# Staff Meeting Bulletin Hospitals of the » » » University of Minnesota



# Heart Disease in Pregnancy

Volume XIX | Friday, December 12, 1947 | Number 10

STAFF MEETING BULLETIN
HOSPITALS OF THE . . .
UNIVERSITY OF MINNESOTA

Volume XIXI | Friday, December 12, 1947 | Number 10

INDEX



Published for the General Staff Meeting each week
during the school year, October to June, inclusive.

I.

# UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
## CALENDAR OF EVENTS

December 15 - December 20, 1947

No. 181

Monday, December 15

9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; Interns' Quarters, U. H.

9:15 - Fracture Rounds; A. A. Zierold and Staff; Ward A: Minneapolis General Hospital.

10:00 - 12:00 Neurology Ward Rounds; A. B. Baker and Staff; Station 50, U. H.

11:00 - 11:50 Physical Medicine Conference; Physical Therapy in Fractures; M. E. Knapp; E-101, U. H.

11:00 - 11:50 Roentgenology-Medicine Conference; Staff; Veterans' Hospital.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and D. State; Eustis Amphitheater, U. H.

12:15 - 1:20 Pediatrics Seminar; Penicillin Therapy; Theodore Smith; 6th Floor Seminar Room, U. H.

12:15 - 1:20 Obstetrics and Gynecology Journal Club; M-435, U. H.

12:30 - 1:20 Pathology Seminar; Congenital Pneumonia; Sheldon Siegal; 104 I. A.

12:30 - 1:50 Surgery Grand Rounds; A. A. Zierold, Clarence Dennis and Staff; Minneapolis General Hospital.

Tuesday, December 16

8:30 - 10:20 Surgery Reading Conference; Lyle Hay; Small Conference Room, Bldg. I., Veterans' Hospital.

9:00 - 9:50 Roentgenology Pediatrics Conference; L. G. Rigler, I. McQuarrie and Staff; Eustis Amphitheater, U. H.

10:30 - 11:50 Surgical Pathological Conference; Lyle Hay and Nathanial Lufkin; Veterans' Hospital.

12:30 - 1:20 Pathology Conference; Autopsies; Pathology Staff; 102 I. A.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III, Veterans' Hospital.

3:15 - 4:20 Gynecology Chart Conference; J. L. McKelvey and Staff; Station 54, U.H.

3:30 - 4:20 Clinical Pathological Conference; Staff; Veterans' Hospital.

4:00 - 5:30 Surgery-Physiology Conference; O. H. Wangensteen and M. L. Visscher; Eustis Amphitheater, U. H.

5:00 - 5:50 Roentgenology Diagnosis Conference; Oscar Lipschultz and Staff of the General Hospital; M-515, U. H.

Wednesday, December 17

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-515, U. H.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker and Joe R. Brown; Veterans' Hospital.

11:00 - 11:50 Pathology-Medicine-Surgery Conference; Carcinoma Head of the Pancreas; E. T. Bell, O. H. Wangensteen, C. J. Watson and Staff; Todd Amphitheater, U. H.

4:00 - 5:00 Infectious Disease Routes; Todd Amphitheater, General Hospital, Veterans' Hospital.

Thursday, December 18

8:15 - 9:00 Roentgenology-Surgical-Pathology Conference; Walter Walker and H. M. Stauffer; M-515, U. H.

8:30 - 10:20 Surgery Grand Rounds; Lyle Hay and Staff; Veterans' Hospital.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:30 - 11:50 Surgery-Radiology Conference; Daniel Fink and Lyle Hay; Veterans' Hospital.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and D. State; Eustis Amphitheater, U. H.

1:00 - 1:50 Fracture Conference; A. A. Zierold and Staff; Minneapolis General Hospital.

1:30 - 3:00 Pediatric Psychiatric Rounds; Reynold Jensen; 6th Floor West Wing, U. H.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hansen and Staff; E-534, U. H.

5:00 - 5:50 Roentgenology Seminar; Report of the Radiological Society of North America; M-515, U. H.

7:00 - 8:00 Urology-Roentgenology Conference; H. M. Stauffer and George Eaves; M-515, U. H.

Friday, December 19

8:30 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphi., U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphi., U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:30 - 11:20 Medicine Grand Rounds; Staff; Veterans' Hospital.

10:30 - 11:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

11:00 - 12:00 Surgery-Pediatric Conference; C. Dennis, A. V. Stoesser and Staffs; Minneapolis General Hospital.

1:00 - 1:50 Dermatology and Syphilology; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U. H.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

3:00 - 3:50 Surgery Literature Conference; Clarence Dennis and Staff; Minneapolis General Hospital.

Saturday, December 20

7:45 - 8:50 Orthopedics Conference; Wallace H. Cole and Staff; Station 21, U. H.

8:00 - 9:30 Psychiatry and Neurology Grand Rounds; Staff; University Hospitals.

9:00 - 9:50 Surgery-Roentgenology Conference; O. H. Wangensteen, L. G. Rigler, and Staff; Todd Amphitheater, U. H.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; M-515, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; M-515, U. H.

10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U. H.

## II. HEART DISEASE IN PREGNANCY

John S. Gillam

The purpose of this study is to demonstrate the effects of pregnancy on organic heart disease. Since 1939, there have been 100 patients with organic heart disease treated on the Obstetrical Service of this hospital. Seventy-two of these patients had rheumatic heart disease. Twenty-three patients had congenital heart disease, and the remaining give cases were complicated by miscellaneous forms. Two patients of this latter group had hypertensive heart disease established prior to the onset of the present pregnancy, 2 had thyrotoxic heart disease, and the last patient had chronic pulmonary hypertension. The abnormally large number of cases with congenital heart disease reflects the type of material referred to the hospital and in addition the interest several members of the staff have in this condition. These 23 cases of congenital heart disease and pregnancy have been published elsewhere.

The criteria used to establish the reality of the heart disease were strict. The patients with congenital heart disease were in almost all cases examined in the non-pregnant state, and the diagnosis of congenital heart disease was agreed upon by at least 2 of the cardiologists. All cases had distinctly abnormal findings. In cases where the true nature of the lesion was in doubt, the opinion of the senior cardiologists was used. In the miscellaneous group definite cardiac enlargement in the non-pregnant stste was the essential criterion. These cases were classified according to the organic lesion or condition producing the heart disease. This group undoubtedly could be greatly expanded were one to include questionable cases.

The most important criterion for diagnosis in the group of rheumatic patients was a diastolic murmur. Sixty-three of the 72 patients showed this. Three patients had loud, coarse, apical systolic murmurs and definite x-ray evidence of both left atrial and pulmonary conus enlargement. The remaining group of 6 patients, all with a history of at least one attack of rheumatic heart disease, had only loud coarse apical systolic murmurs, typical of a simple mitral insufficiency, but, even though the murmurs were in all cases present in the non-pregnant state, they were classified only as probable rheumatic heart disease. Organic involvement could not be definitely proven, so these 6 were not used below in statistical studies. Two of the 6 had dyspnea on moderate exertion. By use of the above criteria, 42 patients with an original diagnosis of organic heart disease were excluded from the study, leaving the 100 patients here reported. The 100 patients were seen in 103 pregnancies in this hospital, and they had 18 pregnancies subsequent to their original admission here in other hospitals. Table I shows the distribution of the patients as to specific nature of the lesion.

The clinical record of each patient was reviewed for the following information which is held to be of some clinical use in the management of the pregnant cardiac patient: age; parity; followed or not in the Obstetric Clinic during the pregnancy; functional classification (using the classification of the New York Heart Association) at the time when first seen, during the non-pregnant state, during the period of peak load after the 28th week, at the time of delivery, during the puerperium, and class during the follow-up study (later than 3 months post partum); year in which the patient was treated; if and when the patient received digitalis; weight gain during pregnancy; blood presure; presence or absence of upper respiratory infection during the pregnancy; that period of time, regardless of duration, during which the load of pregnancy was most apparent; the week of gestation of delivery (all patients who delivered term sized infants were said to have delivered during the 40th week; otherwise the period of gestation was measured from the date of the last menstrual period); the method of the onset of labor; the total duration of labor and the duration of the second stage of labor; the method of delivery; anesthesia during labor and delivery; weight of the infant; survival of the infant; rheumatic fever history; the ante-, intra, and post-partum pulse and respiratory rates if at any time more than 100

