PREGNANCY COMPLICATED BY CARDIAC DISEASE.

An Analysis of a Series of 100 Cases with Particular Reference to the Results of Treatment.

A Thesis for the Degree of M.D.

by

ROBERT AIM LENNIE, M.B., Ch.B.(1912), F.R.F.P.& S.(Glas.).

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Introduction.

The gravity of the complication which arises when pregnancy occurs in association with pre-existing cardiac disease was not sufficiently recognised or at least recorded until the latter half of last century. The earlier contributions to the literature of this subject were exclusively subscribed by observers on the Continent. Hecker[1] (1860), in recording two fatal cases, deserves the credit of having been the first to emphasise the dangers associated with this condition. Other Continental writers, including Spiegelberg[2], Peters[3], and Lebert[4] contributed important views on the subject, and many of these are still accepted. Thus, Spiegelberg pointed out the fact that a cardiac lesion need not necessarily give rise to disquieting symptoms during pregnancy, compensation remaining good throughout. He was also aware of the disturbances of the pulmonic circulation which were liable to occur, more especially during the later months, and he recognised the liability to prematurity in these patients. Peters was aware of the dangerous nature of the condition, and observed that although pulmonary complications were to be expected at some period or other in cardiac affections, pregnancy tended to precipitate these complications and to make them more formidable. He was conversant with the progressive nature of the disease and, consequently, recommended the avoidance of future pregnancy.

He also expressed the opinion that lactation caused too great a strain in these patients. In one instance where there was cardiac embarrassment he resorted to venesection with excellent result - a procedure, the value of which is still apparent and is referred to in the course of my thesis (p.37).

Lebert recognised that pregnancy tended to aggravate the cardiac condition, and that breakdown of compensation was more liable to occur as a result of pregnancy. He was also of opinion that there was the added danger of an acute endocarditis being superimposed on valves already damaged, this being prone to occur during the second half of pregnancy, and in the puerperium.

In this country Angus MacDonald (5) was the first to realise that the subject afforded a wide field for investigation, and he published in 1878 an exhaustive monograph on the results of his observations. Many of his views and conclusions have been confirmed and are still generally accepted. By way of historical interest I have summarised certain important conclusions from his book and compared or contrasted them with my own findings.

He recorded:-

(a). A very special tendency to abortion or premature labour in connection with patients suffering from mitral stenosis.

This tendency has long been recognised and is apparent from my observations on similar cases. Thus, in my own series 43 patients with mitral stenosis had collectively 13 premature births and 27 miscarriages in a total of 195 pregnancies. This liability is noteworthy, and in the text following Table II I refer to a patient who delivered herself normally at the first birth and had subsequently six miscarriages

in succession. Table VII further demonstrates the marked preponderance of abortion and premature labour which is associated with this lesion in comparison with other lesions.

(b). That the prognosis for patients with mitral insufficiency was more favourable than for those with mitral stenosis, although the condition was not devoid of danger.

MacDonald formulated this opinion as his mortality rate for stenosis was 64.4 per cent, whereas for incompetence it was 50 per cent. With such a high mortality rate it is not surprising that he was acutely conscious of the gravity of the lesion.

In my own experience of similar cases I have been more fortunate as I can record only two deaths out of a total of nineteen patients, i.e., 10.5 per cent. Evidence in support of this will be found in Table VIII. Great disparity, therefore, exists between MacDonald's figures and my own.

The lesion, undoubtedly, gives rise to anxiety in certain instances, and reference will be made to the danger being proportionate to the extent of involvement of the myocardium.

(c). That there was equal liability for mitral stenosis and aortic incompetency to cause prematurity.

MacDonald appears to have been justified in holding this view for premature labour supervened in four of his six cases with aortic incompetency. He described three of the four cases as having had a pure aortic lesion.

I cannot speak from experience of the single lesion as all of my four patients in this group were further complicated by mitral stenosis. It will be shown, however, in Table VII that three multiparae

with the double lesion gave no history of prematurity. A fourth patient, however, - a primigravida - came into premature labour, and reference is made to her in the text.

(d). That there was greater liability for acute endocarditis to occur in cases in which the primary valve lesion was of comparatively recent origin.

From a survey of the literature acute endocarditis during pregnancy appears to be a condition of rare occurrence, and I have found it so from my own experience as only two of the hundred cases were thus affected. The cases in question are fully discussed on page 15 of the thesis.

Within recent years many observers have contributed to the literature under review: chief among these may be mentioned Sir James Mackenzie's classical contribution entitled "Heart Disease in Pregnancy." By virtue of his experience both as an obstetrician and as a cardiologist, he approached the subject from an angle denied to others less fortunately placed. His views and conclusions are of supreme importance, and reference will be made to them as occasion demands.

More recently many authors, following on the lines of Mackenzie's investigation, have submitted important contributions, and among these may be mentioned Robinson[6], Hunt[7], Munro Kerr[8] and Fitzgibbon[9] in this country, and Pardee[10], Danforth[11], and Breed and White[12] in America. The subject has also been discussed at the meetings of various learned societies, and conflicting views have been expressed. The Midland Obstetrical and Gynaecological Society[13] held a conference for this purpose in February 1927, and in July of the same year the subject was included in a Special Section at the Annual Meeting of the British Medical

Association[14] at Edinburgh. Certain views expressed in these contributions and at the meetings will be considered in connection with my own observations.

With these remarks, which constitute a brief review of the historical aspect of the subject, I shall now record my personal experience of cardiac disease complicating pregnancy.

The treatment of such cases has given rise to considerable controversy in the past, and it is my hope that this contribution will prove helpful towards establishing a line of treatment which will be universally acceptable.

Intention of the Thesis.

In submitting this Thesis I may say that the subject has been approached with the purpose of determining from observations on a long series of cases, the relationship existing between the various cardiac lesions, the parity of the patients, and the stage of pregnancy at which compensation broke down.

Further, a survey of the previous personal and obstetric histories, and an inquiry into the incidence of prematurity seemed to hold promise of much interesting information.

Finally, by an analysis of the different methods of treatment resorted to in dealing with these patients, it was hoped to arrive at some conclusion as to the best procedure to adopt in this serious complication of pregnancy.

With these objects in view, I have collected one hundred cases which have been admitted during the last seven years to the wards with which I am associated. The case sheets have been taken in rotation, and the journals to date have been searched, but I have had of necessity to discard several owing to their incomplete histories. Certain patients admitted temporarily to the ante-natal wards were lost sight of after dismissal, while others went out irregularly.

Before proceeding further I should like to mention that in recent years either Caesarean section with sterilisation performed in

the early months or near term, or early hysterectomy as a method of treatment in certain cases of cardiac disease has found favour with some of us, and the results so far obtained have been admirable.

I shall refer to this later, amd hope to substantiate by data the fact that this is a valuable and safe line of treatment. The excellent results obtained by Caesarean section even in the worst cases prompted me to look into this subject.

Without digressing further, the salient features of these hundred cases will now be discussed and simplified where necessary by means of statistical tables.

Discussion regarding the Pregnancy in which Loss of Compensation is most liable to occur.

TABLE I.

Showing in which Pregnancy Compensation was Lost.

Loss of Compensation	occurred	during	1st	pregnancy	in			22.
"	"	"	"	2nd	"	"		19.
"	"	"	"	3rd	"	"		10.
"	"	"	"	4th	"	"		6.
"	"	"	"	5th	"	"		9.
"	"	"	"	6th	"	"		7.
"	"	"	"	7th	"	"		7
"	"	"	"	8th	"	"		7.
"	"	"	"	9th	"	"		4.
"	"	"	"	10th	"	"		9.
								100.

It will be apparent from the figures in the above table that of cardiac cases the greatest number show loss of compensation in the earlier pregnancies, especially the first and second, in which the figures are approximately double that of the third pregnancy.

