregnancy, is of bad prognostic significance, and should help towards the decision to terminate the pregnancy. Such a case was seen recently. A patient aged twenty-seven, primigravid, who had a well-compensated mitral stenosis, gave a history of becoming more breathless with pregnancy, and at the fourteenth week was coughing up a considerable amount of blood. With rest in hospital for two weeks, her

condition greatly improved. When seen at the twentieth week of pregnancy, the heart condition was apparently well-compensated, pulse 84, chest clear and no cough, and shortness of breath at the end of effort. She remained in the functional group II during the rest of her pregnancy, and went into premature labour at the thirty-fifth week. In the early part of labour, the patient was not breathless or cyanosed, her lungs were clear, but it was noted that she was anaemic. During the first stage of labour, which lasted twenty-two and a half hours, her pulse increased from 84 to 120, and her respiration to 36 over the last two and a half hours; the second stage was a spontaneous and easy breech delivery. The pulse did not properly settle during the early puerperium and, though regular, the rate was 124 at the end of the first week, and it was noted that there was a spreading oedema up the legs and over the sacrum and lower back, a few moist sounds appeared at the bases of the lungs, and the urinary excretion diminished. In a few more days, the oedema grew more marked and the crepitations increased, and the liver extended to one finger breadth below the costal margin and there was a marked oligura - the patient becoming breathless. She then developed a further complication of cerebral thrombi, which gave her a temporary paresis of speech, and facial and upper limb muscular weakness, a T.100.6, and pneumonic patches in the lungs.

Digitalis, mersalyl and penicillin procured rapid and impressive improvement in her condition, the urinary output increasing from 15 oz. to 66 oz. daily, and the oedema visibly diminishing and eventually disappearing.

It is clear from the above that a history of previous cardiac decompensation is serious and may portend a further failure, and should, therefore, as Gilchrist says (1931), be regarded as an unfavourable prognostic sign. It may be considered that such patients should be advised to have pregnancy terminated, unless there is a special desire on the part of the patient, as there was in the above instance, to continue to term. The above patient had been further instructed to come into hospital fourteen days before term, but, as already stated, premature labour may well preclude this, and it may thus be advisable to keep in hospital those patients who have given a history of previous failure and who wish to proceed with the pregnancy. Another interesting feature of this case is the rise of the pulse and respiratory rates above 112 and 24 in the first stage of labour, which Mendelson and Pardee (1942) regard as a warning of possible failure in the puerperium and an indication for immediate digitalisation. Finally, although in the functional group II such a patient as this,

with a history of past decompensation, should be considered with group IV cases as regards her obstetric treatment.

In the latter part of pregnancy there are few patients, states Mendelson (1944), whose condition cannot be maintained until they reach full term: and Hamilton (1947) goes as far as to say that it is most important not to interrupt the pregnancy in cardiac patients because of the heart condition after the load of pregnancy becomes heavy - even when the patient is in severe heart failure at six months: it would appear safer he asserts, for these latter patients, to nurse them along and wait for the expected improvement following diminution of the load on the circulation in the last four weeks. It is a clinical fact that patients rarely become worse, if under treatment, during the last four weeks of pregnancy, when lightening occurs and when, according to Cohen and Thomson (1939), the burden of work by the heart decreases. Full rest and if necessary digitalisation of the heart are imperative before termination of a pregnancy is considered.

As regards Caesarean section, the risks of performing an abdominal operation should not be forgotten and Hoffman and Jeffers (1942) state that the majority of fatal cases, which they reviewed, who were in group III, were delivered by Caesarean section.

In this series there was one death, a mortality rate of 4.8 per cent, in a patient (2143/38, page 123) aged 32 years, gravid 1, with mitral stenosis and in group III, who, although improved by a month's rest in bed prior to Caesarean section, died on the third day following the operation, with a fibrillating heart and all the signs of congestive heart failure.

The strict place for Caesarean section is where complications other than the heart disease are present, which indicate this operation; or where difficulty or delay during labour is anticipated, as for example in the presence of pelvic contraction, or a malpresentation, and in the rare case, which requires termination of her pregnancy in the last few weeks. No rigid rules can be given on this aspect, however, and in the latter type of patient, for instance, should the cervix admit two fingers and the head come easily into the pelvis, simple rupture of the membranes can procure a quick labour; but too frequently, this operation is associated with delay in labour, the patient becoming restless and anxious and reaching a stage, as in the case 1943/1002 (page 121) of this series, where active and too early interference may be required, with fatal results. Caesarean section, with its known amount of strain and shock is less of a risk in these circumstances.

