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PREGNANCY AND HEART DISEASE FROM A MEDICAL VIEWPOINT*

REPORT OF A STUDY OF FORTY CASES FROM THE SLOANE MATERNITY HOSPITAL, NEW YORK CITY

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INTRODUCTORY

THE obstetrician of the period of medical development now passing, in addition to dealing with the mechanical problems of labor, has too often borne alone the burden of manifold and often vital decisions respecting the care of the infant and the numerous problems of pregnancy proper to internal medicine. Among these the acute infections, the toxemias, and the myocardial insufficiencies in relation to pregnancy, parturition, and the puerperium are of such complexity and importance as to call forth the best endeavor of those practiced in both fields. In recognition of this fact, in the Sloane Hospital for Women during the past two years, a combined study of these problems has been made by the obstetrician and the internist. In publishing the result of these studies, the last named—the myocardial insufficiencies —will be first considered. The acute infections and the toxemias will be taken up in later papers. This paper, therefore, summarizes the experience with all cases showing symptoms of myocardial insufficiency delivered in the Sloane Hospital during the two years from October, 1919, to October, 1921.

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REVIEW OF LITERATURE

In reviewing the recorded experience of others in similar cases, one is impressed with the wide difference in results and opinions. A few examples may be quoted:

Harrar[1], in a report of seventy-five cases with broken compensation during pregnancy, found an immediate maternal mortality of 30 per cent. He expressed the opinion that the prognosis improves the further along in pregnancy the woman is at the time of the first breakdown in circulatory balance. In nine cases in which the breakdown was between the ninth and tenth month, there was one death; of nine with the breakdown before the sixth month, seven died. In women giving history of broken compensation in previous pregnancies or before the present one, the mortality increases 60 to 70 per cent. Age and parity have little effect except in primipara. Hearts with a double mitral lesion are more prone to fail and to recover. Simple aortic lesions are most infrequent and have best recuperative power. Combined mitral and aortic disease is the most serious of all.

Longaker[2] states that the endeavor to carry these patients over months of increasing circulatory burdens to the period of fetal viability by rest and cardiac tonics after serious symptoms have developed is prone to spell disaster. "In the face of decompensation, there remains but one thing—empty the uterus."

Manges[3] proffers the opinion that cases of mitral stenosis when accompanied by mitral regurgitation usually have no trouble in labor and that mitral stenosis of the uncomplicated type is the only lesion giving trouble in pregnancy.

Hussey[4] believes that the majority of women with compensated heart disease go through pregnancy and labor without signs of decompensation. He states that the majority of fatalities occur some time after labor and believes that pregnancy may draw appreciably upon the store of cardiac reserve. He considers that the uterus should be emptied if broken compensation occurs early in pregnancy; if the patient has had broken compensation in former pregnancies; or if the symptoms of decompensation persist despite treatment.

Citing numerous writers, Hirschfelder[5] gives a mortality of 3 to 61 per cent in cases of valvular disease of the heart as a complication of pregnancy. Newell[6] believes that valvular disease of the heart complicating pregnancy may, for therapeutic purposes, be divided into two classes: The first, those showing decompensation early in pregnancy. These should have early abortion. The second, those showing decompensation in the latter period of pregnancy. These should have a second stage shortened and made as effortless as possible. In a later article, Newell[7] states that three per cent of pregnant women have cardiac complications which may be expected to react more or less seriously to the strain of pregnancy and labor. This writer is impressed with the reduction in reserve power in the heart resulting from childbearing and states his belief that every cardiac patient must pay for her child some price in length of days.

Blacker[8], in a very satisfactory review of the entire subject, remarks: "It is certain that the majority of cases of valvular disease of the heart complicating pregnancy do perfectly well and pass through labor and the puerperium with no symptoms." In 453 collected cases, Blacker finds the mortality 12 per cent. He remarks that the nature of the valvular lesion is not of so much importance as the capacity of the myocardium—an opinion with which the majority of earlier writers are at variance—mitral stenosis being as a rule regarded as the more serious menace to the pregnant woman.