The table is slightly inaccurate as ten patients had the causative illness intervening since commencement of the childbearing period. Thus, one multipara (Grav. 5) had rheumatic fever one year previously, and another (Grav. 4) since previous birth. Two additional patients, therefore, had loss of compensation in the first pregnancy following illness. Again, one (Grav. 10) had rheumatic fever three years previously, one (Grav. 11) four years, and so on. As in some instances the journals were incomplete, it was impossible to determine in the latter cases if this were the first pregnancy since illness.

Consideration of the Period of Pregnancy at which Compensation breaks down.

Table II.

Period of Pregnancy at which Compensation broke down.

Gravida.	Unknown.	3 m.	4 m.	5 m.	6 m.	7 m.	8 m.	9 m.	Total.
1st	-	2	4	2	2	3	4	5	22.
2nd	1	2	4	4	3	0	2	3	19.
3rd	1	2	0	2	1	1	2	1	10.
4th	-	0	1	1	0	2	2	0	6
5th	-	0	0	1	7	1	0	0	9
6th	-	1	1	2	1	2	0	0	7
7th	-	1	0	1	2	1	0	2	7
8th	-	3	1	0	1	1	0	1	7
9th X	-	1	1	1	4	2	4	0	13
Total,	2	12	12	14	21	13	14	12	100

It is apparent from Table II that there was a greater tendency for loss of compensation to occur in primigravidae after mid-term, and four patients went to the eighth and five to the ninth month before showing urgent symptoms.

With second gravidae the position was reversed, ten showing distress by the fifth month as against eight in the second half. Of these patients six stated they were breathless at their first pregnancy, one received ante-natal treatment, one had a miscarriage, and three required delivery with forceps. The remainder experienced no untoward symptoms during the first pregnancy or labour. Strictly speaking, therefore, one patient definitely, and ten patients probably, should have been included in the category of primigravidae in Table I.

In the third pregnancy the onset of breakdown was fairly uniform. One of these patients was breathless at both previous pregnancies, and premature labour was induced at the second. In two cases the child was born prematurely, while a third was distressed in her second pregnancy. Another patient who had had two normal full-time deliveries previously was admitted on two occasions for ante-natal treatment during her third pregnancy.

Of the total deaths four died during their third pregnancy or labour.

I think these figures are conclusive of the increasing strain which accompanies each succeeding pregnancy, and this will be even more apparent as we proceed.

In the fourth pregnancy it will be noted that compensation was lost in all cases before the ninth month. The tendency to breakdown, however, occurred in the second half.

In the fifth pregnancy compensation was lost in eight instances by the sixth month, and of the nine patients four had premature labour, one had induction of miscarriage performed, and one was delivered by Caesarean section at the eighth month before labour set in.

In the sixth pregnancy breakdown occurred in all instances by the seventh month.

In the seventh and eighth pregnancies three patients did not experience urgent symptoms until they were near term. One of these with mitral regurgitation had six normal full-time children previously, and another with a similar lesion had seven normal births. These long series of uheventful pregnancies are accounted for by the circumstance that mitral regurgitation is, as a rule, a less serious condition. On the other hand, a patient with stenosis who delivered herself normally at the first birth had subsequently six miscarriages in succession.

During the present pregnancy she was admitted on two occasions for ante-natal treatment and spent over three months in hospital. She eventually died after induction by bougies near term.

The ominous symptoms and wastage evidenced in this obstetrical history are obvious, and in all probability had sterilisation been performed at an earlier period, her life would have been prolonged.

No patients with nine or more pregnancies went to term.

Generally speaking, it is apparent from Table II that loss of compensation occurred in the greatest number of patients during the sixth month, while in the other months the numbers are fairly uniform. It is to be expected that the greatest strain will be met with towards the end of pregnancy, and in many instances this is observed clinically. The apparent discrepancy in the later months is accounted for by the tendency to prematurity in cardiac cases. The subject will be referred

to later.

The Time of Onset of Loss of Compensation Relative to the Cardiac Lesion.

TABLE III.

	3 m.	4 m.	5 m.	6 m.	7 m.	8 m.	9 m.	Unknown.	Total	
Mitral Stenosis	7	5	6	8	3	5	3	-	37	68
Double Mitral	4	3	5	9	7	2	1	-	31	
Mitral and Aortic	0	1	0	1	0	2	0	-	4	
Mitral Regurgitation	1	2	0	3	3	4	5	1	19	
Myocardial	0	0	2	0	0	1	2	-	5	
Paroxysmal Tachycardia	0	1	0	0	0	0	0	-	1	
Lesion unknown	0	0	1	0	0	0	1	-	2	
Total	12	12	14	21	13	14	12	1	99	
Lesion & time unknown									1	
									100	

Table III conclusively proves that of the cardiac lesions complicating pregnancy mitral stenosis is most to be feared, for we find 68 of the 99 cases suffering from this disease, i.e., 68.7 per cent.

A remarkable similarity in the statistics of the various authors regarding the incidence of mitral stenosis may be noted, viz:-

Reference. (15)	Number of Cases.	With Mitral Stenosis.	Percentage.
Pardee	35	20	57.1
Danforth	29	15	51.7
Robinson	39	26	66.6
Breed and White ..	53	36	68.0
Hunt	156	109	63.4
Fitzgibbon	22	13	59.0

These figures give approximately an incidence of 60 per cent.

The table further demonstrates that of 68 cases of stenosis (including double mitral lesions) distress appeared in 30 cases (44.1 per cent) by the end of the fifth, and in 47 cases (69.1 per cent) by the end of the sixth month. Only four patients of the 68 with stenosis actually went to term without showing definite cardiac symptoms.

Of the four patients with a double mitral and aortic lesion one broke down at the fourth month, one at the sixth month, and two were not distressed until the eighth month. The patient with grave symptoms at the fourth month will be referred to later in connection with Caesarean section. The one who suffered breakdown at the sixth month had three normal deliveries previously, but had scarlet fever intervening six years before. Of the remaining two, one had three normal births previously and the other twelve. The previous birth of the latter patient had occurred four years before, and she was credited with a "water-hammer pulse". As this pulse is significant of damage to the heart muscle, it seems probable that the heart condition was of fairly

recent origin.

In the cases with mitral regurgitation three out of nineteen showed urgent symptoms by the end of the fourth month, but the greater proportion were not distressed until the second half, and five went to term (26.3 per cent).

In myocardial mischief urgent symptoms were of late onset in three out of the five cases. The two remaining cases showed urgent symptoms about mid-term. One of them was examined by a physician (Dr. Geoffrey B. Fleming) on the strength of whose unfavourable prognosis it was decided to perform miniature Caesarean section and sterilisation. The patient was dismissed well. The patient with paroxysmal tachycardia was admitted to the ante-natal wards about the fourth month. Apart from rapidity of the pulse-rate no other abnormality was noted. (She was recommended by an Assistant Physician on the Staff of one of the Infirmaries).

For the interpretation of these figures much interesting information is to be obtained in Sir James Mackenzie's book "Heart Disease in Pregnancy"(16). In this very valuable work it is stated that "the heart affection which most frequently causes danger in pregnant women is that associated with mitral stenosis following rheumatic fever". Again,"there are forms of this complaint which are attended with no danger, and there are others which imperil the life of the woman".

Table III conclusively establishes the truth of the first statement, for of the 100 cases 68 had stenosis, and of these 33 had definitely suffered from rheumatic fever. With regard to the latter statement, it was found in the cases under consideration that several women with mitral stenosis went through a succession of pregnancies without apparent embarrassment. Thus, I have records of two patients (both fifth Pregnancy)

with four, one (seventh pregnancy) with seven, and one (eleventh pregnancy) with nine normal full-time births previously.