It is noted, however, that the cardiac delirium which used sometimes to be seen following hasty termination of pregnancy before the patient, admitted in such an extremity, had been properly treated is now a rarity.

There is no place, it is suggested, for the recommendation occasionally heard that pregnancy should be terminated prior to term, so as to have a smaller baby and an easy labour.

Spontaneous labour at term is safest for both mother and child and in the vast majority of patients with heart disease this is the usual course of events. It is considered by some that the repeated effort of bearing down is too great a strain on the already damaged heart and hence the admonition, in practically every discussion in the literature, that the second stage of labour must be eliminated. The amount of muscular effort required during the second stage of labour depends on a combination of various factors such as multiparity, size of the foetus, pelvic measurements, condition of the soft parts, that it is not possible to say, with every certainty, what will happen in the individual case. Yet the outstanding fact about labour in the cardiac patient is that the second stage is short, with a quick and easy descent of the head. This may be associated with an

increased congestion or oedema of the soft parts, especially the cervix, leading to an easy dilatation and effacement in the first stage and quick descent of the foetal head. The first stage of labour, being principally a smooth muscle activity, puts no great strain on the myocardium except that resultant from exhaustion or mental un-rest, both of which are prevented by the use of Morphia. Little straining is needed, therefore, in the labour of many cardiac patients and this feature is even more conspicuous in the multiparous patient: should there be undue delay, however, forceps can be applied by the obstetrician, who should always be present during this stage.

The third stage of labour has a small but definite risk for the cardiac patient. The more or less sudden decrease in intra-abdominal pressure, coupled with the lowering of the diaphragm and the abrupt change in the cardiac axis, can be factors which may lead to cardiac collapse. A tight binder, or sand-bags placed on the abdomen, or even induction of a pneumoperitoneum are recommended by some. These have not been found necessary in the present series.

The collapse occasionally seen at this stage may be, on the other hand, associated with a congestive failure and much benefit has accrued from venesection, removing about half a pint of blood;

the blood drips from the vein slowly at first, but as the circulatory embarrassment lessens, a steady flow occurs. Intravenous strophanthin and coramine are used as cardiac stimulants, with oxygen for the cyanosis.

Sheehan and Sutherland (1940) say that the cardiac deaths during labour or soon after delivery, however, cannot be ascribed to the progressive deterioration of a badly decompensated heart during the physical strain of labour and they consider that they are usually catastrophic failures in patients who have been either not or only slightly decompensated.

The anaesthesia best advised for normal labour is a local pudendal block, which needs little supplementation if forceps are required. It is well known, however, that patients with heart disease stand anaesthetics well and perhaps the way the anaesthetic is given is of greater importance than the actual drug used. Local anaesthesia is ideal for Caesarean section if the patient has the sanfroid to submit to this unperturbed.

The need for Sterilisation is not an indication for Caesarean section, but it can be undertaken if indicated should hysterotomy or Caesarean section be performed. Tying a loop of the tube with some inabsorbable ligature is a quick method, but may be

followed by recanalisation, while resecting a piece of the tube and burying the end, although more certain in its results, has also a high failure rate when done at this time; in many cases it is advisable, therefore, to wait until a more favourable time to do this operation. Attendance at a contraceptive clinic is advised to others.

Deaths may occur during pregnancy as in case 1598/38 (page 122) of this series or due to the strain of an abnormal labour as in cases 518/46 (page 118) and 1002/43 (page 121), but the majority of deaths from heart disease occur during the puerperium. (page 127).

The cause of death in the majority of patients is congestive heart failure (page 131) and bears no relation to the actual heart valve affected. Gilchrist and Murray Lyon (1933) found congestive heart failure the mode of death in 92 per cent of the whole group of cases they reviewed. Sheehan and Sutherland (1940) postulate a recurrent endocarditis, which appears as a result of pregnancy, as the precipitating cause of the congestive heart failure.