Feis[9] remarks upon the rarity of heart failure in pregnancy. He quotes the experience of Wissner in the Berne Frauenklinik, who in 4,000 births, noted but 25 such instances with but one fatality. From the reports of several observers, the same writer cites the following mortality rates in the condition under consideration: Wissner, 37.6 per cent; Schlayer, 48 per cent; Macdonald, 60 per cent; Leyden, 65 per cent; Guerard, 40 per cent; Schneider, 14 cases with one death; Benny Hart, 8 cases of mitral stenosis and 7 deaths; Vinay, in the Hotel Dieu, 20 cases and no deaths. In Schlayer's experience, but 46.5 per cent of children in cases of chronic valvular disease were carried to term. Twenty-nine per cent were born dead. Feis'[9] experience leads him to the dictum that the probability of the survival of the child is so dubious that its life should not weigh greatly in the treatment of the mother.

Recently, Mackenzie[10] has written at length on this subject. His conclusions are, in general, not at variance with those of other writers. Few of these writers have stressed the importance of the purely medical treatment of cardiac decompensation in pregnancy, excepting Mackenzie[11] who says, "By bitter experience general practitioners have recognized that in certain diseased states of the heart there is great danger to the life of the woman and child. For over 50 years there have been many attempts to find out where the danger lies, and to recognize the signs which foretell disaster and the signs which need cause no anxiety. Yet the most recent of our textbooks show no advance on the views of 50 years ago."

"There is a physiologic problem involved, but no physiologist has attempted its solution. There are problems intimately connected with clinical medicine, yet physicians have no opportunity for studying the pregnant woman. The obstetrician sees her, but no obstetrician has yet learned the elements of cardiac symptomatology sufficiently to enable him to acquire the necessary information. Here is a problem of the first importance, which will, time and again, confront every general practitioner, and in the whole hierarchy of a medical school there is not one teacher or even group of teachers, capable of acquiring, far less of imparting, the necessary knowledge."

Most of the case reports deal with the purely obstetrical side of the matter, in which delivery is paramount. The larger number of patients have entered the obstetric hospitals at or near term—often late in labor, and under circumstances not affording opportunity for adequate medical measures. It seems clear that, no matter how skillfully labor be guided, it is the tardy recognition and care which is largely responsible for the high mortality in the cardiac disorders of pregnancy. The advantage to the patient of careful oversight throughout the entire antenatal period with the prompt institution of medical treatment in infections, toxemias, and cardiac conditions is unquestionable. This is more than a matter of opinion; it is a fact capable of ample proof.

MANAGEMENT OF CARDIAC CASES IN THE SLOANE HOSPITAL

The general plan of management of the pregnant woman with chronic disease of the heart is as follows: Pregnancy being a physiological process, we have taken the position that in the case of chronic cardiac disease with broken compensation, it is not the pregnancy, but

the heart condition which is the primary source of trouble. Therefore, where possible, pregnancy has been temporarily ignored and the first endeavor is the restoration of cardiac compensation before considering delivery. Of the greatest importance in accomplishing a satisfactory end result is the early recognition of disorders of the heart in the antenatal clinic. Cases presenting heart lesions are observed at very frequent intervals, and at the onset of unusual dyspnea, edema, palpitation, or other signs of decompensation, are admitted to the hospital and treated as cardiac cases. We would emphasize the importance of not instituting any measure looking toward delivery of the patient while symptoms of decompensation are present. Our own experience and review of the experience of others, has convinced us of the danger in hastening delivery in untreated cases of cardiac decompensation and has furnished proof of the high mortality in cases of decompensated cardiac disease delivered promptly upon their admission to obstetric hospitals. The greatest safeguard of the pregnant woman with chronic cardiac disease is, therefore, the antenatal clinic and prompt hospitalization at the first signs of serious decompensation.

The details of the medical treatment of this series of cases may thus be outlined: Rest in bed is usually enforced, but if the patient is more comfortable sitting in a chair, this is allowed. We have felt that the absolute prohibition of all bodily effort is not in every case necessary, and that it is possible some minor activity is an advantage in promoting the peripheral circulation, favoring venous return and thus reducing the strain on the heart. Adjuncts of value are massage and passive movements.