That the danger is never absent in patients with stenosis is evident from the following histories. Two patients (sixth and seventh pregnancy) who had never experienced any cardiac embarrassment previously, were admitted to the hospital with urgent symptoms about mid-term. Both were seriously ill and were kept under ante-natal supervision for sixty and one hundred days respectively; one of them had auricular fibrillation. The pregnancy was terminated in each instance by Caesarean section and both were dismissed well with live children.

Mackenzie emphasises the truth that mitral stenosis is a progressive process which goes on with varying rapidity, and he lays stress on the fact that a similar cicatrizing process may be going on in the muscle, impairing its functional activity, and, thus, the efficiency of the heart is profoundly affected.

The damage to the heart muscle is an important factor governing the incidence of breakdown. In some patients embarrassment may not occur until the narrowing of the orifice is extreme, and owing to the progressive nature of the disease this may only occur after a prolonged period of time. In all probability gradual narrowing of the valve orifice accounts for the belated onset of urgent symptoms in the two cases to which reference has just been made.

In others loss of compensation may occur at an earlier period while the narrowing is still moderate, and in these instances the muscle wall has been damaged.

With advancing stenosis a diastolic murmur makes its appearance, and this is usually associated with the onset of grave symptoms of cardiac failure. Should auricular fibrillation occur the outlook is ominous.

Another symptom of grave import is oedema of the lungs.

Mackenzie also mentions that if dilatation of the heart appears early it signifies that the rheumatic process has permanently injured the heart muscle, and he further avers that the damage consequent on rheumatic fever is rarely limited to the valves.

Mitral regurgitation is a much less serious condition and it is a common experience in hospital to find patients with a mitral systolic murmur whose child-bearing period has been uneventful, and who have had no suspicion that they suffered from organic disease. In these instances when compensation breaks down it is evident that the cardiac muscle has been involved.

There is, however, another factor which must not be overlooked, and which, if rare, is of grave import, namely, a fresh infection superimposed on diseased valves.

Consideration of Acute Endocarditis.

My own experience of acute endocarditis is limited to two patients in the series, both of whom died. One of these - a primigravida, aged 20 - was admitted when six months pregnant in a semi-conscious condition, with complete loss of compensation. In addition, she was paralysed down the left side. On examination she was found to have a double mitral lesion with auricular fibrillation, and the heart was much dilated. Crepitations were heard over the bases of both lungs. It was ascertained that she had suffered from rheumatic fever one year previously, and shortly before admission there had been a recurrence. After twelve days in hospital she went into premature labour and this was only discovered when the foetal head was on the perineum. She died

almost immediately. Throughout her illness there was no great elevation of temperature, but the pulse remained irregular and very rapid.

The post-mortem examination revealed stenosis of the mitral orifice with recent vegetations on the valves.

The other patient (Gravida 12), aged 38, was admitted very gravely ill when fully seven months pregnant. She could recall no illness but stated that her breathing had been greatly distressed during the five previous pregnancies. She was found to have a double mitral lesion, and the pulse was very rapid and of poor quality; oedema was negligible and the lungs were clear. Two days after admission she delivered herself spontaneously of a premature child and she died within two hours. Prior to the commencement of labour the temperature rose to 103°.

At the post-mortem examination recent vegetations were found on previously narrowed mitral valves.

Cruickshank (17), who performed the post-mortem examinations on these cases, records the case of a patient who developed sudden and severe decompensation when about three months pregnant, and who only survived a few days. The temperature remained afebrile throughout. She gave a history of having had acute rheumatism during her last puerperium five years previously.

At the postmortem examination a chronic mitral stenosis with recent vegetations on the mitral, aortic, and tricuspid valve segments was revealed.

Croom (18) (1906) drew attention to the rarity of the occurrence of malignant endocarditis during pregnancy, and in an extensive review of the literature could find records of only six cases. He was of opinion that it was an accidental complication, and that pregnancy had merely a

slight predisposing influence. He recorded the case of a patient who came under his care when eight months pregnant. Six years previously she had pneumonia, and she had suffered from attacks of dyspnoea and oedema since that illness. When admitted to hospital her symptoms were not unduly urgent, but on the following day the temperature rose to 103°F. and the pulse rate increased to 126 beats per minute. Her condition remained grave for three days when premature labour occurred and she was delivered with forceps. Death resulted twenty hours later.

Septic endocarditis of the aortic and mitral valves, together with chronic endocarditis and acute myocarditis were found at the post-mortem examination.

From a consideration of these cases Croom's contention that the complication is an accidental one during pregnancy has much to commend it. It has been known to occur in pregnancy in the healthy heart from causes such as pneumonia and pyelitis (Cruickshank), but is more frequently met with during the puerperium, especially in puerperal sepsis where it may be the terminal phase.

Cowan (19) writes with regard to acute endocarditis "It is important to realise that the fatal termination in cases of chronic valvular disease is very frequently accompanied by and most often due to an acute re-infection of valves which are already damaged". He substantiates this statement by observing that in a post-mortem series of 107 cases of chronic valvular disease, 57 were found to have an acute endocarditis. Cowan's observations were based on the study of chronic heart disease apart from pregnancy, but his statistics show the clamant danger which is always present irrespective of any strain such as pregnancy.

In my series of 100 cases only two patients are definitely known to have had acute endocarditis. It is reasonable to assume that if pregnancy had a predisposing influence on this complication, there would have been a greater proportion of the cases thus affected. I incline, therefore, to Croom's view that its occurrence is accidental in pregnancy, although, possibly, the extra strain is a slight predisposing factor. My experience fails to support the opinions of Lebert and MacDonald that in pregnancy this danger is much more acute.

Attributable Causes of the Cardiac Disease.

To return to the main series, it is apparent from the perusal of the personal histories that rheumatic fever is by far the most responsible factor in the causation of cardiac disease.

TABLE IV.

Rheumatic Fever	41
Chorea	11
Scarlet Fever	6
Influenza	4
Pneumonia	1
Diphtheria	1
Heart Disease (origin.unknown)	5
Claimed no illness	18
Not noted in Journal	13
	100

In several instances the patient had two illnesses either of which might have been responsible for the presence of the cardiac lesion.

For example, two patients gave histories of having had rheumatic fever and chorea, one, rheumatic and scarlet fevers, one, chorea and scarlet fever, and so on. In compiling the above table these patients have been classified according to the precedence of the causative illnesses in the column. Thus, those who had suffered from rheumatic fever and chorea are classified under rheumatic fever.

Breed and White, in an analysis of 102 cases, assert that the lesion is almost invariably of rheumatic origin, and that unusual cardiac affections due to pneumococcus, gonococcus, streptococcus or influenza bacillus, etc. are seldom met with as the patient, as a rule, does not survive the illness.

The figures in Table IV verify this assertion.

The majority of the patients contracted these diseases in childhood. In sixteen cases, however, eleven of which were multiparae and five primigravidae, the cardiac lesion was of comparatively recent origin; the longest period which had elapsed between the causative illness and the present pregnancy was six years, and the shortest, twelve months. With one exception compensation was lost in all instances by the seventh month, and the tendency was towards an even earlier onset of urgent symptoms. There were five deaths in this group alone, giving a mortality rate of 31 per cent. Eight of the patients who survived were under ante-natal treatment in hospital for periods varying from 30 to 100 days.

It is obvious, therefore, that the prognosis is much more unfavourable when pregnancy follows the heart affection within a few years than when the history of the cardiac lesion dates from childhood.

The Manner in which the Pregnancies Terminated.

TABLE V.

Normal deliveries	15	18
Birth before Arrival (B.B.A.)	3	
Premature spontaneous	15	17
Miscarriage	2	
Forceps	14	
Manual dilatation and forceps	2	
Induction by bougies	10	
Medical induction	1	
Induction of abortion	4	
Caesarean section	16	24
Miniature Caesarean section	4	
Hysterectomy	4	
Version	1	
Died undelivered	5	
	96	
Unknown	4	
	100	

It is apparent, therefore, that of the 96 known cases 18.7 per cent had normal full-time, and 17.7 per cent premature deliveries while 58.3 per cent required interference of some description on account of the gravity of their illness. It is interesting to note that Fellner (20)

in a series of 94 cases had a 20.2 percentage of premature deliveries.