As regards the distant prognosis it was not possible to say from the present follow-up series

that parity has any detrimental effect on the course of the disease. Gilchrist and Murray Lyon (1933) state that one or two children may be borne without detriment by the majority of cardiac women, but repeated pregnancies tend to shorten the span of life of women suffering from rheumatic heart disease and ultimately increase the risk of death from congestive heart failure. It was noted, however, that patients who changed their group to a less favourable one under the strain of pregnancy were more liable to be in a less favourable group at follow-up compared with patients who did not change their group.

No difference of age at death could be shewn by Boyer and Nadas (1944) among women who bore children and those who did not: and Gilchrist and Murray Lyon (1933) did not find any significant difference in the duration of the disease in nulliparous and parous women. It might be considered therefore that those women who die as a result of the strain of pregnancy are those who would anyway die at an early age with some other precipitating cause of death. It is also true that the intensive antenatal care of recent years has done much to help the woman with heart disease through her pregnancy and no doubt accounts, and along with the fact suggested by Gilchrist (1931) that it is the women with the less affected heart who marries and bears children, accounts for their equal longevity of life.

Subacute bacterial endocarditis, occurring during pregnancy is a serious complication, which can be suspected if the patient has a marked anaemia, an irregular temperature, a heart lesion and signs of emboli dissemination. An early diagnosis should greatly improve the chance of penicillin therapy towards the cure of the condition, although the sensitivity of the organism to penicillin has an important bearing on response to treatment. To give the mother a full chance it may be found necessary to terminate an early pregnancy as in the case described by Dobson (page 250). Vaginal delivery at term is otherwise indicated, the penicillin preventing the possibility of transplacental infection of the foetus which sometimes occurs.

Congenital lesions are treated according to the degree of reserve of the heart muscle, except for those conditions, in which an arterior-venous shunt may occur, or where rupture of the aorta may result from strain. In these latter lesions Caesarean section is advisable.

The after care of the patient is a final responsibility. It is not only during the pregnancy that the heart may be damaged and the old saying is true that the baby in the crib is a greater danger than the baby in the womb. The unending toil, with absence of home-help to relieve her of some of the

cares and burdens of daily task, may well prove too much for the patient, and finally exhaust the heart's reserve, producing a further limitation of her activity and shortening the expectation of life. It should, therefore, be within the province of hospital facilities to provide home-help to all patients with heart disease when they leave hospital; and many local authorities, already extending such assistance under a Home Help Scheme, could be asked to include the cardiac patient on this list. She should, also, have the opportunity to discuss with a sympathetic Health Visitor the future limitation of her life, and to be advised on the extent of her physical boundaries.

The liability of the Obstetrician does not end with the postnatal examination, but arrangements should be made for the patient's attendance at a cardiac clinic, where she can receive continued supervision, and advice regarding another pregnancy.

The irradication of Rheumatic heart disease is to be aimed at as the final goal in the treatment of this fell disease.

S U M M A R Y.

A series of two hundred and twenty-five patients with heart disease is reviewed, showing its incidence in pregnancy to be 0.8 per cent, and its mortality 3.1 per cent.

With better obstetrics and fewer deaths from sepsis, heart disease is occupying a relatively more prominent position as a cause of maternal deaths, being the fourth most common cause in this series, and accounting for 11.6 per cent of all maternal deaths.

Rheumatic fever was responsible for 93 per cent of cases, and the mitral valve was the site of the lesion in all but a few.

Observation of patients with heart disease at weekly intervals is recommended, owing to the danger of their condition deteriorating and in order immediately to observe any adverse change: and it is recommended that all patients should be admitted for one week about the twenty-eighth week, for rest and assessment, and again seven to ten days before full term. Antenatal care is one of the major factors in lowering the mortality rate, and each visit of the patient should include careful medical examination, especially of her lung bases for the early crepitations, close questioning as to her daily routine and

an assurance that she keeps within the limits of her cardiac reserve. It is possible that the more frequent examination and earlier advice and treatment of the Group II patients may be the key to the avoidance of the occasional failure in this group.

Respiratory infections, however trivial, have to be treated seriously, as they have a tendency to resist ordinary treatment and may precipitate heart failure; anaemia, too, should be avoided, and, if marked, a careful look-out kept for signs which might suggest subacute endocarditis, and the patient should be advised to report should she develop any new symptom.

The functional heart grouping of the American Heart Association gives a good indication of the cardiac reserve, and offers a common nomenclature for reporting results.