The diet is an ordinary, simple, light, well-balanced ration, the fluid content of which is somewhat limited, the average allowance of fluid being about fifteen hundred cubic centimeters in twenty-four hours. In cases with edema and signs of circulatory stasis, the Karrel diet, consisting of one quart of milk in twenty-four hours without intake of other solid or fluid, may be given for from two to five successive days until results appear. If, on this diet, thirst is annoying, cracked ice, fruit pulp, or chewing gum may be allowed. It is our experience that the Karrel diet is as satisfactory in the anasarca of pregnancy as in that accompanying decompensation apart from pregnancy. That there is an increased blood volume in pregnancy seems well established and is undoubtedly of great importance, and its reduction is accompanied by a very great improvement in most cases. With this purpose in view, along with diminished fluid intake, restriction of salt is enforced. Hydrogogue catharties are of use in a few cases as an accessory measure in this comparative dehydration.

Digitalis is the most valuable single agent in restoring compensation. In cases presenting only slight symptoms of decompensation,

digitalis is given by the so-called "small dose method"—fifteen to twenty minims of a standardized tincture being administered three or four times a day until physiological effects are obtained. In the more pronounced cases of cardiac insufficiency requiring more prompt effects, one dram of the tincture is given three or four times daily for one or two days, followed by smaller doses. In a very few cases presenting grave symptoms of cardiac decompensation, the Eggleston "body weight" method is employed. In this, fifteen cubic centimeters of a standardized tincture for each one hundred pounds of body weight are administered within a period of twenty-four hours. The initial dose is one-half the total quantity. The three later doses consist of one-half the remaining amounts given at six hour intervals. In only one case—an emergency—was strophanthin, one milligram, given intravenously. This was an example of acute cardiac dilatation accompanying influenza. It is probable that the method by which digitalis is given is, in most cases, unimportant. The aim is to administer sufficient to produce the desired physiological action.

When satisfactory digitalis results are secured, as shown by lowered pulse rate, lessened pulse deficit, improvement in subjective symptoms, and in the establishment of diuresis, the effect of the drug is continued by administration of the tincture, minims twenty, every twenty-four hours—an amount which the studies of Pardee[12] have shown adequate for the continued action of the drug in the individual of average weight when once digitalization has been accomplished. In this plan of treatment, diuretics play a small part but are often useful in initiating diuresis when digitalis effects have begun to appear. One or two doses of diuretin, grains ten, or theocin, grains ten, may be given with profit.

The response of the majority of pregnant women with cardiac decompensation to treatment of this kind, suitably modified to fit individual requirements, is most satisfactory and is further proof of the familiar statement that "the average case of chronic valvular disease bears pregnancy well." In general, in this treatment, pregnancy may be ignored. If proper response is had, pregnancy may be allowed to proceed, the patient generally returning to her home to report for examination at weekly intervals and to be again hospitalized with the return of decompensation. Frequently, a woman will be admitted several times for weekly periods during the later months of pregnancy for the restoration of circulatory equilibrium. By such a program of intermittent hospitalization, the majority of women with weak hearts can be carried along, decompensation kept in abeyance and term approached with the heart in condition to bear the strain of labor.

Not every case responds happily to medical treatment. When satisfactory results do not ensue, decision as to the proper procedure frequently taxes the wisdom of the most experienced obstetrician and

internist, and is a matter of judgment in the individual case. There are so many factors of weight in this decision that no formula applicable to all cases can be worked out. These factors may be brought under two heads: First, medical, including the efficiency of the myocardium as shown by the degree of decompensation, the response to treatment, and, in multipara, its behavior in previous pregnancies. Second, the obstetric, including the size of the pelvis, size of the fetus, stage of pregnancy, state of the cervix, parity, efficiency of the mechanism of parturition, etc. If decompensation is marked and persists despite careful medical treatment, pregnancy should be terminated by the method entailing the least effort on the part of the patient. Any other course is full of danger. Further, the high fetal mortality already mentioned (Feis[9]), argues against the continuation of pregnancy when decompensation is unyielding and severe. If decompensation occurs early in pregnancy and if former pregnancies have resulted in severe damage to the myocardium, abortion is generally advisable.

There are numerous cases in which cardiac symptoms do not become severe until the latter half of pregnancy and, while showing improvement, do not entirely disappear under medical treatment. A certain number of these borderline cases may be carried along with careful observation in the hospital to term or to fetal viability when normal labor or induction may be passed through with success. It may be said that more of these cases than seems possible go through successful childbearing without serious event. In making decision in cases of this sort, much depends upon the attitude of the woman. If, for the sake of offspring, she is willing to take the risk of acute dilatation during or after labor and of decline in cardiac efficiency afterward, this has a certain weight in the decision.