Reference to Premature Deliveries, and Observations on the Incidence of Prematurity relative to the Lesion.

When the number of pregnancies which had of necessity to be terminated before term is taken into consideration the prematurity percentage is raised by 27 per cent, and in addition there is what might be termed a wastage of 17.7 per cent.

TABLE VI.

Premature Deliveries.

Forceps	2	
Dilatation and forceps	2	
Caesarean section	10	27 per cent.
Induction	11	
Version	1	
	26	

Wastage.

Induction of abortion	4	
Miniature Caesarean section	4	
Hysterectomy	4	17.7 per cent.
Died undelivered	5	
	17	

It is somewhat difficult to assess the prematurity rate of the previous obstetrical histories as these are only available from statements made by the patients, which are, in many instances, unreliable. From the material at my disposal, however, the following figures have been compiled from 64 patients (excluding primigravidae and cases with intervening illnesses).

TABLE VII.

Analysis of Previous Obstetrical Histories.

		Full-time.	Premature.	Miscarriage
43	Mitral Stenosis	90	6	15
	Double Mitral	65	7	12
		155	13	27
3	Double Mitral & Aortic	15	0	0
14	Mitral Regurgitation	53	1	3
4	Myocardial	15	0	2
64		238	14	32

Thus, 43 multiparae with mitral stenosis (including double mitral lesions) gave a history of 155 normal births, thirteen premature births and twenty-seven miscarriages. Three multiparae with mitral and aortic disease give a remarkable history of fifteen normal births previously. One of these patients has been referred to as having a "water-hammer" pulse, while another had her last birth fourteen years before.

Lack of damage to the cardiac muscle might account for the uneventful nature of these births, but in all probability the disease has been acquired at a later date from some cause unknown.

A combination of mitral and aortic lesions can, however, be very serious, as is instanced in the case of the fourth patient in this group - a primigravida - who was dangerously ill from the commencement of pregnancy and subsequently died.

With regard to mitral regurgitation, 14 patients had 53 normal births, 1 premature birth and 3 miscarriages previously, and 3 patients with myocardial mischief had 15 normal births and 2 miscarriages.

The table, at least, has some value in demonstrating the greater liability of stenosis cases to have premature labour or miscarriage.

The Lesions responsible for the Fatal Termination.

With regard to the deaths, we find that 23 of the series of 100 patients succumbed during the present pregnancy, labour or puerperium.

Fitzgibbon in a series of 22 cases records a fatality rate of 22.7 per cent.

TABLE VIII.

Lesions responsible for the Fatal Termination.

68	Mitral Stenosis	11	17
	Double Mitral	6	
4	Double Mitral and Aortic	1	
5	Myocardial	2	
19	Mitral Regurgitation	2	
1	Paroxysmal Tachycardia	1	
		23	

From Table VIII it is evident that mitral stenosis was responsible for the great majority of the deaths, i.e., 17 out of 23. An investigation of the various lesions shows that death occurred in 17 of the 68 cases of mitral stenosis, while one of the four patients with a double mitral and aortic lesion died. The fatality rate for these lesions is, therefore, the same, i.e., 25 per cent. The highest fatality rate of all is met with in pure myocardial disease, i.e., 2 out of 5 or 40 per cent. The lowest occurs in patients with mitral regurgitation as only 2 out of 19,or 10 per cent, died. One of the latter patients had definite dilatation and, consequently, a damaged heart muscle while the other, who was a cardio-renal subject, died during an operation for secondary suture following Caesarean section. Reference will be made to this case in due course. The unusual complications present in the second patient probably makes the percentage for mitral regurgitation unduly high; six per cent might be a more accurate estimate. As comparatively few cases of aortic or pure myocardial disease are met with, the greatest danger is to be expected from mitral stenosis owing to the great preponderance of these cases. As the only patient with paroxysmal tachycardia succumbed, no accurate information regarding this lesion is available.

With regard to the parity of the fatal cases, 8 out of 22 primigravidae and 15 out of 78 multiparae died, giving a fatality rate of 34.7 and 19 per cent respectively.

Certain other features are worthy of attention in connection with the fatal cases. Of the 23 patients who died 5, or 21 per cent had the causative illness within the past few years, and two had been in hospital on account of cardiac disease a short time before pregnancy occurred. Munro Kerr refers to the danger pregnancy imposes when the heart condition has recently given rise to symptoms of urgency, thus,

"Specially anxious must one be if prior to the pregnancy there is a history of unsatisfactory compensation". The significance of a history of recent illness has been referred to, and I regard with apprehension any patient who claims to have suffered recently from loss of compensation. The fatal termination in the two cases mentioned above is ample justification for a grave view being taken.

With the exception of two instances in which the time of breakdown is unknown, sixteen of the fatal cases had lost compensation by the sixth month. The gravity of the complication when loss of compensation occurs early is further demonstrated.

The Significance of Cardiac Disease in Association with Primigravidae.

The high mortality rate associated with the first pregnancy merits special consideration.

TABLE IX.

Giving Particulars of the Primigravidae who Died.

No.	Lesion.	When Compensation Failed.	Condition at Delivery.	Method of Delivery.
1.	Double Mitral & Aortic	4 months.	Compensation lost.	Caesarean section.
2.	Mitral Stenosis	5 "	-	Died in two days - undelivered.
3.	Mitral Stenosis	6 "	Compensation lost.	Forceps.
4.	Mitral Stenosis	3 "	Compensation lost.	Caesarean section.
5.	Paroxysmal Tachycardia	4 "	Compensation lost.	Manual dilatation and forceps.
6.	Mitral Stenosis & dilatation	4 "	-	Died on admission.
7.	Double Mitral	6 "	Compensation lost.	Premature - spontaneous.
8.	Double Mitral	Unknown.	Moribund on admission. ($7\frac{1}{2}$ months)	Caesarean section.

It will be noted that mitral stenosis was the lesion present in six of the eight fatal cases, while one had both mitral and aortic disease and the remaining one paroxysmal tachycardia. The early onset of symptoms is rather impressive for in only two instances did the patient reach the sixth month without cardiac embarrassment. The patient admitted when moribund was seven and a half months pregnant and, obviously, should have been in hospital at a much earlier period. Several of the fatal cases required ante-natal supervision in hospital and were carried on by palliative measures until such time as their pregnancies were completed. In one instance compensation was regained and the patient was discharged only to be re-admitted almost immediately in worse plight than before.

It is apparent from the table that the prognosis among primigravidae is most unfavourable when there is an early manifestation of breakdown in compensation. Further evidence in support of this conclusion will be found in the next table which deals with the primigravidae who recovered. There it will be shown that in only three instances was compensation lost at an early period, and in two of these the pregnancy was terminated almost immediately by hysterectomy and miniature Caesarean section respectively. It is reasonable to assume that these patients owed their recovery to the urgent measures adopted. The third patient was continuously in hospital for three months until the pregnancy ended prematurely.

To return to the consideration of the fatal cases, it will also be noted that, apart from the three patients who died on admission or within two days, compensation was lost in all instances at the time pregnancy was terminated.

Remarks referring to the method of delivery of these patients will be included in their appropriate sections.

Turning now to the details regarding the primigravidae who recovered, a striking contrast is presented.

TABLE X.

Giving Particulars of Primigravidae who Recovered.