Some additional factors in assessment in individual cases, as suggested by Lamb (1934), may help to lessen the dangers of cardiac decompensation and, towards this end, the co-operation between the cardiologist and the obstetrician is essential.

A history of previous failure is a bad prognostic sign, and, although there is an apparent recovery, such patients should be regarded as group IV cases. It may, in fact, be advisable, as regards the obstetric treatment, to place the patient in the least

favourable group to which she may have reached, no matter what the improvement obtained with treatment, or what more favourable group she may later occupy during pregnancy.

Spontaneous labour, aided, if necessary, by forceps, would seem to be the best mode of delivery, and is possible in the vast majority of cases; Caesarean section being reserved for the presence of other obstetrical complications, or, in some cases, where surgical termination of the pregnancy is indicated.

It has been noted in this series, that patients with heart disease do not stand up well to prolonged labour or to accouchement force, and, if these are anticipated, a Caesarean section may be less of a risk to the patient. Observation of the pulse and respiration rates during the first stage of labour, as recommended by Mendelson and Pardee (1942), and immediate digitalisation, if necessary, may serve to prevent decompensation developing.

The anaesthetic which is best advised is morphia for the first stage and pudendal block for the second stage of labour, supplemented by a light gas and oxygen and episiotomy when forceps are used.

There is a definite place for therapeutic abortion in the group III and IV patients; and in others, who have given a history of previous heart failure, it is assessed individually.

The selection of bad-risk cardiac patients should be done at cardiac clinics, where they should be advised against becoming pregnant: and this precaution together with careful choosing of patients who are to proceed with pregnancy and avoid decompensation, with its attendant risks, can serve greatly to lower the mortality rate. Gilchrist and Murray Lyon (1933) say that one or two pregnancies do not shorten the expectation of life in the cardiac: and Jones (1944) advises against a third pregnancy. These observations have shown, however, that, although pregnancy may not induce heart failure, it may leave the heart severely crippled. It has been shown in this series that patients who have changed to a less favourable functional heart group during pregnancy are more liable to show these adverse effects in later years. No difference, however, in the age of death has been shown in nulliparous and parous women.

Of congenital lesions, unless there is persistent cyanosis, which would justify advising against pregnancy, or securing an early termination, the patient is treated as for other heart lesions: the exception being in those lesions where excess straining must be avoided and when, therefore, a Caesarean section should be performed.

A guarded prognosis has to be given until the end of the puerperium, since the majority of

fatalities occur then, and additional rest in hospital should be advised for patients at this time.

Before the patient leaves hospital, to assume the extensive burdens of the care of her child, a full opportunity should be taken to discuss with her the domestic responsibilities which she has to meet: and if indicated, as it is in most cases, arrangements made whereby she may have the assistance of a Home-help, such as is now available from most local Authority Departments. Finally, a date is given to the patient for attendance at a Cardiological clinic for future advice and guidance.

ABBOTT, M.	(1936)	Atlas of Congenital Cardiac Disease N.Y.
ALWARD, H.C.	(1932)	Amer. J. Obstet. Gynec. 20 : 373.
ANDERSON, D.F.	(1933)	Brit. Med. J. 1 : 224-225.
ANDERSON & HANLEY.	(1935)	Amer. J. Obstet. Gynec. 30 : 243-248.
BABER, M.D. & DALEY, D.	(1947)	J. Obstet. Gynae. Brit. Emp. 54 : 91.
BELL, W.B.	(1931)	Lancet. 1 : 1171.
BOYER & NADAS.	(1944)	Amer. J. Int. Med. 20 : 99-107.
BRADFORD, W.Z.	(1934)	Amer. J. Obstet. Gynec. 27 : 296.
BRAMWELL, C.	(1935)	Lancet. 1 : 629.
BRAMWELL, C.	(1935)	Brit. Med. J. 1 : 1132.
BRAMWELL, C.	(1931)	Pro. R. Soc. Med. 24 : 709.
BRAMWELL, C. & JONES, A.M.	(1944)	Brit. Heart J. 6 : 129-134.
BRAMWELL, C. & LONGSON, E.A.	(1938)	Heart Disease and Pregnancy, Oxford University Press, London E.C.4.
BROWNE, J.S.L.,) HINEY, J.S. &) VENNING, E.H.)	(1938)	J. Clin. Invest. 17 : 503.
BROWNING & CLARK.	(1941)	J. Indiana Med. Ass. 34 : 21.
BURWELL, C.S.	(1936)	South Med. J. 29 : 1194-1197.
BURWELL, C.S.	(1937)	Trans. Ass. Amer. Phys. 52 : 289.
BURWELL, C.S. & STRAYHORN.	(1933)	J. Clin. Investig. 12 : 977