If the period of fetal viability is reached, further decision must be made. Shall the pregnancy proceed to term? If not, how shall it be ended? If obstetric conditions indicate the necessity of cesarean section and if decompensation impends, this measure may be taken so soon as a living child is assured. If, however, obstetric conditions promise a second stage free from undue effort and decompensation can be warded off, it is usually wiser to allow continuation to term when a more facile parturition is assured than to embark upon the uncertain course of an induced labor in advance of term. In certain cases at term in which an easy second stage is not probable, cesarean section may be the wise course even if the heart condition is well under control.

Upon the management of labor depends, to a large extent, the future efficiency of the myocardium. While the majority of women with heart disease will pass through labor with safety, a certain number will show permanent decline in reserve vigor of the heart. It is to

spare this that cesarean section is often resorted to. Again, this gives opportunity for sterilization when this seems advisable.

During labor, the physician is chiefly concerned with the shortening and lightening of the second stage. It is probable that the uterine contractions do not in themselves greatly burden the circulation. Aside from the circulatory demands of increased metabolism and the psychic effect of the pain, such contractions of masses of involuntary muscle may be disregarded as important factors in any circulatory disturbances accompanying pregnancy. It is the play of the entire mechanism of the second or expulsive stage that is the chief danger to the compromised heart. The contraction of the voluntary muscles, the forced depression of the diaphragm, the holding of the breath during efforts to expel the fetus, is a strain as real as it is immeasurable in precise terms of work performed. The expulsion of the fetus with the consequent decrease in intraabdominal pressure is itself a source of embarrassment to the circulation. The intrathoracic pressure relations are altered greatly and suddenly. As Pouliot[13] has insisted, the lower position of the diaphragm immediately following parturition has an effect on the lungs not unlike that following the removal of a large pleural effusion, under which conditions one may rarely observe an acute edema of the lungs. This, in Pouliot's mind, explains the pulmonary edema occurring so frequently immediately postpartum. That this sudden decline in intrathoracic pressure is more of a menace in mitral stenosis than in other conditions is understandable when we consider the greatly increased tension in the pulmonary circulation in this valve lesion. It would appear that herein lies one of the important causes which justifies the view that mitral stenosis is attended by more risk in childbearing than is any other heart lesion. The necessity of making this second stage of labor as short and easy as possible has long been recognized by obstetricians. The means of accomplishing this must vary with each case and are important enough to demand separate discussion.

COMPLICATIONS

With the completion of labor, the menace which threatens the decompensated heart is by no means abolished. The obstetrician is well aware of the possibility of an acute pulmonary edema within a few hours, or even twelve or fourteen days following parturition. The cause of this dangerous symptom is a matter of debate. Three factors, operating singly or in combination, seem important. We have already mentioned the sudden diminution of pressure upon the lung resulting from the expulsion of the fetus which, particularly in mitral stenosis, with its greatly increased pressure in the pulmonary circulation, may act in the same manner in promoting pulmonary edema as the with-

drawal of a large pleural effusion. Similar train of cause and effect might rarely be observed in mitral insufficiency as well.

The mechanical theory of Welch may be invoked as a fairly satisfactory explanation of certain types of pulmonary edema,—particularly those following severe toxemia with prolonged arterial hypertension. It is probable that sudden failure of the left ventricle under the strain with continued normal and, therefore, disproportionately great activity on the part of the right ventricle may result in great pulmonary congestion and transudation. This type of circulatory imbalance often occurs several days after parturition and especially with the establishment of lactation. The metabolic demands of this function, the redistribution or increase in amount of body fluids may, each or all, be factors. There would seem little question of the important rôle of increased blood volume in this type of pulmonary edema of pregnancy. The frequent practice of flooding the body with enormous quantities of water when toxemia is present is one that requires great discrimination. When toxemia is accompanied by arterial hypertension or circulatory stasis, great increase in fluid intake may spell disaster. Toxic effects on the endothelium of pulmonary capillaries increasing permeability is doubtless a real but variable and imponderable factor. So is the toxic degeneration of the myocardium in the so-called mechanical type. Bronchospasm as a cause of the pulmonary edema of childbearing seems of little importance.