TABLE I

DISTRIBUTION OF 94 CASES (6 PROBABLES EXCLUDED)

| | | | |
|---|---|---|---|
| A. | Rheumatic heart disease | 66 | |
| | 1. Mitral stenosis with/without mitral insufficiency | 50 | (2 of which stenosis was doubtful) |
| | 2. Mitral stenosis and aortic regurgitation and/or stenosis | 11 | |
| | 3. Aortic insufficiency and/or aortic stenosis | 5 | (2 of which had a ? mitral lesion also) |
| B. | Congenital heart disease | 23 | |
| | 1. Patent ductus arteriosus | 9 | |
| | Patent ductus plus possible patent interauricular septum | 1 | |
| | 2. Interventricular septal defect | 6 | |
| | a. Plus pulmonary arteriosclerosis | 1 | |
| | b. Plus pulmonary stenosis | 1 | |
| | 3. Patent interauricular septum | 5 | |
| | Possible Leutenbacher syndrome | 2 | |
| | 4. Pulmonary stenosis and possible septal defect | 1 | |
| | 5. Type undetermined | 2 | |
| C. | Miscellaneous | 5 | |
| | 1. Hypertensive cardiovascular disease | 2 | |
| | 2. Thyrotoxic heart disease | 2 | |
| | 3. Chronic pulmonary hypertension | 1 | |

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and 20 respectively on three separate observations; complications other than the heart disease; special features in the individual cases; cardiac findings as reported by members of the staff of the Department of Internal Medicine either in the out-patient or in-patient departments; laboratory data on blood, urine, blood chemistries, vital capacity, venous pressures, circulation time, and blood volume in the few cases where determined; sterilization if done; and finally what were apparently errors, either by the patient or physicians caring for her. Much data obtained has not been discussed below, being beyond the scope of this presentation.

The use of the classification of cardiac patients during the various stages of pregnancy to determine the effect which the pregnancy has upon cardiac reserves has not been previously reported. Most authors[9,12,15,16] have simply reported the functional class as determined when the patient was first seen as we have done in Table II. As can be seen here, 35 of the patients were class 3, and 13 were class 4. One of the class 3 patients was decompensated, with hepatomegaly, marked edema, diffuse pulmonary rales, and pulmonary edema and congestion by x-ray, yet had no symptoms at rest. All of the class 4 patients had left heart failure, making a total of 14 who were decompensated when first seen.

TABLE II

FUNCTIONAL CLASS WHEN FIRST SEEN IN THE PRESENT PREGNANCY

| Functional Class | Preg. Total | Congen-ital | Rheumatic Poss | Rheumatic Actual | Miscel. | Registered | Unregistered |
|---|---|---|---|---|---|---|---|
| Class 1 | 35 | 9 | 4 | 21 | 1 | 33 | 2 |
| Class 2 | 40 | 9 | 2 | 29 | 0 | 29 | 11 |
| Class 3 | 15 | 5 | 0 | 9 | 1 | 5 | 10 |
| Class 4 | 13 | 1 | 0 | 9 | 3 | 0 | 13 |
| TOTALS | 103 | 24 | 6 | 68 | 5 | 67 | 36 |

TABLE III

TYPE OF LESION AND FUNCTIONAL CHANGES IN 24 PREGNANCIES COMPLICATED BY CONGENITAL HEART DISEASE IN 23 PATIENTS

| Type of Lesion | Worsened | Remained Same | Total |
|---|---|---|---|
| Patent ductus arteriosus | 6 | 3 | 9 |
| Patent interventricular septum | 2 | 5 | 7 |
| Patent foramen ovale | 0 | 5 | 5 |
| Pulmonary stenosis | 0 | 1 | 1 |
| Type undetermined | 1 | 1 | 2 |
| TOTALS | 9 | 15 | 24 |

Table III shows changes of functional class in patients with congenital heart disease. It appears as if patent ductus arteriosus and interventricular septal defect are the serious congenital lesions seen in the patients who survive to a child-bearing age. In these same 2 lesions another interesting abnormality was noted by Lund in these patients.[10] Six of the 16 patients showed an increase in the systolic and pulse pressures during labor. The systolic pressure rose over 140 in these 6 and in 3 of these it exceeded 160 mm Hg. However, the diastolic pressure rose over 90 in only one case. The blood pressure fell precipitously in all 6 at the time of delivery, 2 patients actually going into deep shock without apparent obstetric cause. One of these 2, the same patient whose pressure was over 90 mm Hg. diastolic, died in shock. The explanations are theoretical. Hamilton has also noted the peculiar pressure changes.[7] Two patients have subsequently had successful ligations of the patent ductus. The other interesting features of this group have been reported elsewhere.[10] With the exception of the already noted changes, there was no demonstrable difference clinically between the courses of patients with congenital heart disease and those with rheumatic heart disease. The maternal mortality in this group was 4%. The 5 patients with the miscellaneous types of heart disease are too small a group from which to draw any conclusions. With these reservations in mind, and in order to keep the groups as identical as possible, further discussion, for the most part, has been limited to those patients with rheumatic heart disease.

In Table 4 the functional class changes over the non-pregnant state have been summarized. Slightly more than 50%

TABLE IV

SUMMARY OF CLASS ADVANCE IN RHEUMATIC HEART DISEASE OVER NON-PREGNANT STATE

| | | |
|---|---|---|
| Remained the same | 34 | 46% |
| Advanced 1 class | 24 | 32% |
| Advanced 2 classes | 11 | 15% |
| Decompensated | 16 | 21% |
| Died | 5 | 6.7% |
| Totals | 74 | |

showed advanced in symptomatology during the pregnancy-puerperal period and 20% decompensated. Seven per cent died, three times the expected mortality in non-pregnant compensated patients with rheumatic heart disease, but equal to the expected yearly mortality of those patients who had decompensated.[8] We have considered one year, the 9 months of pregnancy and the first three months of the puerperium, to constitute that period of time during which the pregnancy per se is exerting an influence. To my knowledge, there is no report stating the changes in functional classification of a group of non-pregnant women of the same age group who have rheumatic heart disease during the same length of time. Even in the absence of such a control, it is fairly safe to assume that during the year of the pregnancy, the pregnant patients will show an increase in frequency and magnitude of functional class changes. But fortunately this is not the problem with which we should really be concerned. We must determine whether a group of women who have heart disease, if they can be carried through a pregnancy, will either have materially shortened their lives or decreased materially their functional capacities because of their pregnancies. But first the immediate problems associated with the pregnancy will be discussed.

The uncorrected 6% mortality here reported is somewhat higher than some clinics report.[1,5-9,12,15,16] The mortality for registered patients is no higher. The mortality here from subacute bacterial endocarditis is higher than most authors show. Until recently, this mortality was uncontrollable. No significant difference between the mortality here and the maternal mortality as reported from other teaching institutions could be demonstrated.

One aim of the study was to determine factors which would have prognostic significance in the individual patient. This meant testing statistically the numerous factors thought to be of importance by others, as well as some of our own. Few indeed were the factors which stood these tests, but it must be said in defense of the other authors, that with a much larger series, many would most certainly be better able to do so.

The age of the patient has been noted by most observers to be of significance.[1,8,etc.] In our study, as is seen in Table V and Table VI, it is of considerable importance. Eleven per cent of the patients under 30 decompensated and 67% of those over 30 decompensated. This is a highly significant difference. The difference between the age groups 25-29 years and 30-34 years is also significant.

Table V

AGE DISTRIBUTION IN THE 103 PREGNANCIES

| Age Group | No. of Cases | No. who Decomp. | % Decomp. |
|---|---|---|---|
| Less than 20 | 12 | 0 | 0% |
| 20-24 | 42 | 2 | 4.8% |
| 25-29 | 28 | 7 | 25% |
| 30-34 | 12 | 7 | 58% |
| 35-39 | 6 | 4 | 67% |
| 40 and over | 3 | 3 | 100% |

TABLE VI

| | Decompensated | Did not | Totals | |
|---|---|---|---|---|
| Patients less than 30 years | 9 | 73 | 82 | 11% |
| Patients over 30 years | 14 | 7 | 21 | 67% |
| Totals | 23 | 80 | 103 | |

$X^2 = 32.5$

Probability: Less than .0005

| | | | | |
|---|---|---|---|---|
| Patients between 25-29 years | 7 | 21 | 28 | |
| Patients between 30-34 years | 7 | 5 | 12 | |
| Totals | 14 | 26 | 40 | |

$X^2 = 4.2$

Probability: .046

- - -

In our patients parity was not of significance in producing decompensation. In addition, by studying parity figures in over 2500 cases reported in the literature, it also could not be demonstrated to effect the decompensation rate. Table VII is a summary of the findings in our series. As is obvious we cannot demonstrate statistically here, nor has any other author to my knowledge, that

TABLE VII

PARITY

| Entire Group | Decompensated | % |
|---|---|---|
| Primipara | 7 | 16 |
| Multipara | 16 | 28 |
| Under 30 | | |
| Primiparas | 6 | 14 |
| Para 1-3 | 3 | 9.4 |
| Para 4-7 | 0 | 0 |
| Over 30 | | |
| Primipara | 1 | 33 |
| Para 1-3 | 5 | 71 |
| Para 4-8 | 8 | 72 |

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parity plays a role in the mortality or decompensation rates in pregnancy. All of us recall, however, the oft quoted dictum of "a babe in the crib is more work for the maternal heart than one in utero". Perhaps true, but not proven. We are studying this problem now as part of our follow-up program, which is discussed below.