No.	Lesion.	When Compensation Failed.	Condition at Delivery.	Method of Delivery.
1.	Double Mitral	8 months.	Compensation regained.	Mid-forceps.
2.	Mitral Stenosis	8 "	"	Induction (8 months)
3.	Mitral Regurgitation	8 "	"	Forceps.
4.	Double Mitral	3 "	Improved	Hysterectomy.
5.	Double Mitral	4 "	Compensation regained (3 months in hospital).	Premature - spontaneous.
6.	Myocarditis	Term	Distress in labour.	Forceps.
7.	Mitral Regurgitation.	Term.	Distress in labour.	Forceps.
8.	Mitral Stenosis.	3 months.	Compensation regained.	Miniature Caesarean section.
9.	Mitral Stenosis.	8 months.	Compensation regained.	Spontaneous - Full-time.
10.	Double Mitral.	5 "	Compensation regained. (one month in hospital)	Forceps.
11.	Mitral Regurgitation.	Term.	Distress in labour.	Spontaneous.
12.	Mitral Stenosis	Term.	Distress in labour.	Forceps.
13.	Mitral Stenosis	No loss of compensation. (Admitted in labour).	Normal.	Low-forceps.
14.	Double Mitral	7½ months.	Compensation regained. (2 admissions to hospital)	Spontaneous.

It is at once apparent that urgent symptoms were not experienced by the majority of these patients until towards the end of pregnancy. Reference has already been made to the exceptions. Further, compensation was regained in most instances by the time the patient was delivered. It is also of interest that the four patients who showed distress were admitted in labour, and, consequently, had not been under ante-natal supervision in hospital. The importance of rest and pre-natal care for such patients is, therefore, obvious.

With regard to the cardiac lesions in these cases, it will be observed that mitral stenosis was present in ten instances. Thus, of the twenty-two primigravidae comprising the series, this lesion was met with in sixteen cases, while one had further the complication of aortic disease. Again we are confronted with the gravity and frequency of this form of the complaint.

These tables, therefore, emphasise the dangers which are attendant on an early manifestation of urgent symptoms in primigravidae.

It seems disastrous to procrastinate so far as terminating the pregnancy is concerned. An attempt may be made, often at the express desire of the mother, to carry on the pregnancy in the hope of securing a viable child. Several instances are recalled where better judgment was perhaps swayed by sentiment in trying to attain this object, and the results proved the folly of the procedure.

On the other hand, attention has been drawn already to the two instances in which the patients recovered, probably as a result of the early termination of their pregnancies.

These remarks only apply to cases in which severe cardiac embarrassment is experienced in the early stages of the first pregnancy. When symptoms are of late onset it is reasonable to expect a favourable conclusion to the pregnancy.

Fitzgibbon states,"When compensation fails in a primigravida during pregnancy it can usually be re-established and when labour subsequently occurs it is well borne".

I am in agreement with this statement in so far as it applies to cases in which compensation is lost in the later months, but when this occurs early I consider that the outlook is extremely grave.

It is apparent in Table X that in only two cases of early breakdown has it been possible to re-establish compensation and to prolong the pregnancy.

Consideration of the Cause of Death in its Relationship to the Method of Treatment.

TABLE XI.

Method of Delivery.	Number.	Died.
Spontaneous Full-time Births	18	0
Spontaneous Premature "	15	3
Spontaneous Miscarriage	2	1
Induction of Premature Labour (Bougies).	10	4
Induction of Abortion	4	1
Medical Induction	1	0
Forceps	14	1
Manual Dilatation and Forceps	2	1
Caesarean section	16 } 20	5 } 5
Miniature Caesarean section	4 }	0 }
Hysterectomy	4	0
Version	1	1
Died during Pregnancy	5	5
Journal incomplete	4	1
	100	23

Spontaneous delivery at term occurred in eighteen instances, three of the patients being primigravidae. Of the fifteen multiparae two were distressed when admitted in labour. Of the remainder the majority had lost compensation during the last two months, while in eight cases symptoms were manifest by the sixth month. Five of the eight cases were admitted on two occasions to the ante-natal wards, and three were in hospital for over two months. Apart from the two patients admitted in labour, all reacted well to treatment and compensation was regained when labour set in. There were fifteen cases of premature spontaneous delivery and compensation was lost in all by the seventh month. Seven of the patients in this group were under ante-natal supervision in hospital and had regained compensation when premature labour commenced. The three patients who succumbed had lost compensation by the sixth month and all were in a serious condition when admitted to hospital. Two died within a few days of acute endocarditis, and the third, admitted in premature labour with severe loss of compensation, died five days after delivery.

Miscarriage occurred in two cases and one of these patients died. Compensation had broken down by the fifth month in both instances and the one who died had experienced urgent symptoms at her two previous pregnancies. The progressive nature of the disease in this case was evident.

Table XI shows that the results obtained from induction of labour by bougies are disastrous, the death rate being no less than 40 per cent. One of the ten patients had a cardio-renal condition and induction was performed more/on account of the urgency of the nephritic symptoms than for cardiac decompensation. Caesarean section was not considered in this instance as pronounced albuminuria is a contra-indication for this operation.

As the introduction of bougies did not promote labour pains the bougies were removed. Three days later, however, the patient delivered herself prematurely. Although the bougies may have been indirectly responsible for the onset of labour, it is possible that this resulted either on account of the albuminuria or from the cardiac distress. For accuracy, therefore, this case should not be included in the group. The death-rate in relation to this operation in the present series is, thus, four out of nine or 44 per cent. The dangers accompanying this operation in patients with heart disease have long been recognised.

Jardine (21) wrote in 1901, "The results from induction are so bad that I am inclined to question the propriety of doing the operation. The risk is exceedingly great".

Herbert Williamson (22) held similar views, thus, "In the later months the danger of collapse and sudden death shortly after delivery renders induction a somewhat risky method of procedure".

Williamson's statement is corroborated in the present series, for of the deaths following induction one died at delivery and two at the fourth and fifth day. The remaining one died later from sepsis.

Robinson in an analysis of 39 cases records the death of two of the four patients in which induction was the method of delivery adopted. One of the two fatal cases had an aortic and double mitral lesion with auricular fibrillation, while the other had an advanced mitral stenosis. The first died two days and the second three days after delivery, which further corroborates Williamson's view of the danger of sudden death following this operation.

Ignoring a mortality rate of 50 per cent, Robinson writes "Induction of premature labour when child is viable and cardiac condition good is a sound precaution for any type of cardiac disease".

Despite the proviso regarding the state of compensation, it is difficult to accept this statement. From my own experience, which seems to be in accordance with that of most observers, induction is a method of treatment for this complication which might well be discarded.

At first sight forceps-delivery seems a favourable method of treatment as it gives only a percentage of seven, but it is well to remember that these patients, with two exceptions, were in labour when admitted, and thus we can conclude that the lesions were not unduly grave. Further, the forceps operation was a simple one in all instances. It is worthy of note that the patient who died after forceps delivery had lost compensation by the sixth month.

Induction of abortion gives a fatality rate of twenty-five per cent and accouchement forcé one of fifty per cent.

In considering the death from version, it is not conclusive evidence as labour in this instance was further complicated by placenta praevia. This patient had, however, the prolonged strain consequent on the expulsion of the breech.

On the other hand, the results obtained from abdominal section have been exceptionally promising. All patients submitted to operation were, without question, in an extremely grave condition. Five of them had been admitted for ante-natal treatment on two occasions during pregnancy. The average duration of the patients in hospital was forty days before operation, the longest period 102 days, and the shortest a few hours. The operations were performed, regardless of the lesion, on patients with auricular fibrillation, praecordial pain, haemoptysis and other symptoms of advanced disease.

The lesion, parity and method of delivery of the complete series have been discussed, and now I shall make particular reference to the fatal section cases.

The Relationship existing betwen the Method of Delivery, the Number and Parity of the Fatal Cases, the Cardiac Lesion, and the Time of Death.

TABLE XII.