The immediate treatment of severe pulmonary edema calls for prompt bleeding. During labor, this may be accomplished by promoting loss of blood from the uterus. Otherwise, venesection is demanded. Since reduction in venous pressure follows only after the withdrawal of sixteen ounces or more of blood from the individual of average weight, at least that amount should be taken. Morphine, grains one quarter, hypodermically is essential. Entire quiet is enforced. In cases with arterial hypertension, nitroglycerine is valuable. One one-hundredth grain is given every five minutes until effect is had. The further treatment of such a case is that of the decompensated heart after labor.

TREATMENT OF THE DECOMPENSATED CASE AFTER LABOR

This includes bed rest, the Karrel diet in cases with edema, otherwise moderate restriction of fluids, digitalis to tolerance, sedatives as required, and diuretin at intervals if indicated. Massage and passive movements are begun promptly. Later, exercise is allowed provided the response is satisfactory. If a given effort does not result in an acceleration of the pulse of more than twenty beats per minute, and if, after two minutes, the rate return to within five or ten beats of the previous resting rate, the bounds of cardiac capacity have probably not been passed. The more complex and, theoretically, more exact

tests of the functional capacity of the myocardium are probably no more satisfactory than this simple exercise test. Governed by this test, the patient may gradually resume activity. In general, about one month of rest in bed or chair, followed by graduated activity under careful supervision is necessary. In cases exhibiting dilatation of the heart, the return of the apex impulse to its former site, a change to a more normal character, and the disappearance of the murmurs indicating a relative mitral insufficiency, are important preliminaries to activity and must, in selected examples, supplement the exercise test.

Digitalis should be continued in therapeutic doses during this convalescent period. The person of average weight will tolerate about twenty minims of the standard tincture daily. Some will profit by larger amounts. Others will exhibit extrasystoles, ventricular tachycardia, nausea, or other evidences of toxic action, and should have the drug at intervals rather than by a continuous method.

Many women tend to gain weight unduly during the months following childbirth. The danger of this to the cardiac patient is obvious. In controlling this matter, studies of the basal metabolism and chemical survey of the blood are desirable and may unmask metabolic or endocrine defects of importance. Where such refinements are not available, quantitative restriction of diet with disproportionate reduction in carbohydrate often gives practical results that are satisfactory. Judicious exercise and thyroid extract given with caution may supplement this diet.

Anesthesia.—It is our opinion that much of the apprehension of the possible ill effects of anesthesia in instances of chronic heart disease is not justified by practical experience. In our series, a judiciously given ether anesthesia was, without exception, well borne. In these cases, ether has been preferred to other anesthetics by the operating staff.

Mitral Stenosis.—While the not infrequent statement that this lesion is incompatible with a safe pregnancy is certainly not warranted by experience, mitral stenosis should be regarded more earnestly than other valvular defects. This is especially true of cases giving such evidence of great narrowing of the mitral ring as a lengthy murmur and thrill extending throughout diastole, constant rapidity of the heart rate, cyanosis, exertional dyspnea, and signs of stasis however slight. In such cases, pregnancy should be avoided or terminated. Curiously, the danger of embolism does not seem to be increased by pregnancy.

One case of mitral stenosis (hospital number 44733) illustrated very convincingly the mechanism of the Graham-Steell murmur of relative pulmonary insufficiency. Before labor the signs were those of a marked involvement of the mitral valve with stenosis and insufficiency and accentuation of the second sound at the pulmonic area. After delivery, a soft diastolic murmur appeared at the pulmonic area and

in the third and fourth spaces to the left of the sternum. With this, the second sound at the pulmonic area became very faint. In ten days the diastolic murmur vanished and the pulmonic sound was again loud and distinct.

Auricular Fibrillation.—In 1921, Thomas[14] remarked that he could find in all the literature no "mention of cardiac arrhythmia in pregnancy or any cases of auricular flutter or fibrillation accompanying that state." To Thomas' report of a seriously decompensated case with fibrillation that went through labor without great embarrassment of the heart, I add four cases showing fibrillation of the auricle. All of these underwent therapeutic abortion excepting one in whom the arrhythmia was transitory and immediately followed labor. In this case, within a few hours, a normal rhythm was restored and no cardiac symptoms have since been experienced.