There was a history of rheumatic fever or chorea in 70% of our patients with rheumatic heart disease. Seventeen per cent of the patients with congenital heart disease also gave a history of one or the other. Only 16 patients in the group with rheumatic heart disease gave no history, yet 4 of the 5 deaths in this rheumatic group had no history of rheumatic fever. This diagnosis was confirmed at autopsy in all those who died.

OBSTETRICAL FEATURES:

The average duration of labor for the primiparous patients was 11.06 hours and for the multiparous patients was 8.91 hours. These are almost identical to durations of labor in a series of 400 normal deliveries in this hospital in normal patients used as a control series.

From Table VIII it appears as if those delivered per vaginam fared better than those delivered by the abdominal route. This is well supported in the literature of recent years. The reasons for the improved results with the vaginal deliveries are unexplained. Delivery from below is one of the few recent contributions in the management of pregnant cardiac patients.[5-8,12,14-16] There has not appeared in the literature of the last ten years a single paper that advocates cesarean section for cardiac reasons, while prior to that time the reverse was almost true. We now do cesarean sections only for obstetrical reasons in patients with heart disease.

The questions as to the advisability of interruption of pregnancy with sterilization and of sterilization alone have been given much attention in the literature and have caused some disagreement between departments in our own group.

TABLE VIII

METHOD OF DELIVERY IN 103 PREGNANCIES COMPLICATED BY ORGANIC HEART DISEASE

| Method | Cases | % | Mortality | % |
|---|---|---|---|---|
| Spontaneous abortion | 2 | 2 | 0 | |
| Therapeutic abortion | 4 | 4 | 0 | |
| Cesarean section | 6 | 6 | 2 | 33% |
| Breech extraction | 4 | 4 | 0 | |
| Low or outlet forceps | 25 | 25 | 1 | 4% |
| Spontaneous* | 59 | 58 | 1 | 2% |
| Died undelivered | 2 | 2 | 2 | 100% |
| No data | 3 | 3 | 0 | |
| Total deliveries | 105 | | | |
| Total pregnancies | 103 | | | |
| Total patients | 100 | | | |

*2 sets of twins

- - -

Only two pregnancies have been interrupted because of primary cardiac disease in recent years in the department. One of these was done because of almost continuous very severe angina pectoris in a patient with a failing heart. The other was in a patient with very severe cardiac disease who had decompensated in a previous pregnancy and who had recently been decompensated in the non-pregnant state. The results of this study seem to justify the department's concept that with careful handling the usual patient with organic heart disease may be carried through pregnancy safely and shows no evident shortening of her life expectancy for the cardiac disease because of the pregnancy. Therefore there can be no indication for interruption of pregnancy in other than the most unusual circumstances. These exceptions are extremely rare.

The department has been more liberal in the use of sterilization of cardiac patients. The rearing of children involves heavy cardiac work. Particularly where the degree of cardiac damage involves the necessity of limitation of activities, it seems wisest to preserve what cardiac reserve is available for the reasonable care of a limited number of children. It is impossible to obtain control groups of patients with cardiac disease which would allow an objective evaluation of the salvage effect of this

policy.

Recently within our department there has been established a routine for care of cardiac patients during labor, delivery and first post partum day. Pulse and respiratory rates are followed at 15 minute intervals during labor. A persistence of pulse rate over 110 or respiratory rate over 24 for three 15 minute periods is an indication for rapid digitalization.[12,13] Vital capacities are checked at 1-2 hour intervals. Sedation is always maintained slightly above that used in the normal pregnant patients. During second stage, the patient receives a 50-50 mixture of nitrous oxide and oxygen during contractions and pure oxygen between contractions. The perineum is anesthetized locally with 1% procaine. Delivery ideally is accomplished by low forceps extraction to relieve second stage effort.[13,etc.] Every four hours on the day of and the day following delivery, a brief respiratory and cardiac check is made to guard against impending complications. This above routine cannot in all cases be followed strictly because of the rapidity of many of the labors. The first post partum day is the most critical individual day during the pregnancy, but as will be shown, the 33-36 week period constitutes the most critical period during the pregnancy.

The average weight of all viable infants was 2999 Gms., and the average weight of all term infants was 3277 Gms. The average lengths were 47.8 and 49.6 cms. respectively. These are within the range of normal.

The gross fetal mortality of 13% (including abortions), the gross premature mortality of 25%, the premature neonatal mortality of 5%, and the gross mortality of term infants of 4% differ little from comparable figures for normal material, and similar to the findings of Teel.[17] Of interest was one of the 2 term neonatal deaths. The mother had an interventricular septal defect diagnosed by the cardiologists with confirmatory x-ray evidence. The mother had no infectious disease during her pregnancy. The infant died on the first post partum day. A very large interventricular septal defect, diagnosed by the pediatricians antemortem, was confirmed at autopsy.

PEAK LOAD OF PREGNANCY:

An effort was made to determine the period during pregnancy at which time patients showed maximum changes for the worse. In 58 cases who survived the delivery and puerperium and in which there was believed to be adequate data, the period of the peak load was estimated. This is shown in Table IX.

TABLE IX

CLINICAL PEAK LOAD PERIOD IN 64 PATIENTS

| Period of Gestation | No. of Patients | Time of Death |
|---|---|---|
| 28 or less weeks | 7 | |
| 29-32 weeks | 11 | |
| 33-36 weeks | 32 | 2 |
| 37-40 weeks | 3 | |
| Labor and delivery | 3 | 1 |
| Puerperium | 2 | 3 |
| Total | 58 sur-vivals | 6 deaths |

Also included for the sake of completeness was the time of death of the patients who died. Apparently the peak load is met most often between the 33rd and 36th weeks. This has been the general experience of others reporting in recent years. Until we can supply accurate data from both the normal and abnormal, which can be considered an accurate estimate of cardiac and pulmonary functional capacity, we are not justified in saying more than perhaps the 33rd to 36th weeks is the critical period in the majority of cases.

LABORATORY FINDINGS:

A statistical study of a large amount of accumulated laboratory data has been made. Our findings have borne out the contentions of other authors in many respects, and are contrary to the findings of others in a few instances. But as intimated earlier, we are forced to agree with Hamilton and Thomson when they said, in 1941,[8] "to date, no satis-

factory method of classifying poor from good risk patients with heart disease has been evolved by physiological studies of the circulation". The data presented below is drawn from the 68 pregnancies complicated by well substantiated rheumatic heart disease.