Delivery.	Number.		Died.	Lesion.	Time of Death.
Caesarean Section	16		Grav.I.	Aortic & Mitral.	5 days later.
			Grav.I.	Double Mitral.	Immediately.
Miniature Caesarean section	4	24	5 Grav. II.	Double Mitral.	3 days later.
			Grav. I..	Stenosis.	12 hours later (Embolism & secondary suture).
Hysterectomy	4		Grav.XIV.	Mitral regurgitation.	4 days later.
Induction by bougies	9	4	Grav.XI.	Stenosis.	5 days later.
			Grav.VIII.	Double Mitral.	4 days later.
			Grav.III.	Stenosis.	Sepsis later.
			Grav.XII.	Stenosis.	At delivery.
Induction of abortion	4		1 Grav.XII.	Stenosis.	Sepsis later.
Accouchement Force	2		1 Grav.I.	Paroxysmal Tachycardia	14 hours later
Forceps	14		1 Grav.I.	Stenosis.	4 days later.
Premature	14		Grav.VI.	Stenosis.	5 days later.
		3	Grav.XII.	Double Mitral.	Immediately.
			Grav.I.	Double Mitral.	Immediately.
Version	1		1 Grav.VI.	Stenosis.	Few hours later
Miscarriage	2		1 Grav.VI.	Stenosis.	4 days later.
Unknown	1		1 Grav.III.	Mitral Regurgitation.	Unknown.
		18			
			Grav.VIII.	Double Mitral.	
			Grav.III.	Myocardial.	
Undelivered	5	5	Grav.I.	Stenosis.	
			Grav.II.	"Cardiac".	
			Grav.I.	Stenosis.	
		23			

It will be noted that of the twenty-four cases submitted to abdominal section five died - a percentage of 20. This figure is unduly high, as one primigravida who succumbed was moribund on admission and the operation was performed with no hope of success; she only survived a few minutes. In another patient - a multipara - who was a cardio-renal subject, the abdominal wound became infected and eventually gave way. Seven days after the operation it was found necessary to repair the wound by a secondary suture, and she died while under the anaesthetic.

In a recent communication of my own (23) in which a series of seventeen cases delivered by abdominal section was considered, the fatality rate was 11 per cent. My personal experience has been one fatality in thirteen cases - a percentage of seven.

The fatal cases, three of whom were primigravidae, were very gravely ill. One primigravida was in the ante-natal wards for fully three months, and at the time she came into premature labour and was sectioned, compensation was completely lost. This patient had never reacted to treatment. A notable circumstance in her case was the almost entire absence of haemorrhage during the operation. The uterine wall seemed to be devoid of blood. A similar experience was met with on two other occasions and both patients died, although in one instance death did not occur until, suddenly, a week after dismissal from hospital. The operation in this instance was borne well and during convalescence there was no cause for anxiety. (Case 18, Appendix).

It is difficult to account for the anaemia of the uterine wall in these cases. The condition was not attributable to contraction of the uterine muscle, and was obviously the result of deficient blood supply; the musculature looked pale. In contrast to the free haemorrhage which accompanies the operation in ordinary circumstances,

it was difficult to encourage bleeding from the cut surface of the uterus, and, intentionally, no attempt was made either to control the large uterine vessels or to stimulate contractions in the organ. It is possible that the anaemia is to be ascribed to syncope as a result of the anaesthesia.

Another primigravida died from an embolism twelve hours after the operation. She appeared to have become pregnant when compensation was already lost, and had suffered from several attacks of pulmonary oedema during the preceding six months.

Of the remaining fatal cases reference has already been made to the moribund condition of one when submitted to operation, and of the misfortune which necessitated a second operation in another. The remaining patient (Case 22, Appendix), Gravida II, Aged 36, had chorea when 14 years of age, and had remained well until her first pregnancy about two years previously. This terminated as a premature spontaneous birth, and thereafter she had been attending a General Hospital more or less constantly on account of her cardiac condition. She again became pregnant and at the sixth month was sent to the Maternity Hospital. On admission she was profoundly distressed, with severe dyspnoea and orthopnoea, but there was no evidence of oedema. The urine was loaded with albumen, the Esbach being 12. The cardiac lesion was a double mitral one, and there was great irregularity of the heart's rhythm; the blood pressure was 120. It was afterwards ascertained that she had a positive Wassermann reaction, but it was decided that this had no bearing on the cardiac lesion as there was a definite history of chorea in childhood. Her condition improved with rest and simple treatment and the pregnancy was not terminated immediately owing to her anxiety to have a living child. After a month in hospital premature labour unfortunately supervened and her symptoms became acute. Caesarean section was

performed, and although in a precarious state, she survived the shock of the operation but died a few days later with complete loss of compensation.

This case resembles that of one of the primigravidae who succumbed (Case I, Appendix). Both remained well until pregnancy occurred, after which they required more or less constant supervision in Hospital. Each expressed the wish that the pregnancy should not be terminated for the sake of a living child. A note of warning has already been given regarding the danger of thus yielding to sentiment in certain cases. Again, in both instances the operation was performed during premature labour when compensation was completely lost. Death occurred on the fourth and fifth day respectively.

Two outstanding features are worth recording in connection with the fatal section cases. In the first place, compensation was lost in all instances by the sixth month, and, in the second place, four of the five patients had lost compensation and were in labour when the operations were performed.

Table XII further demonstrates that in cardiac cases the time of occurrence of death following parturition is rather striking. The greater number either died within a few hours after delivery, or on the fourth or fifth day. Both cases who developed sepsis died during the second week.

The danger of syncope either immediately following parturition or on the fourth or fifth day of the puerperium is a matter of common knowledge. It is attributable to engorgement and dilatation of the right heart, which, in consequence, becomes paralysed. It is, therefore, desirable in cardiac cases to encourage haemorrhage in the third stage of labour as otherwise the blood which is thrown back on the circulation on the completinn of labour is returned to the right heart.

Jardine used to lay great stress on this danger and pointed out the necessity of opening a vein should urgency demand it.

Regarding this liability to syncope, it will be of interest to know that apart from the fatal cases, none of the Caesarean or hysterectomy patients gave the slightest cause for alarm during the puerperium.

From the foregoing observations and figures I feel that a strong claim for Caesarean section as a method of treatment in cardiac disease has been established.

As already stated, the operation has been performed successfully on patients who have had the gravest of lesions, and most urgent of symptoms, but in these instances the patient was submitted to operation before she was in labour. Considerable judgment is necessary in deciding the most suitable time to operate. If the cardiac condition has improved with rest, the operation may be carried out although urgent symptoms such as dyspnoea and orthopnoea are present. Should premature labour supervene while compensation is lost, or should grave symptoms arise as a result of the strain of labour, the outlook is ominous. It is doubtful in these cases if Caesarean section is of any advantage other than that it entails a rapid evacuation of the uterus. It has been shown that four of the five fatal cases were in labour when the section was performed, and apart from the one who succumbed from an embolism, the others, although they survived the operation, died within a few days. It is probable that these patients would have died during labour if this had been allowed to proceed. The fifth fatal case had pronounced albuminuria and reference has been made to the inadvisability of performing an abdominal operation in patients thus affected.

The ideal time for operation is after a prolonged period of rest and before labour commences. If circumstances permit it is desirable to delay operation until there is reasonable hope of the child surviving. Certain of the patients under discussion have been carried on by complete rest and simple treatment, viz., 5 m. of strophanthus four hourly, with special attention to diet and the regulation of the bowels. Jardine recommended this treatment as a routine. With auricular fibrillation digitalis is substituted for strophanthus.

One such patient, who had been in bed since her last confinement three months previously, was admitted to one of the infirmaries with loss of compensation; after a few months she was found to be pregnant and was transferred to the Maternity Hospital. On compensation being regained, she insisted on leaving the Hospital only to return a month later with a recurrence of breakdown. She was ultimately sectioned and sterilised, and was dismissed well with a live child. Prior to that she had been confined to her bed for a year.