BURWELL, C.S., STRAYHORN, W.D., FLICKINGER, D., CORLETTE, M.B., BOWERMAN, E.P., KENNEDY, J.A.,	(1938)	Arch. Int. Med. 62 : 979
CAIRNS.	(1937-1938)	Tr. Roy. Med. Clin. Soc. Glasgow, 79-80.
CAIRNS.	(1938)	Glasgow Med. J. January.
CAMPBELL, M.	(1926)	Guy's Hospital Reports, Page 76.
CARR, F.B.	(1938)	New England J. Med. 219 : 231-232.
CARR, F.B. & PALMER, R.S.	(1932)	Amer. Heart J. 8 : 238-241.
CASSIDY, M. & CAMPBELL, M.	(1944)	Brit. Med. J. 1 : 565.
CLAHR, J. et al.	(1940)	New York State J. Med. 40 : 1242-1247.
CLAHR, J. et al.	(1937)	Amer. J. Obstet. Gynec. 33 : 414-421.
CLAHR, J., GREENSTEIN, N.M., & KLEIN, M.D.	(1939)	Amer. J. Obstet. Gynec. 38 : 39-47.
CLUTE, H.M. & DANIELS, G.H.	(1930)	Amer. J. Med. Sc. 179 : 477.
COHEN, M.E. & THOMSON, K.J.	(1936)	J. Clin. Invest. 15 : 607
COHEN, M.E. & THOMSON, K.J.	(1939)	J. Amer. Med. Ass. 112 : 1556-1562.
COHEN, M.E. & THOMSON, K.J.	(1939)	Nebraska Med. J. 24 : 219.
COLWIN, J., HERRICK, W.W., VALENTINE, M., WILSON, J.M.	(1927)	Amer. J. Obstet. Gynec. 13 : 617.

COOMBS, C.F.	(1924)	Rheumatic Heart Disease, Bristol.
COUDERC, V.	(1912)	Grossesse et Cardiopathies Congenitales, Paris.
DALEY, P.A. & STROUSE, S.	(1931)	J. Amer. Med. Ass. 96 : 1655.
DANFORTH, W.C.	(1938)	Illinois Med. J. 74 : 88-91
DANFORTH, W.C.	(1944)	Wisconsin Med. J. 43 : 693-703.
DAWSON, J.	(1940)	New Zealand Med. J. 39 : 6-10.
DE GRAFF, A. & LINGG, C.	(1935)	Amer. Heart J. 10 : 459, 630.
DIECKMAN, W.J. & WEGNER, C.R.	(1934)	Arch. Int. Med. 53 : 71.
DILL & ERICKSON.	(1938)	Proc. Soc. Exper. Biol. & Med. 39 : 362.
DOBSON, C.	(1946)	Amer. J. Obstet. Gynec. 51 : 427.
DONAVAN, A.C.E.	(1936)	Brit. Med. J. 1 : 104-105.
EASTMAN, N.J.	(1937)	Med. Clin. North America. 21 : 1407-1416.
EASTMAN, N.J.	(1936)	Amer. J. Obstet. Gynec. 31 : 563.
EASTMAN, N.J.	(1933)	Internat. Clinic. 2 : 238-264.
FELDMAN & HILL	(1934)	Amer. Heart J. 10 : 110
FELLNER, O.O.	(1901)	Mschr. Geburtsh. Gynäkol., 14 : 370.
FELSEN et al.	(1937)	J. Amer. Med. Ass. 108 : 1783-1787.
FINDLEY, P.	(1921)	Amer. J. Obstet. Gynec. 2 : 278.