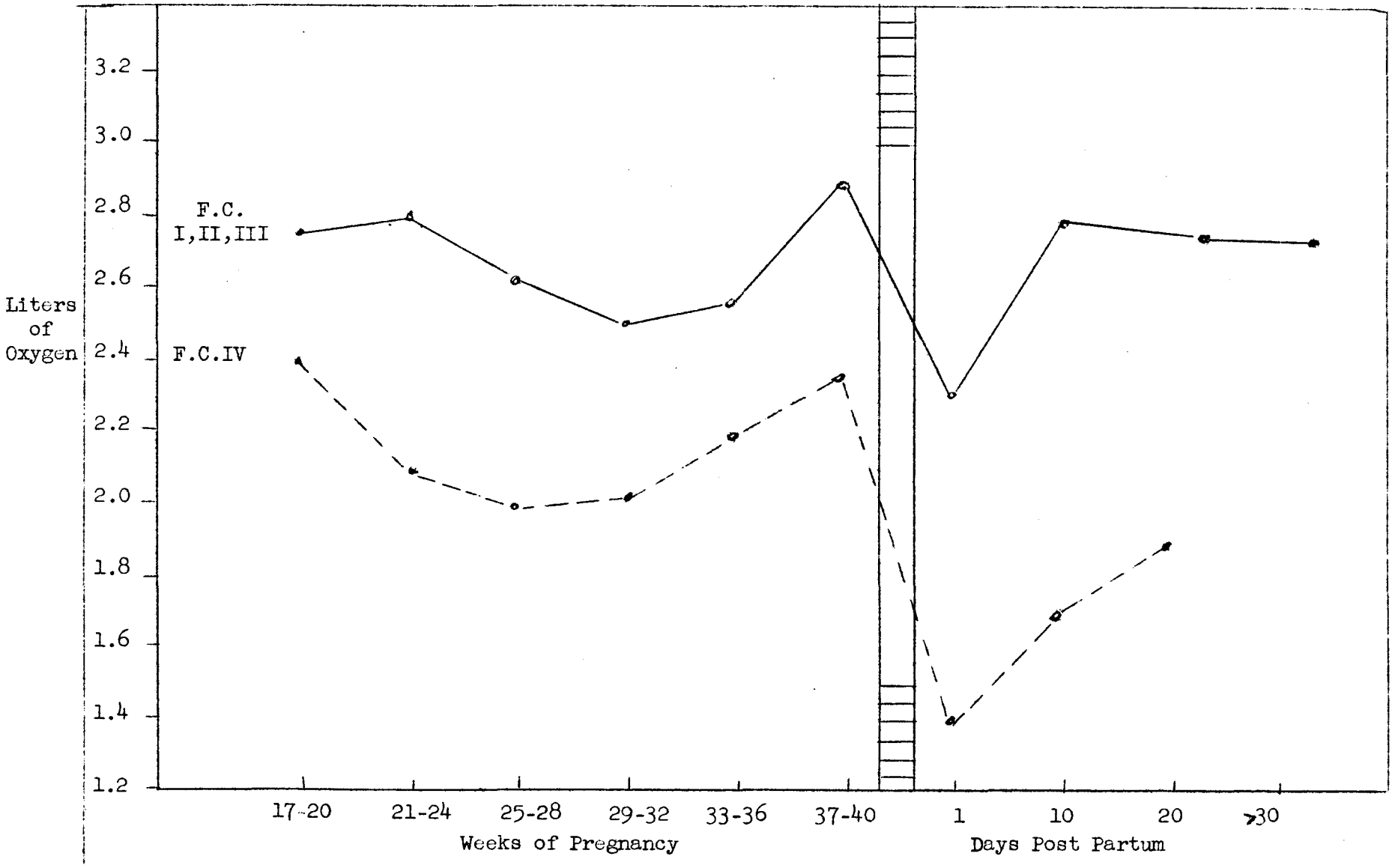
Three of the 16 patients (19%) in this group who decompensated had hemoglobin values of less than 10 Gms/100 cc. This is no higher incidence than seen in the group as a whole (18%). Others have claimed that a high per cent of their patients who decompensate have an anemia, but they fail to control their series. The anemia problem in pregnancy requires considerable work. The basic question of hemodilution has not been satisfactorily settled. The true status of the physiologic anemias awaits combined plasma volumes with dyes and cell volumes with tracer substances.

Blood volume studies to date are incomplete. The oft-quoted curve of the rise of blood volume during pregnancy as determined by Thomson, et al., on 14 patients lacks statistical confirmation[8]. All that has been shown to date is that during pregnancy, there is a rise in blood volume. The percentage increase varies widely from month to month in the same individual, and the group distribution also varies widely regardless of the period of pregnancy. The control of blood volume, if that were possible, would appear to have therapeutic value. To be able to reduce cardiac output by reducing the blood volume would alleviate cardiac strain. Conversely, it seems reasonable that if we see a rapid increase in blood volume, we are seeing an increasing load placed upon the heart. Thus far we have only individual case histories to support these concepts. For the group, the data gathered to date is too incomplete to justify dogmatic conclusions.

Many vital capacity determinations were made on the cardiac patients. As noted in Figure I, in the 60 patients with rheumatic heart disease who had vital capacity determinations done, 258 antepartum and 76 postpartum determinations were made, an average of 5.6 per patient. However, the curves as drawn give a false impression of the true status of vital capacity during pregnancy. Most of our patients were seen for the firs time during those periods where the low readings are seen on the graph. Thus their initial readings, taken before any therapeutic regime was instituted, tended to pull the curve downward in this range. With this in mind, a study was made of 12 patients who had at least 4 V.C. determinations prior to the 28th week and were seen with determinations at no longer than 2 week intervals from then until a term delivery. These patients were also FCI or FC2. The average of the 4 readings taken prior to the 28th week was called the "rise of therapy", although other factors than the therapeutic regime may play a part. But with a well established baseline in each individual, we could then plot any variations from that individual mean on a graph for the group. From this, as one can see in Figure II, there is a definite rise in vital capacity near the end of pregnancy. Actually the only significant figure is that of the 40th week compared with the early pregnant mean. It will also be noted that on the day of or the day following delivery, there is a sharp drop in the vital capacity. This has been noted by many, in normal as well as pathological. We recently began to study this and have noted that the drop is not immediate. In fact, for three hours following delivery, the vital capacity was higher than that noted on several occasions during labor. However, after the three hours it begins to fall, and the decline is steady, usually for as long as 24 hours after delivery. The nature of these curves discourages the implication that the drop is due to fatigue. Vital capacity gradually reaches normal near the end of the puerperium. The curve in cardiac patients is the same as in normals, but the variations are greater in the cardiac patients. Recently an excellent article by Widlund[18] has appeared. He has a large series of carefully, well controlled experiments on pulmonary functions during pregnancy. He showed a definite rise in vital capacity during pregnancy, the vital capacity being highest between the 25th and 36th weeks. His paper on cardio-pulmonal function during normal pregnancy is highly recommended to all those interested in investigative work in this field.

Fig. I

UNCORRECTED VITAL CAPACITY

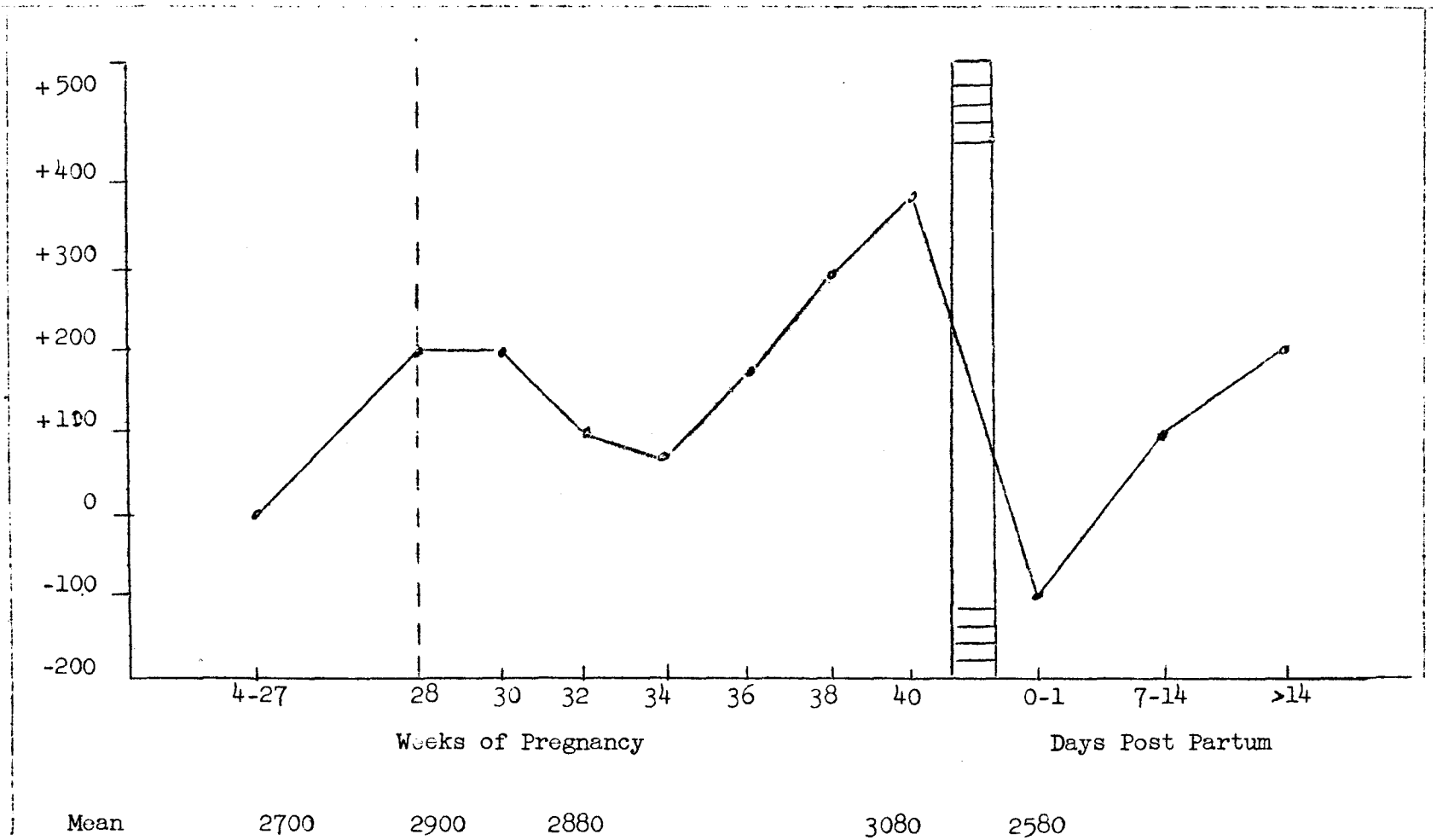



Observations

| | A.P. | P.P. | Pts. | Av./Pt. |
|---|---|---|---|---|
| Total | 258 | 76 | 60 | 5.6 |