Again, although the results from hysterectomy have been excellent similarly good results have been obtained from miniature Caesarean section, and in the latter operation there is probably less shock, and the physiological function of the uterus has been conserved. No patient who was submitted to either operation died.

The ultimate object of preventing future conception is achieved by sterilisation, and the mental effect the fear of future pregnancy has on many of these patients is well known.

At the same time, owing to the progressive nature of the disease and to the increasing damage which results with each succeeding pregnancy, it is desirable to prevent future pregnancies.

The desirability of performing Caesarean section and sterilization in primigravidae may be questioned, but it seems justifiable to adopt this procedure when a patient experiences grave symptoms in her first pregnancy. I do not contend that the operation should be carried out as routine. On the contrary, if compensation breaks down in the late months there is every expectation of a favourable termination by spontaneous delivery, or preferably by forceps. The position, however, is different when breakdown occurs early, and Table X demonstrates that in such cases it was possible in only two instances to prolong the pregnancy; in one of these the birth was premature.

Many authorities contend that in the gravest cases the uterus should be emptied in the early months, and by the quickest possible method. In miniature Caesarean section we have an excellent and safe way of doing so.

Paramore[24] has suggested that when Caesarean section has been performed in the treatment of cardiac cases the operation should be followed by hysterectomy, as this procedure would preclude the possibility of further damage to the heart should the products of involution become infected. MunroKerr considers that this danger requires investigation as fresh vegetations are frequently found on the valves at autopsies.

With the object of investigating the bacterial content of the uterus during the puerperium, I procured a long glass tube shaped with a pelvic curve, through the bore of which ran a wire surrounded by a sterile swab. On the third or fourth day after delivery the tube was inserted directly through the cervix and the swab was introduced into the cavity of the uterus by pressure on the outer end of the wire

which projected beyond the tube. The specimen was then withdrawn into the tube. By this method four swabs were procured, but difficulty was experienced in passing the tube in three instances as the operation had been performed before the patient was in labour, and the cervix, consequently, was not well dilated. The fourth swab was taken from a patient who had been delivered with forceps. Owing to the dearth of material at present I have been unable to augment this number. The danger of introducing infection and of exciting these patients to their detriment, has also to be taken into consideration.

The first swab was taken from a patient who, unfortunately, died;(Case 22, Appendix). A direct film was found to contain very numerous gram positive diphlococci, probably pneumococci.

The second swab was obtained from a patient delivered by Caesarean section, and the third from the case delivered with forceps, and from both were grown colonies of the staphylococcus albus.

From the fourth swab a few streptococci were incubated. These were probably benign, as the patient had an uneventful convalescence. No information of any great significance has been obtained from this investigation.

The first swab was taken from a patient who was extremely ill following operation and who developed urgent pulmonary symptoms and died. The presence of these organisms, therefore, was probably of haemotogenic origin. Had hysterectomy been performed in this case the source of a future acute endocarditis would not have been eradicated even if the patient had recovered.

In the second case, the patient had a slight temperature during the first week of convalescence, but was dismissed apparently well on the twenty-first day following operation. After ten days at

home she was admitted to a Medical ward in a General Hospital with a recurrence of urgent cardiac symptoms and died three weeks later. As a post-mortem examination was refused, the condition of the heart is unknown. Clinically there was nothing from her symptoms to suggest that acute endocarditis was responsible for the fatal termination.

The remaining cases from which swabs were obtained had uneventful puerperia and were dismissed well.

As against Paramore's theory, several of the patients who have been submitted to Caesarean section have been followed up, and the results are promising. One patient whose pregnancy was terminated by miniature section three years ago (Case 5, Appendix) has enjoyed good health since. Her doctor writes "she is very hale and well. Her operation made a great improvement on her, and her heart condition seemed to improve very much afterwards. She is able for any kind of light work, in fact, I do not remember seeing any case where general improvement was so marked".

It is probable that this woman's health is benefited from the knowledge that she is sterilised.

Six other patients who were submitted to Caesarean section during the last three years report from time to time, and all keep reasonably well. One patient, however, died suddenly at home about a week after dismissal from hospital.

Two patients on whom hysterectomy was performed during the same period also enjoy good health.

In several other instances the patients have not been traced.

From a general consideration of these cases there seems to be no apparent advantage in performing hysterectomy, and, on the contrary, there is the disadvantage that the operation produces more shock.

Consequently, as I do not consider that Paramore's theory regarding a possible danger from the products of involution has been established, I contend that hysterectomy is unnecessary.

The operation of vaginal Caesarean section in the early months has been recommended as a rapid method of emptying the uterus. By this method sterilisation may be difficult, more especially in primigravidae.

With regard to the technique of the Caesarean operation, the classical incision has been used in all instances. It is desirable to have dilators at hand lest the cervix be closed when the operation is performed before term. Otherwise drainage may be interfered with. The anaesthetic used in all instances has been chloroform, ether being contraindicated owing to the susceptibility of these cases to pulmonary oedema. It has been found necessary on occasion to anaesthetize the patient in a sitting posture on account of the urgent orthopnoea. Under the influence of the anaesthetic she can be placed in the dorsal position without respiratory embarrassment. Almost immediately following the evacuation of the uterus, the breathing becomes relieved, and patients who may have been unable to lie flat prior to operation can do so within twenty-four hours. MacKenzie recommends chloroform and states that he gave it to all his midwifery patients suffering from cardiac disease, even to full anaesthesia, and never had any trouble. This has also been my experience.

I contend, therefore, that Caesarean section with sterilisation is the best method of treatment for dealing with the graver cases of cardiac disease when complicated by pregnancy.

GENERAL CONCLUSIONS

As a result of my observations on the foregoing series of 100 cases of pregnancy complicated by cardiac disease I have reached the following conclusions:-

1. There is a greater liability for compensation to be lost in the earlier pregnancies especially in the first and second.

2. In primigravidae, compensation tends to be lost in the later months, but as the number of pregnancies increase urgent symptoms appear at a progressively earlier period. To some extent this is dependant on the severity of the cardiac disease, and is specially noticeable in patients with mitral stenosis.

3. There is undoubtedly an increasing strain with each succeeding pregnancy but in the milder forms of the disease this may not be apparent until after a long series of pregnancies.

4. A greater number of patients lose compensation during the sixth month than at any other period of pregnancy.

5. Of all types of heart disease, mitral stenosis is most to be feared and occurs approximately in 60 per cent of cases: a very small minority of patients with this lesion reach term without cardiac symptoms of some description.

6. Mitral regurgitation is a much less serious form of the complaint, and provided the myocardium is healthy this lesion can, as a rule, be regarded without anxiety.

7. Rheumatic fever is by far the most common factor in the causation of cardiac disease.

8. When the disease is of recent origin the symptoms are more urgent and the prognosis is most unfavourable.

9. The danger of acute endocarditis being superimposed on the heart valves is present, but is of infrequent occurrence during pregnancy.

10. Relative to other lesions mitral stenosis shows a much greater liability to abortion or premature labour.

11. The death rate from mitral stenosis is almost three times that of all other lesions combined.

12. As aortic disease is comparatively rare it is difficult to arrive at any definite conclusion regarding this lesion. It appears however to be a less serious condition than mitral stenosis.

13. With regard to the delivery of patients with cardiac disease, fully half of them require operative interference, or mechanical aid of some description.

Conclusions as to Prognosis.

1. When, regardless of the parity of the patient, compensation remains good throughout pregnancy, or when distress only appears in labour, a favourable result is to be expected.

2. When loss of compensation occurs from approximately the thirty second week onwards, there is every probability of a successful termination to the pregnancy provided the patient is kept at rest under strict supervision. There is however a tendency to premature labour.

3. If compensation is lost in the late months and the condition is neglected the patient will be in grave danger when labour sets in.