FITZGERALD, J.E.	(1935)	Amer. J. Obstet. Gynec. 29 : 53-61.
FITZGIBBON, G.	(1927)	Brit. Med. J. 2 : 253.
FRENCH, H. & HICKS, H.T.	(1906)	Brit. J. Obstet. Gynae. 10 : 201.
FREUND, H.A.	(1913)	J. Michigan Med. Soc. 12 : 648.
FRITSCH, H.	(1876)	Arch. f. Gynäk. 8 : 373.
GAMMELTOFT, S.A.	(1928)	Surg. Gynec. Obstet. 46 : 382.
GIBBERD, G.F.	(1947)	A Short Text Book of Midwifery. Page 246.
GILCHRIST, Rae A.	(1930-31)	Tr. Edinb. Obstet. Soc. Pages 121-135.
GILCHRIST, Rae A.	(1931)	Edinb. Med. J. September.
GILCHRIST, Rae A. & MURRAY-LYON, D.M.	(1933)	Edinb. Med. J. 40 : 587-597.
GORENBERG, H.	(1943)	Amer. J. Obstet. Gynec. 45 : 835-840.
GORENBERG, H. & MCGLEARY, J.	(1940)	J. Med. Soc. New Jersey. 37 : 114-115.
GORENBERG, H. & MCGLEARY, J.	(1941)	Amer. J. Obstet. Gynec. 41 : 44-52.
GREENHILL, J.P.	(1933)	Amer. J. Obstet. Gynec. 25 : 125-128.
GREENHILL, J.P.	(1931)	Surg. Gynec. Obstet. 53 : 547.
GREENHILL, J.P.	(1930)	Amer. J. Obstet. Gynec. 19 : 613.
GREENHILL, J.P.	(1946)	Year Book of Obstet. Gynec. Page 64. Year Book Publishes, Chicago.
GREENSTEIN, N.M. & CLAHR, J.	(1937)	Amer. J. Obstet. Gynec. 33 : 414.

HAMILTON, B.E.	(1927)	Boston Med. Surg. J. 197 : 763.
HAMILTON, B.E.	(1928)	New Eng. J. Med. 198 : 292.
HAMILTON, B.E.	(1937)	Amer. J. Surg. 35 : 284.
HAMILTON, B.E.	(1937)	Amer. Heart J. 14 : 555-561.
HAMILTON, B.E.	(1947)	J. Missourie Med. Ass. 44 : 17.
HAMILTON, B.E. & CARR, F.B.	(1933)	Amer. J. Obstet. Gynec. 26 : 824-839.
HAMILTON, B.E.,) CARR, F.B. &) PALMER, R.S.)	(1933)	Amer. Heart J. 8 : 519-524.
HAMILTON, B.E. & KELLOG, F.S.	(1928)	J. Amer. Med. Ass. 91 : 1942.
HAMILTON, B.E. & THOMSON.	(1941)	The Heart in Pregnancy and the Childbearing Age. Boston.
HANDFIELD-JONES,M.	(1896)	Lancet. 1 : 145
HANLEY, A. & ANDERSON	(1935)	Amer. J. Obstet. Gynec. 30 : 243.
HARE, D.C. & KARN, M.N.	(1928)	Quart. J. Med. 22 : 381
HARRIS, K.	(1937)	Lancet. 1 : 667.
HARRISON, T.R.	(1935)	Failure of Circulation, Baltimore. William and Wilkins Company.
HAY, J. & HUNT, E.	(1928)	Lancet. 1 : 271.
HENDERSON, D.N.	(1936)	Canad. Med. Ass. J. 35 : 394.
HENRY, J.S.	(1936)	Brit. J. Obstet. Gynae. 43 : 908.
HERRICK, W.W.	(1923)	New York Med. J. 117 : 546.