Fig. II

VITAL CAPACITY IN 14 PATIENTS WITH
RHEUMATIC HEART DISEASE FC I & II




| | | | | | |
|---|---|---|---|---|---|
| Mean | 2700 | 2900 | 2880 | 3080 | 2580 |
| S.D. | ±580 | ±242 | ±298 | ±231 | ± 436 |
| S.E. | ±155 | ± 74 | ± 80 | ± 68 | ± 145 |

Venous pressure determinations in most cases were of little value. Three observations of interest were the initially high venous pressure on most patients, the increase in venous pressure immediately following delivery, and the one instance where venous pressure elevations led to the diagnosis of chronic pulmonary hypertension, confirmed, of course, by other findings. The mean venous pressure was 11.1 ± 2.6 (2 S.D.) cm. of water, which is 2.8 cm higher than the mean venous pressure observed in a series of normal pregnant women in this hospital by Dr. McLenna, but still fell with the range of probability of his series[11]. Venous pressure is of value in following the individual patient as confirmatory evidence of apparent clinical changes of cardiac function, and in the rare cases of pure right heart failure.

Circulation time is usually within normal limits. No significant decrease in circulation time was noted, although a slight decrease was noted between the 28th and 34th weeks of gestation. This could be consistent with the decrease in blood viscosity noted during that period by other observers. Circulation time is a better aid in determining cardiac function in the individual patient than is venous pressure, because the lung to tongue circulation time is a measure of left heart function, the left heart failure being the first to be noted in all our patients with rheumatic heart disease. Decholin and calcium gluconate showed significantly shorter arm-to-tongue circulation times than did fluorescein in pregnant cardiac patients.

To further discuss the numerous physiological changes noted during pregnancy is beyond the scope and intent of this paper. The accurate tests of physiologic function either have not been done during pregnancy or have not been made clinically applicable.

CARDIAC EVALUATION:

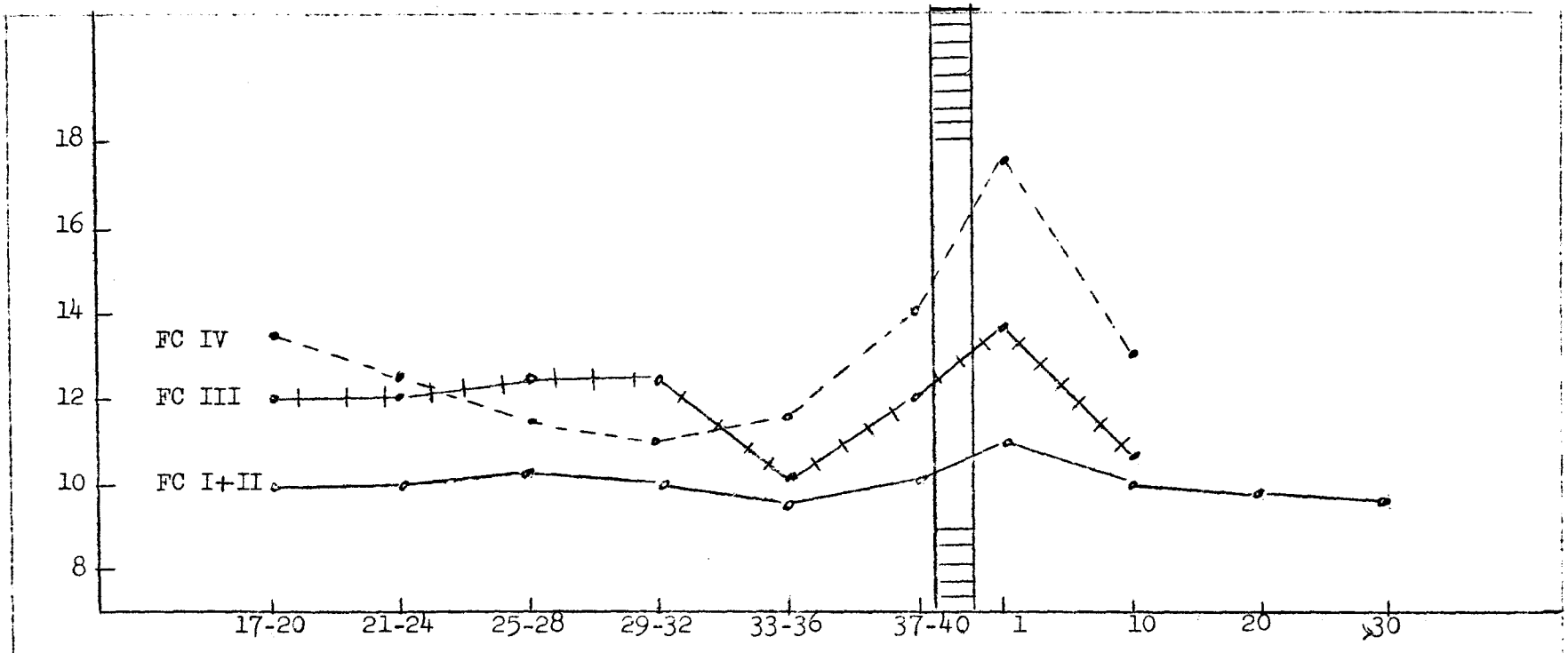
In an attempt to learn if there were any cardiac findings which would give a key to the prognosis in the individual cases, the reports of the cardiologists were analyzed. Again we restrict ourselves to the rheumatic group. The findings in the congenital groups have been previously reported.

In the 51 pregnancies of uncomplicated mitral stenosis and/or insufficiency cardiac findings were as follows: 6 patients had paroxysmal auricular tachycardia, and of these, 2 were class 2, 1 was class 3, and 3 were class 4. Classes noted are the maximal class reached at any time during the pregnancy. Six patients complained of paroxysmal nocturnal dyspnea, and of these, 3 were class 3 and 3 were class 4 patients. Four patients had previously decompensated, and of these, 1 was class 3 and 3 were class 4. Two class 4 patients had auricular fibrillation. One class 4 patient had a gallop rhythm. From these findings no new information was obtained, these findings being noted frequently by others. The only electrocardiographic findings which might have some significance were p wave changes in 17 of 47 patients, and a prolonged P-R interval in 7 of 47 patients noted on repeated cardiograms. No patient with either of these changes remained in FCI throughout her pregnancy, but the advances in functional class were evenly distributed between classes 2, 3, and 4.

Left atrial enlargement was shown by esophagogram in 43 of the 51 patients. If determination of the cardiac enlargement was none, minimal, moderate, or marked, as recorded on the x-ray reports, either on the esophagogram or the 6-foot heart plate, it was noted that no patient decompensated with less than a moderately enlarged heart with the exception of 2 patients who eventually decompensated as a result of subacute bacterial endocarditis. However, it required more than a moderately enlarged heart to result in decompensation, because many of the younger patients had moderately enlarged hearts and did not decompensate, and 4 patients with moderately enlarged hearts in fact did not even advance 1 functional class over their non-pregnant functional class. Thus the only positive quantitative conclusion that can be drawn is that all patients who decompensated had "moderately enlarged hearts" with the exception of those with subacute bacterial endocarditis.

Fig. IV

VENOUS PRESSURE




Determinations

| | A.P. | P.P. | Pts. | Av/Pt |
|---|---|---|---|---|
| FC I & II | 60 | 18 | 30 | 2.6 |
| FC III | 52 | 14 | 12 | 5.5 |
| FC IV | 58 | 13 | 13 | 5.5 |
| Totals | 170 | 45 | 55 | 3.9 |