4. In primigravidae, if compensation is lost by mid-term the prognosis is very grave. Should decompensation persist in spite of treatment a fatal issue is to be expected. If compensation can be restored and the pregnancy is terminated immediately the outlook with regard to the mother is good. On the other hand if the pregnancy is prolonged there is the grave danger of a relapse with the probable death of the mother, and in any event miscarriage or premature labour is almost certain to occur.

5. In multigravidae, if compensation is lost in the early months the prognosis though grave, generally is less serious than in primigravidae: there is a greater likelihood of compensation being restored and maintained until such time as the child is likely to survive. The prognosis in these cases is influenced by the parity of the patient, the nature and extent of the cardiac lesion, the degree of decompensation, and the behaviour of the heart at previous pregnancies.

6. If prior to the present pregnancy there has been a recent history of urgent cardiac symptoms, the outlook is ominous unless the pregnancy is terminated.

7. If labour supervenes while compensation is lost the prognosis is most unfavourable.

Conclusions as to Treatment.

1. When compensation remains good throughout pregnancy and in labour the case may be regarded without anxiety and no special treatment is called for.

2. If urgent symptoms first make their appearance in labour, forceps should be applied at the end of the first stage.

3. If the patient does not experience cardiac distress until the later months complete rest will probably restore compensation, and forceps should be applied in labour.

4. When compensation is lost in the early months treatment is influenced by the parity of the patient. (a) In primigravidae, the pregnancy should be terminated after a short period of rest, and the patient sterilised. This can best be achieved by abdominal section. (b) In multiparae, the extent of the cardiac disease and the behaviour of the heart at previous pregnancies will have to be considered. Immediate evacuation of the uterus with sterilisation may be advisable, or the pregnancy may be allowed to proceed until the child is viable. In the latter event section and sterilisation should be performed before labour sets in.

5. Induction of abortion or of premature labour is a dangerous procedure which might well be discarded as a method of treatment in cardiac disease. The former though less dangerous than the latter is nevertheless accompanied by considerable shock, and in both, the obvious indication for sterilisation is ignored.

6. Accouchment forcé is to be condemned.

7. In the graver manifestations of the disease the best results are obtained by Caesarean section and sterilisation. In the early months it is an excellent and safe way of evacuating the uterus when the condition demands it.

8. No advantage is to be gained by performing hysterectomy.

9. The knowledge of being sterilised adds much to the peace of mind of women suffering from heart disease.

10. Should compensation be lost and the patient comes into labour practically no advantage is to be gained by performing Caesarean section. The prognosis is very bad.

11. Chloroform should be the anaesthetic of choice.

A P P E N D I X

Case 1. Gravida 1, admitted at 4th month: double mitral lesion and aortic regurgitation: on admission compensation lost: haemoptysis: in hospital 102 days before operation: Compensation I.S.Q. at operation: Caesarean section: died.

Case 2. Gravida 3, admitted at 3rd month: double mitral lesion: on admission compensation lost: in hospital 63 days (2 admissions) before operation: compensation regained at operation: Caesarean section: dismissed well.

Case 3. Gravida 2, admitted at 6th month: mitral stenosis: on admission compensation lost: in hospital 60 days before operation: compensation regained at operation: Caesarean section: dismissed well.

Case 4. Gravida 1, admitted at 4th month: mitral stenosis: on admission compensation lost: pulmonary oedema: in hospital 90 days before operation: compensation lost at operation: Caesarean section: died 12 hours later from embolism.

Case 5. Gravida 6, admitted at 3 months: mitral stenosis: on admission compensation lost: in hospital 42 days before operation: compensation regained at operation: Caesarean section (5th month): dismissed well.

Case 6. Gravida 8, admitted at 3 months: mitral stenosis: on admission cardiac asthma: in hospital 21 days before operation: condition improved at operation: hysterectomy: dismissed well.

Case 7. Gravida 1, admitted at 3 months: double mitral lesion: on admission dyspnoea praecordial pain: in hospital 41 days before operation: condition improved at operation: hysterectomy: dismissed well.

Case 8. Gravida 4, admitted at 7 months: mitral regurgitation: on admission compensation lost - orthopnoea, haemoptysis: in hospital 36 days before operation: condition improved at operation: Caesarean section early 9th month - not in labour: dismissed well.

Case 9. Gravida 3, admitted at 2½ months: mitral stenosis: on admission severe dyspnoea: in hospital 30 days before operation: condition improved at operation: Caesarean section (3½ months): dismissed well.

Case 10. Gravida 7, admitted at 8 months: mitral stenosis and aortic regurgitation: on admission severe orthopnoea - breathlessness for 3 months before admission: in hospital 28 days before operation: condition improved at operation: Caesarean section: dismissed well.

Case 11. Gravida 3, admitted at 5 months: mitral stenosis: on admission compensation lost: in hospital 46 days (2 admissions) before operation: compensation regained at operation: Caesarean section: dismissed well.

Case 12. Gravida 4, admitted at 5½ months: double mitral lesion: on admission compensation lost: in hospital 60 days before operation: condition improved - orthopnoea at operation: Caesarean section 7½ months - not in labour: dismissed well.

Case 13. Gravida 2, admitted at 8 months: mitral stenosis: in bed for 4 months before admission: in hospital 14 days before operation: condition at operation not improved - severe orthopnoea: Caesarean section 8½ months - not in labour: dismissed well.

Case 14. Gravida 2, admitted at 4½ months: mitral stenosis: compensation lost - auricular fibrillation - in bed for 3 months before admission: in hospital 45 days before operation: Caesarean section (6 months) dismissed well.

Case 15. Gravida 2, admitted at 3 months: mitral stenosis: on admission orthopnoea - transferred from medical ward for operation: in hospital 20 days before operation: condition at operation I.S.Q.: hysterectomy: dismissed well.

Case 16. Gravida 1, admitted at 7 months: double mitral lesion: on admission compensation lost - orthopnoea, oedema: in hospital 17 days before operation: condition improved - no oedema at operation: Caesarean section - not in labour: dismissed well.

Case 17. Gravida 7, admitted at 3 months: mitral stenosis: on admission severe orthopnoea - auricular fibrillation: in hospital 20 days before operation: condition improved at operation: hysterectomy: dismissed well.

Case 18. Gravida 7, admitted at 5 months: double mitral lesion: on admission, orthopnoea, oedema-auricular fibrillation: in hospital 60 days before operation (two admissions): condition improved at operation: Caesarean section - not in labour: dismissed well after uneventful convalescence: died suddenly at home 1 week later.

Case 19. Gravida 14, admitted at 8 months: mitral regurgitation: on admission cyanosis, orthopnoea - cardio-renal: in hospital 4 days before operation: Caesarean section: wound sepsis - secondary suture: died during second operation.

Case 20. Gravida 2, admitted at 5 months: on admission orthopnoea, tachycardia, myocarditis: in hospital 19 days before operation: condition improved at operation: miniature Caesarean section: dismissed well.

Case 21. Gravida 5, admitted at 7 months: double mitral lesion: on admission, dyspnoea, orthopnoea, extensive oedema: in hospital 30 days before operation: Caesarean section - not in labour: dismissed well: died three weeks later in a medical ward from cardiac disease.

Case 22. Gravida 2, admitted at 6 months: double mitral lesion: on admission dyspnoea and orthopnoea: urine - Esbach 12: Wassermann ++ : in hospital 33 days before operation: premature labour - compensation lost - Caesarean section: died.

Case 23. Gravida 1, admitted at $7\frac{1}{2}$ months: double mitral lesion: on admission, moribund: Caesarean section: died immediately.

Case 24. Gravida 7, admitted at 6 months: mitral stenosis: on admission dyspnoea, orthopnoea, oedema: in hospital 78 days before operation (two admissions): condition improved at operation: Caesarean section early 9th month - not in labour: dismissed well.

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