HERRICK, W.W.	(1933)	Foetal, Newborn, and Maternal Morbidity and Mortality. Page 95.
HOFFMAN & JEFFERS	(1942)	Amer. J. Med. Sc. 204 : 157-163.
HUNT, G.H.	(1926)	Guy's Hosp. Reports, April. 76.
JENSEN, J.	(1936)	South. Med. J. 29 : 572-574.
JENSEN, J.	(1936)	J. Amer. Med. Ass. 107 : 329-331.
JENSEN, J.	(1938)	The Heart in Pregnancy. Henry Kimpton: The C.U. Mosby Co., St. Louis.
JENSEN, J.	(1941)	Proc. Amer. Cong. Obstet. Gynec. (1939). 1 : 6.
JENSEN, J.	(1943)	Wisconsin Med. J. 42 : 1043-1046.
JENSEN, J., WEGNER, C., KEYS, E.H. & SMITH, H.R.	(1940)	Amer. J. Obstet. Gynec. 39 : 443-449.
JONES, A.M.	(1944)	P.G. Med. J. 20 : 176-184.
KIBEL, J.	(1944)	Amer. J. Obstet. Gynec. 48 : 553.
KING, E.L.	(1939)	Ohio State Med. J. 35 : 497.
KINNEY, T.D., SYLVESTER, R.F. & LEVINE, S.A.	(1945)	Amer. J. Obstet. Gynec. 210 : 725.
KOBACKER, J.L.	(1930)	J. Amer. Med. Ass. 95 : 266.
LAMB, A.E.	(1934)	Amer. J. Med. Sc. 187 : 177-184.
LAMB, A.E.	(1937)	Amer. J. Obstet. Gynec. 34 : 456-469.

LANDT, H. & BENJAMIN, J.E.	(1936)	Amer. Heart J. 12 : 592-607.
LENNIE, R.A.	(1937-1938)	Tr. R. Med. Clin. Soc. Glasgow, 125-138.
LENNIE, R.A.	(1938)	Glasgow Med. J., May.
LENNIE, R.A.	(1927)	J. Obstet. Gynae. Brit. Emp., 34 : 331.
LEWIS, T.	(1933)	"Heart Disease," London.
LIEBERMAN, B.L.	(1934)	J. Michigan Med. Soc. 33 : 305.
LIERE & SLEETH.	(1938)	Amer. J. Physiol. 122 : 34-37.
MACDONALD, A.	(1878)	The Bearing of Chronic Diseases of the Heart upon Pregnancy etc., London.
MACDONALD, A.	(1879)	Edinb. Med. J. 24 : 1110.
MACKENZIE, J.	(1904)	Lancet. 2 : 454.
MACKENZIE, J.	(1921)	Heart Disease and Pregnancy, Oxford Medical Publications, London.
MACLENNAN, H.R.	(1933)	J. Obstet. Gynae. Brit. Emp. 40 : 251-256.
MACRAE, D.J.	(1937)	J. Obstet. Gynae. Brit. Emp. 44 : 300-309.
MALPAS, P.	(1938)	J. Obstet. Gynae. Brit. Emp. 45 : 932.
McCLURE, H.I.	(1932)	J. Obstet. Gynae. Brit. Emp. 39 : 357.
McCLURE, H.I.	(1936)	Ulster Med. J. 5 : 234-240.
McILROY, L. & RENDEL, O.	(1931)	J. Obstet. Gynae. Brit. Emp. 38 : 6.
MENDELSON, C.L.	(1940)	Amer. J. Obstet. Gynec. 39 : 1014-1021.

MENDELSON, C.L.	(1944)	Amer. J. Obstet. Gynec. 48 : 329-338.
MENDELSON, C.L. & PARDEE, H.E.B.	(1941)	Amer. J. Med. Sc. 202 : 392-402.
MENDELSON, C.L. & PARDEE, H.E.B.	(1942)	Amer. J. Obstet. Gynec. 44 : 370.
MENGERT, W.F.	(1933)	Amer. J. Obstet. Gynec. 25 : 121.
MILLER, J.R.,) KEITH, N.M. &) ROWANTREE, L.G.)	(1915)	J. Amer. Med. Ass. 64 : 779.
MUNRO KERR, J.M.	(1931)	Brit. Med. J. 1 : 221.
MUNRO KERR, J.M. & MACLENNAN, H.R.	(1932)	Lancet. 1 : 633.
NAISH, F.C.	(1937)	J. Obstet. Gynae. Brit. Emp. 44 : 659-686.
NELSON, H.B. & EADES, M.F.	(1935)	New Eng. J. Med. 213 : 1057.
NIEHAUS, L.	(1939)	Nebraska Med. J. 24 : 219
OPPEL, T.W.	(1940)	Amer. J. Obstet. Gynec. 39 : 24-32.
PAGE & CAMPBELL.	(1939)	Amer. J. Obstet. Gynec. 38 : 97-104.
PARDEE, H.E.B.	(1929)	Amer. J. Obstet. Gynec. 17 : 255.
PARDEE, H.E.B.	(1930)	Arch. Int. Med. 46 : 470.
PARDEE, H.E.B.	(1937)	Amer. J. Obstet. Gynec. 34 : 557-564.
PARDEE, H.E.B.	(1934)	J. Amer. Med. Ass. 103 : 1899-1902.
PARDEE, H.E.B.	(1922)	Amer. J. Med. Sc. 164 : 847.
PETER, M.	(1874)	Brit. Med. J. 2 : 289.
PETER, M.	(1874)	The Obstet. J. 2 : 426.