Antepartum Mean

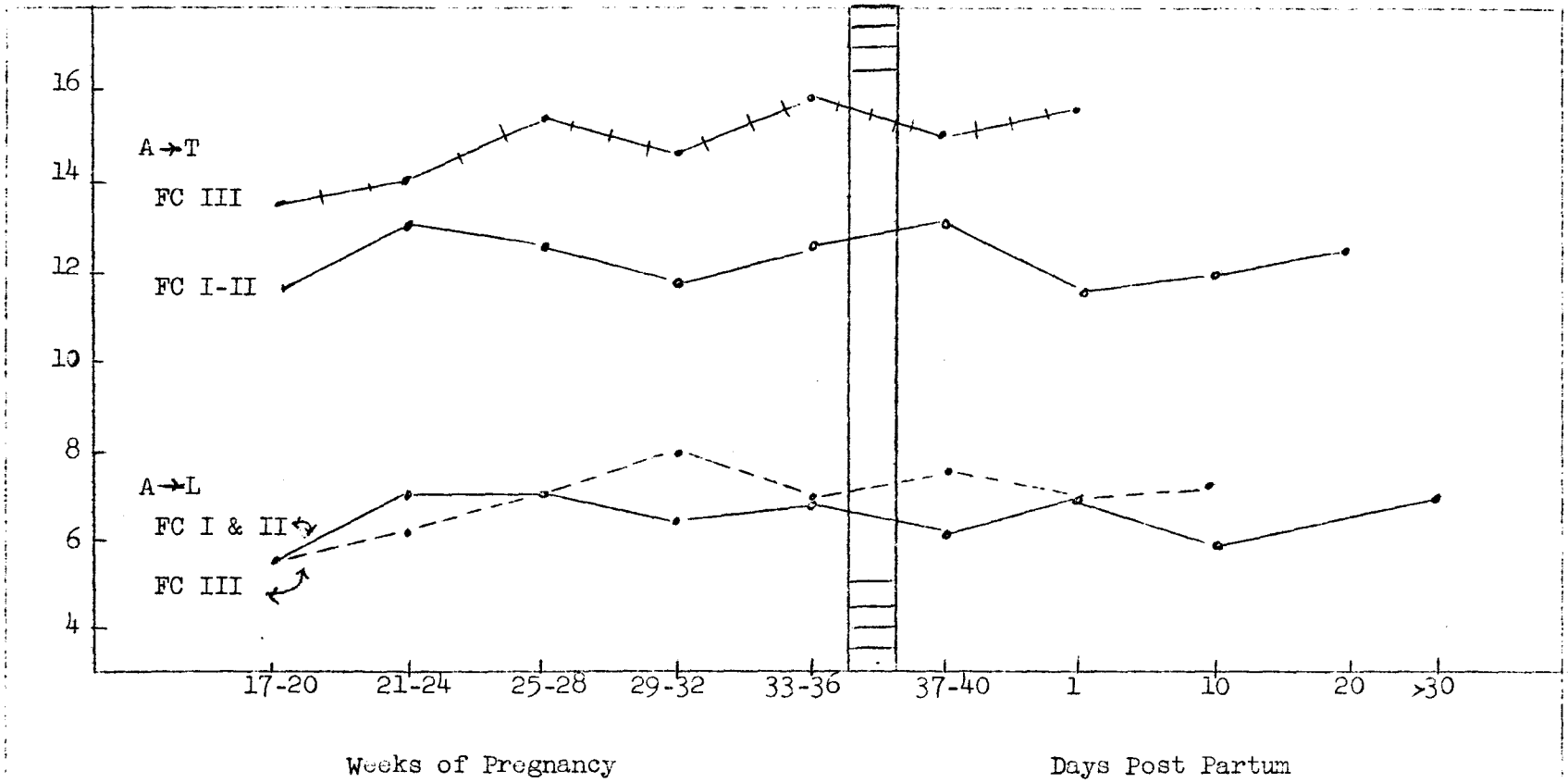
11.1 ± 2.6 (2 S.D.)

Cm. $H_2O$

F.C. I & F.C. II & F.C.III

Fig. V

CIRCULATION TIMES




Observations

| | A.P. | P.P. | Pts. | Av/Pt. |
|---|---|---|---|---|
| FC I & II | 52 | 17 | 34 | 2.0 |
| FC III | 38 | 7 | 13 | 3.5 |
| Totals | 90 | 24 | 47 | 2.6 |

Auscultatory findings proved to be of interest in the 50 patients (51 pregnancies).

| | |
|---|---|
| Apical systolic murmur | 43 |
| Apical presystolic murmur only | 44 |
| Apical mid-diastolic murmur only | 3 |
| One apical diastolic murmur only | 47 |
| Apical presystolic and mid-diastolic in same patient | 14 |

There were three patients included who did not have diastolic murmurs, and all three had definite evidence of left atrial enlargement by x-ray studies. The most interesting feature noted from this part of the study was that the mid-diastolic murmur appeared to have a serious prognostic significance. As was previously stated, 17 of the 50 patients with mitral lesions alone had mid-diastolic murmurs. Only one patient was class 1 maximum during her pregnancy, and her rheumatic heart disease began during one pregnancy with no diastolic murmurs of any nature noted. When she returned in one year for her second pregnancy, a mid-diastolic murmur was noted. Only 2 patients were class 2 and 4 were class 3. All six of these patients were FCI in the non-pregnant state. Ten of the 14 patients with pure mitral disease who decompensated had mid-diastolic murmurs. There is good statistical evidence to support the seriousness of this finding. Most cardiologists agree that the mid-diastolic murmur occurs rather late in the course of rheumatic heart disease, and that the duration of the course is inversely proportional to the cardiac insult received during the active rheumatic state. Thus we might conclude that the duration of the rheumatic heart disease is more important than the age of the patient in years. In support of this concept, barring those who developed subacute bacterial endocarditis, there was no patient under the age of 30 who decompensated or died who did not have a mid-diastolic murmur. Lamb noted its significance[9]. Further observations in this regard would be interesting, because from this study, age and a mid-diastolic murmur have evolved as the only 2 findings which have statistical significance in determining factors of prognostic import. Others undoubtedly should also be added and would have been had our series been large enough.

Two patients had acute rheumatic fever during the pregnancy, but neither advanced beyond FC2.

There were 11 patients followed through 12 pregnancies who had combined mitral and aortic lesions. Rhythm irregularities were slightly more frequent in this group. Paroxysmal tachycardia was noted in the history of 2 of the 12 pregnancies, 1 class 3 and 1 class 4. Paroxymsal nocturnal dyspnea was noted in 25% of the pregnancies, 2 class 3 and one class 4. Auricular fibrillation was noted in one class 3 patient who spontaneously aborted at 20 weeks gestation, fortunately, and was also noted in one class 4 patient. One class 4 patient was decompensated during and between previous pregnancies. Ten of the 11 patients showed left auricular enlargement, and 8 of the 11 had left ventricular enlargement to a degree greater than would be expected from the pregnancy alone. There were no constant electrocardiographic findings. Again no patients decompensated who had less than moderate cardiac enlargement, and no patients remained class 1 who had P-wave changes.

The auscultatory findings in the 11 patients were again of interest:

| | |
|---|---|
| Apical systolic murmur | 8 |
| Apical presystolic murmur | 11 |
| Apical mid-diastolic murmur | 2 |
| Basal aortic systolic murmur | 8 |
| Basal aortic diastolic murmur | 10 |

Again the serious nature of the mid-diastolic apical murmur was brought out in that one patient was class 3 at bed rest for the last 10 weeks of her pregnancy and the other patient was decompensated. The only class 4 who did not have it was interrupted in the 20th week because of a most severe angina of effort. Of interest is the fact that there were 8 patients with an aortic systolic murmur, but in only one of these 8 was there good evidence of an actual aortic stenosis.

There were 5 patients with pure aortic lesions. One patient had a pure aortic stenosis with a pressure of 76/52. The other 4 had aortic insufficiency. There was also one of this group who was believed to have an Austin Flint murmur. Noe of this group advanced beyond FC2 during the pregnancy.

CARDIAC DECOMPENSATION:

Those patients who decompensated during the pregnancy were closely scrutinized in an effort to determine further the factor leading to the decompensation. Much of this has been presented before but will be repeated for emphasis.

There were 23 patients who decompensated during pregnancies observed here. Five of the patients had been followed, usually very irregularly, in the outpatient clinic and the remaining 18 were admitted directly to the hospital in a decompensated state. With one exception, that of the patient with chronic pulmonary hypertension, the patients primarily had left heart failure. The distribution of these patients as to location and type of lesion is shown in Table X. The age distribution and parity is shown also in Table XI. These last two features have been emphasized previously. Twenty-one of the 23 patients received digitalis therapy prior to de-

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TABLE X

LESIONS IN DECOMPENSATED PATIENTS AND CAUSE OF DEATH

| Lesions | Patients | Deaths | Cause of Death |
|---|---|---|---|
| Patent ductus arteriosus | 2 | 1 | Failure, pre-eclampsia, shock. |
| Interventricular septal defect | 1 | 0 | |
| Hypertensive cardiovascular disease | 1 | 0 | |
| Thyrotoxic heart disease | 2 | 0 | |
| Chronic pulmonary hypertension | 1 | 0 | |
| Mitral stenosis | 14 | 4 | 2-S.B.E., 2-failure, undel. |
| Mitral stenosis and aortic regurgitation | 2 | 1 | S.B.E. |
| Totals | 23 | 6 | |

- - -

TABLE XI

AGE DISTRIBUTION AND PARITY

| Age | Patients | Parity | Deaths | Parity |
|---|---|---|---|---|
| 20-24 | 2 | 0-1, I-1 | 1 | I-1 |
| 25-29 | 7 | 0-5, I-1, III-1 | 3 | 0-1, I-1, III-1 |
| 30-34 | 7 | 0-1, L-1, II-1, III-4 | 1 | III-1 |
| 35-39 | 4 | III-1, IV-2, V-1 | 1 | III-1 |
| Over 39 | 3 | V-1, VI-1, VIII-1 | 0 | |
| Totals | 23 | 0-7 | 6 | 0-1 |
| | | I & II - 4 | | I-2 |
| | | III or more- 12 | | III-3 |

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livery. One patient was admitted ready for the delivery table and was delivered prior to any knowledge of her cardiac status. She was digitalized within a few hours of delivery. The other patient improved so markedly on bed rest and low salt diet that digitalis was not deemed necessary. Two patients who have been treated for subacute bacterial endocarditis, in recent years, have now been successfully carried through pregnancies.