Auricular fibrillation being most often a sequel of marked mitral stenosis would, in general, interdict pregnancy. However, those rare cases with adequately maintained circulation and some margin of cardiac reserve power might bear children with reasonable safety.

Auricular Flutter.—This occurred once as an incident immediately postpartum. It vanished within a few hours and no trace of heart trouble could be detected several months later.

THE PERMANENT EFFECTS OF PREGNANCY ON THE DISEASED HEART

Do pregnancy and labor permanently reduce the reserve power of the diseased heart? Only careful study of cases for several months or years postpartum can give a satisfactory answer to this question. In general, experience indicates that the scriptural principle applies: "To him that hath shall be given, and from him who hath not shall be taken away even that which he hath." The woman with little dilatation and fair reserve power in the heart muscle may, six months after parturition, reveal no decline in circulatory efficiency, while the more gravely afflicted show permanent injury. Even among this latter class, however, are startling exceptions. One may see with astonishment a woman with a narrow mitral ring and permanent auricular fibrillation, or one with recurring and severe decompensation, pass through more than one labor without apparent increase in cardiac discomfort. Satisfying explanations of some of these cases cannot be given. Of one matter, however, we may be certain; such good results depend very largely upon the care given before, during and after labor. Of greatest importance is the prevention of serious decompensation by the prompt institution of medical treatment with the earliest onset of its symptoms. If this is accomplished, if the labor is made free from undue stress by appropriate obstetric measures, and if resumption of activity postpartum be carefully adjusted to the

capacity of the heart, the patient may, as a rule, look forward to a restoration of cardiac reserve power at or near the former level.

For this, there seems good reason. Pregnancy is a physiological process. Cardiac decompensation accompanying it is the result of added burdens largely of a mechanical sort, such as increased blood volume, increased body bulk, increased metabolic demands, high position of the diaphragm with its effect on heart and lungs. In the absence of toxemia, such decompensation is not the result of any inflammatory or degenerative process involving the myocardium such as so generally underlies the cardiac failure of most other states. The therapeutic problem is, therefore, measurably simpler, more promising and more successful in pregnancy than under most circumstances.

Examination of some thirty of our cases from six weeks to twelve months postpartum reveals the satisfactory fact that there are few exceptions showing either by history or physical signs appreciable decline in circulatory efficiency as a result of the pregnancy. Exceptions have been those becoming obese, those suffering further inroads of rheumatic infection and one example of syphilitic aortitis that has continued on the expected downward course.

Details of the forty cases upon which this paper is based are found in the accompanying tabulation. Of the two deaths, one was a case of influenzal pneumonia with decompensated heart and edema of the lungs. She entered in a moribund state and died shortly after admittance. The second death was in a case of marked mitral stenosis delivered in the Sloane Hospital one year before. This patient was advised to consent to termination of pregnancy, but refused, dying from acute dilatation the day following admission.

THE EFFECT OF CHRONIC HEART DISEASE UPON THE FETUS

In this series of forty cases, there were ten fetal deaths. Of these, three were therapeutic abortions, four were stillbirths, one died about one hour after birth and one was a stillbirth following a postmortem cesarean section. One maternal death occurred without delivery. As might be expected, the fetal mortality was greatest in mitral stenosis with concomitant toxemia of pregnancy. The influence of chronic valvular disease of the heart upon the size of the fetus is of interest. The average weight of the children of the cardiac cases delivered at term and here reported was six pounds and ten ounces. This may be compared with the average weight at birth of seven and one-tenth pounds as given by Holt.

SUMMARY

Experience of two years with forty cases of chronic valvular disease in pregnancy has emphasized the importance of the following points:

1. An antenatal clinic is essential in detecting the early evidences of decompensation.

2. All cases showing decompensation in any period of pregnancy should be admitted to the hospital and given medical treatment, no matter what the stage of pregnancy.

3. Induction of labor should never be attempted in a case of decompensation until thorough trial has been made of medical measures.

4. The response to medical treatment of the average case of chronic valvular disease of the heart in pregnancy is satisfactory, and the same principles govern its treatment as govern the treatment of such cases not associated with pregnancy.