PLASS, E.D. & YOAKAM, W.A.	(1929)	Amer. J. Obstet. Gynec. 18 : 556.
REGISTRAR GENERAL	(1946)	Health Report for England and Wales. March.
REID, W.D.	(1930)	Amer. J. Obstet. Gynec. 19 : 63.
REIS, R.A. & FRANKENTHAL, L.E.	(1935)	Amer. J. Obstet. Gynec. 29 : 44-52.
ROBINSON, A.L.	(1927)	Lancet. 1 : 170.
SAMPSON, J.J.	(1943)	West. J. Surg. 51 : 107-118.
SCOTT & HENDERSON.	(1934)	Amer. J. Obstet. Gynec. 27 : 342.
SHEEHAN, H.L. & SUTHERLAND, A.M.	(1940)	J. Obstet. Gynae. Brit. Emp. 47 : 597-668.
SHULEZ, A.V.	(1928)	Wein Klin Wehnschr. 41, 1374.
SHUMAN, W.	(1935)	Amer. J. Obstet. Gynec. 29 : 64.
SODEMAN, W.A.	(1940)	Amer. Heart J. 19 : 385-397.
SODEMAN, W.A. & KING, E.L.	(1944)	South Med. J. 37: 235-239.
SPARKS, T.P.	(1929)	South Med. J. 22 : 571.
STANDER, H.J.	(1934)	Amer. J. Obstet. Gynec. 27 : 528-536.
STANDER, H.J.	(1935)	Amer. J. Obstet. Gynec. 29 : 53.
STANDER, H.J.	(1938)	Amer. J. Obstet. Gynec. 36 : 413-419.
STANDER, H.J.	(1942)	Amer. J. Obstet. Gynec. 44 : 714-722.
STANDER, H.J. & CADDEN, J. F.	(1932)	Amer. J. Obstet. Gynec. 24 : 13.
STANDER, H.J. & CUDER, K.	(1937)	J. Amer. Med. Ass. 108 : 2092-2096.

STRAYHORN, W.O.	(1935)	J. Tennessee Med. Ass. 28 : 492.
STRAYHORN, W.O.	(1936)	"Medical Papers Dedicated to H. Christian," 134.
TEEL, H.M.	(1935)	Amer. J. Obstet. Gynec. 30 : 53-61.
TEEL, REID & HERTIG.	(1937)	Surg. Gynec. Obstet. 64 : 39.
TERWILLIGER, W.G.	(1934)	Amer. J. Obstet. Gynec. 27 : 248.
THOMSON, K.J. & COHEN, M.E.	(1938)	Surg. Gynec. Obstet. 66 : 591.
THOMSON, K.J.,) COHEN, M.E. &) REID, D.E.)	(1939)	Amer. J. Med. Sc. 198 : 665-679.
THORN, G.W. & HARROP, G.A.	(1937)	Science. 86 : 40.
TITUS, R.S. & STEVENS, W.B.	(1931)	Amer. J. Obstet. Gynec. 22 : 773.
TUNIS, B.	(1929)	Med. Klin. 25 : 1969.
TURINO, T.R. & ANTONY, A.T.	(1938)	Amer. J. Surg. 41 : 453.
WALKER, A.B. & EWING, A.C.	(1931)	Brit. Med. J. 1 : 221.
WALKER, C.W.	(1943)	Brit. Med. J. 1 : 190-191
WALSER, H.C.	(1921)	Amer. J. Obstet. Gynec. 2 : 278.
WEYLER, H.L.C. & DUSTIN, C.C.	(1942)	New Eng. J. Med. 227 : 785.
WHITE et al.	(1937)	J. Amer. Med. Ass. 109 : 863.
WILLIAMS, L.	(1937)	Delaware State Med. J. 9 : 207