Table XII shows the changes in functional classifications of these patients. It is of interest to note that only 1 patient who was class 1 in the early pregnant state decompensated, and this was a result of subacute bacterial endocar-

TABLE XII

FUNCTIONAL CLASS CHANGES IN DECOMPENSATED PATIENTS

| Period | Class 1 | Class 2 | Class 3 | Class 4 | Dropped |
|---|---|---|---|---|---|
| When first seen | 1 | 2 | 7 | 13 | |
| Non-pregnant | 7 | 10 | 5 | 1 | |
| Early pregnant | 1 | 7 | 8 | 7 | |
| Peak | 0 | 0 | 2 | 18 | 3 - Rx Abort. |
| Delivery | 0 | 1 | 10 | 7 | 3 - Rx Abort;<br>2 - Died undel. |
| Post partum | 0 | 4 | 8 | 2 | 3 - Rx Abort;<br>2 - Died undel;<br>4 - Died post partum |

- - -

ditis. Eight of the patients, one of which remained FCIII, decompensated prior to the expected period of the peak load of pregnancy. Three had therapeutic abortions. The remaining 5 were carried through and all obtained living infants. One of these patients had subacute bacterial endocarditis and died during the puerperium. Another patient was so severely ill that it was feared that the trauma of abdominal hysterotomy would surely prove fatal, and it was thought wisest by all to trust to conservative measures although this seemed hopeless for a time. She tolerated delivery from below 10 weeks later remarkably well. She died suddenly at home during her 4th post partum month. A third patient had severe infectious hepatitis in addition to her heart disease and decompensated from the combination early in pregnancy. As the hepatitis improved, so did the heart failure and she was FC2 at the time of term delivery. She is living and FC2 four years post partum, having well tolerated 2 subsequent pregnancies. The 4th improved so much on cardiac therapy that it was not deemed necessary to interrupt the pregnancy. The last of the five permitted to carry their pregnancies through to term was the patient with cor pulmonale who delivered with no difficulty. She was readmitted at three months and sterilization was done. The patient was cyanotic for days after the procedure and extremely dyspneic for two weeks. However, she recovered, and was admitted to this hospital 7 years postpartum with a diagnosis of pulmonary hypertension and pulmonary arteriosclerosis which was confirmed at autopsy. Of the three patients who had therapeutic abortions, two are still living, one FC2 one year postpartum, and the other FC4 six months postpartum. The third died 2 years postpartum.

In Table XIII are listed the features which were noted to be of special importance from the study of decompensated patients. Cardiac enlargement was omitted only because it was impossible to know, in many of the cases, what degree of the enlargement was present prior to the failure or not. All cases had at least moderate cardiac enlargement on roentgenography. Using the chi square test as a measure of probability, we can again say that it is at least highly unlikely that age and the mid-diastolic murmur are not excellent prognostic signs in determining those pregnant patients with rheumatic heart disease who will decompensate dur-

TABLE XIII

CLINICAL FINDINGS IN DECOMPENSATED PATIENTS

| Cardiac Findings | Patients | Non-cardiac Findings | Patients |
|---|---|---|---|
| Mid-diastolic murmur | 11 | Age over 30 | 14 |
| Previous decompensation | 7 | Upper respiratory infection | 9 |
| Auricular fibrillation | 3 | Anemia less than 10 Gms/100cc | 4 |
| Complete heart block | 1 | Toxemia of pregnancy | 4 |
| Bacterial endocarditis | 3 | Primary hypertension | 2 |
| Paroxysmal tachycardia | 3 | Thyrotoxicosis | 2 |
| Severe angina pectoris | 1 | Severe infectious hepatitis | 1 |
| Total patients | 21 | Total patients | 21 |

Two patients decompensated without any of above, having only cardiac enlargement.

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ing the pregnancy or puerperium. And one also must accept the other findings listed above as having a prognostic significance in many patients as determined from the experiences of investigators in this field. And still, in spite of the above statements there were 2 patients who decompensated that showed not a single one of the above clinical or cardiac findings, but both had subacute bacterial endocarditis.

FOLLOW-UP STUDIES:

The mortality statistics of patients with rheumatic heart disease was reviewed in an effort to form a basis whereby we might better determine the effects of pregnancy on the duration of life. Boyer and Nadas[2] stated that the average duration of life of those patients with rheumatic heart disease who had reached the age of 20 was identical, whether males or females, and in the females whether they had borne children or not. It could be assumed, however, that the nulliparous women had no children chiefly because they had severe heart disease and were advised against pregnancies. To overcome this factory, they reasoned that those with severe cardiac lesions should be dead by the age of 40. So applying the same statistics to those patients who had reached 40, they were again unable to demonstrate any effect of parity. Cohn and Lingg[3] reported the vital statistics of a group of 1566 males and 1563 females with rheumatic heart disease followed until death. They found that roughly 10% of all patients live more than 30 years after the onset of the rheumatic heart disease, 25% live more than 14 years and 50% die within 9 years. The average duration of life was 13 years. Their statistics are incorporated in Table XIV. The authors made no special study of the nulliparous and the parous women. There was no significant difference between the average duration of life of the males and females. If parity did play an important role in survival one would have expected it to reveal itself. Hamilton and Thomson[8] had a maternal mortality in their "favorable" cardiac patients who were seen regularly throughout their pregnancies which was the same as the expected mortality of "favorable" non-pregnant cardiac patients during the same period of time. However, in the "unfavorable group", those who were fibrillating, had had decompensation previously, or had a serious complicating condition other than the heart disease, the maternal mortality was three times that of the same non-pregnant "unfavorable group" over the same length of time. At the present time the available evidence cannot disprove the conclusions that:

1. The duration of life is not shortened by parity, regardless of the degree, if the woman survives the pregnancy.

2. Well-compensated patients followed throughout pregnancy have no higher

TABLE XIV (a)

ONSET OF RHEUMATIC FEVER

| | Females | Males |
|---|---|---|
| 67% begin in childhood | up to 12 years | up to 14 years |
| 10% begin in adolescence | 12 to 18 years | 14 to 20 years |
| 10% begin in early maturity | 19 to 29 years | 21 to 29 years |
| 10% begin in middle maturity | 30 to 45 years | |
| 3% begin in late maturity | 46 plus years | |

(After Cohn and Lingg)

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TABLE XIV (b)

SURVIVAL RATES OF VARIOUS GROUPS IN %

| Period of onset | Sex | Survival rate through Childhood | Adolescence | Early Maturity | Mid-Maturity |
|---|---|---|---|---|---|
| Childhood | male | 66 | 30 | 16 | 4 |
| | female | 73 | 40 | 20 | 7 |
| | average | 69 | 35 | 18 | 5 |
| Adolescence | male | | 89 | 63 | 26 |
| | female | | 83 | 50 | 17 |
| | average | | 85 | 55 | 21 |
| Early maturity | male | | | 86 | 29 |
| | female | | | 78 | 18 |
| | average | | | 81 | 23 |
| Mid-maturity | male | | | | 45 |
| | female | | | | 43 |
| | average | | | | 44 |

Average male $12.5 \pm 0.3$ years
Average female $13.0 \pm 0.3$ years

(After Cohn and Lingg

- - -

mortality than a comparable group over a comparable period in the non-pregnant state.

3. The functional capacities of patients with families is materially impaired, although duration of life may remain the same.

At the present time we are engaged in a follow-up study of all our pregnant cardiac patients in an effort to prove or disprove the above. This must, by its very nature, be a long time study, and must also, as is undoubtedly the case[4], be engaged in by many others in the field, in order that, by pooling all available information, conclusions drawn may be warranted. Studies as to the actual duration of the heart disease are of extreme value.

The main obstacle we have encountered in our follow-up study has been the tracing of the 30% of our group who were illegitimately pregnant. In most cases

we have not been able to determine the status of these patients at present, but we dare not ask for clinic visits.

Because the N. Y. Heart classification is based on symptomatology alone, it is especially amenable to a follow-up study by questionnaires. The questionnaires were sent to all the patients. The questionnaire was designed to supply adequate information from which to accurately judge the present functional class. Many of the patients returned to the out-patient department for examinations. But others simply filled out and returned the questionnaire. It was obvious that it not only had value to us, but also to the patient. From this alone we advised 7 patients to get under the care of a physician, either locally or here.

The data here presented are included only for the sake of completeness and interest. No valid conclusions can be drawn from the material as yet.

It will be noted that there were 24 subsequent pregnancies after the initial examination here. Only 3 of these were delivered here. None of the 24 pregnan-

TABLE XV

RHEUMATIC HEART DISEASE FOLLOW-UP

| | |
|---|---|
| Total rheumatic patients | 72 |
| Died during pregnancy | 5 |
| Eligible for follow-up | 67 |
| Patients delivered elsewhere | 1 |
| Probable cardiac patients | 6* |
| Included in follow-up (62 pregnancies) | 60 |
| Duration of follow-up | $\frac{1}{2}$ to $8\frac{1}{2}$ years |
| Lost | 2 |
| Died (1 at 4 mos.; 1 at $1\frac{1}{2}$ yrs.; 1 at 5 yrs.) | 3 |
| Evaluated (including three who died) | 47 |
| Known living but not evaluated | 11 |
| Patient years known after pregnancy | $193\frac{1}{2}/204$ |
| Mortality per 1000 patient years | 15.2/1000 |
| Expected mortality from non-decompensated rheumatic heart disease | 20/1000 |
| If both "lost" patients have died | 25.2/1000 |
| Subsequent pregnancy after first pregnancy here (18 of these elsewhere) | 20 |
| Maximum functional class III | 1 |
| II | 8 |
| I | 4 |
| ? | 7 |
| Average age of living patients | 28.4 |

*Probable rheumatic heart disease patients who were in series were followed: 5 were the same and diagnosis still in question. One was advanced functional class II and diagnosis still in question.

TABLE XVI

CONGENITAL HEART DISEASE FOLLOW-UP

23 patients - 1 died during pregnancy
3 lost - one at 8½ years, one at 5 years
19 followed:
16 evaluated
3 known living but not evaluated
Patient years of follow-up 77½/91 years
Died - 1 11/1000 patient years
Decompensated - none
Advanced two classes - 1
Advanced 1 class - 1
Remained same - 10
Improved 1 class - 2
Improved 2 classes - 1
Change unknown - 6
Known subsequent pregnancies - 4 (one here)
Average present age - 28.9 of known living patients

- - -

TABLE XVII

MISCELLANEOUS TYPE FOLLOW-UP

5 patients All followed
25/25 years
2 deaths
1 unchanged
2 better, one is F.C.II and one is F.C.III
Average present age 40.2 years.

- - -

cies occurred in patients who exceeded a maximum functional class 2 in the pregnancy observed here.

The mortality in the follow-up series is slightly, but not significantly less, as expressed in 1000 patient years, than would be expected in a comparable group of non-pregnant patients with rheumatic heart disease. There are no studies in the literature with which we can compare the survival of the group with congenital heart disease.

CONCLUSIONS

1. The functional class determinations as now defined by the New York Heart Association before, during, and after pregnancy as used above has been shown to be of value in individual and group studies. In heart disease complicated by pregnancy, a simple method of denoting overall changes in the patients would appear to be of value.

2. During pregnancy, 50% of the patients advanced at least one functional class, 20% decompensated, and 6% died. The last two figures are similar to those reported elsewhere. No other study in pregnant or non-pregnant patients in a comparable period of one year has been done with which the first figure may be compared.

3. That period during pregnancy when patients showed the maximum functional impairment of activity was, in the vast majority of cases, between the 33rd and 36th weeks of pregnancy.

4. Conservative measures of obstetrical management have been shown to be most satisfactory. Delivery per vaginam is the method of choice. During labor, delivery, and the first post partum day the patient must be carefully watched for clinical signs of early decompensation. Fetal salvage rate in well compensated patients is no lower than in normal pregnancies.

5. Physiologic tests of cardiac function have shown:

   a. The determination of vital capacity, using the McKesson-Robbins machine, was the most valuable of the earily applied functional tests. It was not only of value in following the individual patient, but also of value in showing that during pregnancy there is a progressive increase in vital capacity with a pronounced drop within 24 hours of delivery, followed by a return to the status quo ante.

   b. Blood volume studies, after standards have been established in each patient, may prove an aid in determining the effectiveness of therapy designed to decrease cardiac

output.

c. Venous pressures, circulation times, blood and urine studies have limited value in this study.

6. With the exception of one patient with subacute bacterial endocarditis, no patient under the age of 30 decompensated who did not have a mid-diastolic murmur. Patients who had this murmur usually showed advanced stages of rheumatic heart disease. This finding was of significance at any age. A sampling error was highly unlikely, and we are therefore justified in stressing its prognostic significance.

7. All patients over the age of 30 must be carefully watched throughout their pregnancies. Two of every three will decompensate, regardless of other findings. Only one in ten under 30 will decompensate.

8. With the exception of two patients with subacute bacterial endocarditis, no patient decompensated with less than "moderate" cardiac enlargement. However, many with this degree of enlargement did not decompensate.

9. We could demonstrate no difference in functional class advance, decompensation rate, or mortality rate between patients with pure mitral lesions and those with combined mitral and aortic lesions. The pure aortic lesions, only five in number, were relatively benign.

10. Parity, per se, did not appear to affect either the mortality or functional class change during pregnancy. It also has not been shown to effect a shorter duration of life.

11. Other findings, such as auricular fibrillation, paroxysmal nocturnal dyspnea, serious complicating diseases, etc., certainly are serious conditions when they exist. They are of great prognostic value in the individual cases, but are relatively infrequent.

12. From our follow-up study thus far we have found 15.2 deaths/1000 patient-years, a figure not significantly less than the general experience of all who have followed their non-pregnant rheumatic patients.

13. Finally, we wish to express our gratitude to the members of other departments who have helped us with the study and management of these patients.

REFERENCES


1. Boyer, N. H., Leach, C. E., and White, P. D.
Underlying Causes and Precipitating Factors in Congenital Heart Failure
Blood, Heart and Circulation, Science Press, '40.

2. Boyer, N. H., and Nadas, A. S.
Ann. Int. Med. 20:99, '44.

3. Cohn, A. E., and Lingg, Claire
Natural History of Rheumatic Cardiac Disease
J.A.M.A., Vol. 121; 1 and 113, '43.

4. Gilchrist and Murray Lyon
Edinburgh, M J. 40:587, '33.

5. Gorenberg, H.
Am.J.Obs.Gyn., 45:835, '43.

6. Gorenberg, H. and McGeary, J.
Am.J.Obs.Gyn., 41:44, '41.

7. Hamilton, Burton E.
Report from the Cardiac Clinic of the Boston Lying-In Hospital for the First 25 Years.
Am.Heart J., 33:663, '47.

8. Hamilton, B. E. and Thomson, K. J.
The Heart in Pregnancy and the Childbearing Age
Little, Brown and Co., '41.

9. Lamb, H. E.
Heart Disease in Pregnancy
Am.J.Obs.Gyn., 34:456, '37.

10. Lund, Curtis
Congenital Heart Disease in Pregnancy
(In publication)

11. McLennan, C. E.
Antecubital and Femoral Venous Pressure in Normal and Toxemic Pregnancy.
Am.J.Obs.Gyn. 45:568, '43.

12. Mendelson, C. L.
Management of Delivery in Pregnancy Complicated by Serious Heart Disease
Am.J.Obs.Gyn., 48:329, '44.

13. Mendelson, C. L. and Pardee, H. E. B.
The Pulse and Respiratory Rates during Labor as a Guide to the Onset of Failure in Women with Rheumatic Heart Disease.
Am.J.Obs.Gyn., 44:370, '42.

14. Sampson, J. J., Rose, E. M. and Quinn, R.
Estimation of the Work of Obstetric Labor and Its Significance in Heart Disease
Am.J.Obs.Gyn., 49:719, '45.

15. Stander, H. J.
Treatment of Heart Disease in Pregnancy.
Am.J.Obs.Gyn. 44:714, '42.

16. Stromme, W. B. and Kuder, K.
Heart Disease in Pregnancy
Am.J.Obs.Gyn., 52:264, '46.

17. Teel, H. M.
Study of Fetal Mortality in Patients with Organic Heart Disease.
Am.J.Obs.Gyn., 30:53, '35.

18. Widlund, Gerhard
The Cardio-Pulmonal Function During Pregnancy
Acta Obs. et Gyn.Scan. Vol. 25: Supp.1, '45.