5. The termination of pregnancy in the presence of chronic valvular disease of the heart is not a matter about which hard and fast rules can be laid down. In general, if decompensation occurs early in pregnancy, or if it does not respond to medical treatment, if it has occurred and been severe despite proper care in previous pregnancies and if the signs and symptoms indicate serious lesion, termination is usually wise.

6. The method by which pregnancy should be terminated is largely an obstetric question, the point of greatest importance being the guarantee of a short and easy second stage.

SUMMARY OF CASES

1. Age twenty-six. Gravida 2. Admitted August 31, 1921. Complaint of headache and edema when admitted. Signs of mitral stenosis. Pulse rate 100. Delivered normally on day of admission. Discharged September 12, 1921, in good condition. Diagnosis: mitral stenosis.

2. Age twenty-two. Primipara. Admitted November 26, 1920. History of sore throats, scarlet fever and tonsillitis. Admitted with mild cardiac insufficiency. Signs of mitral stenosis. Delivered at term by Maurician extraction. Postnatal examination, June 14, 1921: No symptoms of heart weakness, slight hypertrophy; response to exercise, fair. Diagnosis: mitral stenosis, moderate hypertrophy, little dilatation. General outlook good.

3. Age thirty-seven. Gravida 3. Admitted December 13, 1919. Tonsillitis and arthritis in 1911. Symptoms of moderate cardiac insufficiency with signs of mitral stenosis. Blood pressure, 190/130; albuminuria. Improvement under treatment. Normal delivery at term six days after admission. Postnatal examination, June 14, 1921: Dyspnea on exertion, moderate hypertrophy of heart; blood pressure, 190/130. Diagnosis: essential hypertension, mitral stenosis; hypertrophy, slight dilatation of heart; toxemia of pregnancy.

4. Age thirty-three. Gravida 3. Admitted January 12, 1921. Pneumonia at 18 followed by heart trouble. Rheumatic fever at 13. Admitted with cardiac insufficiency, considerable hypertrophy and dilatation, and signs of mitral stenosis. Improvement under treatment. Delivery January 16th by medium forceps. Diagnosis: mitral stenosis. Outlook good.

5. Age thirty-one. Gravida 4. Admitted August 25, 1920. History of diphtheria and rheumatic fever. Cardiac symptoms for four years. Signs of mitral insufficiency on admission. Normal delivery on day of admission. Convalescence satisfactory. Diagnosis: mitral insufficiency.

6. Age twenty-six. Gravida 2. Admitted April 23, 1921. Past history negative. Dyspnea and signs of mitral insufficiency on admission. April 23, normal delivery. Postnatal examination June 28, 1921, showed the heart normal in size, position, and sounds. Diagnosis: mitral insufficiency, possibly relative.

7. Age twenty-three. Gravida 2. Admitted April 11, 1921. History of cardiac symptoms for several years. Signs of mitral insufficiency on admission but no signs of decompensation other than dyspnea on effort. Delivery was normal at term, April 11th. Postnatal examination July 5, 1921, showed no cardiac symptoms. A faint systolic murmur could be heard at the apex of the heart which was normal in size. Diagnosis: mitral insufficiency.

8. Age thirty-seven. Gravida 6. Admitted December 7, 1920. History of scarlet fever with kidney trouble at seven years, also a history of chorea and rheumatic fever. Threatened miscarriage three weeks before admission. Dyspnea with headache and edema and signs of mitral insufficiency and toxemia of pregnancy on admission. Blood pressure, 200/130; albuminuria, 100 per cent by volume. Labor was induced at the sixth month and a stillborn child delivered. Satisfactory convalescence. Diagnosis: mitral insufficiency; toxemia of pregnancy. At postnatal clinic, May 26, 1921, no complaint of cardiac symptoms was made. There was slight cyanosis and some edema of ankles, moderate hypertrophy and dilatation, signs of mitral insufficiency and hypertension. Blood pressure, 206/116. Outlook poor.

9. Age thirty-five. Gravida 4. Admitted January 29, 1920. History of influenza and malaria. Marked dyspnea and palpitation on admission. There were signs of mitral insufficiency. Blood pressure, 146/88. Labor was induced at the eighth month and a living child delivered. Condition on discharge was excellent. Diagnosis: mitral insufficiency. Examination in postnatal clinic, June 14, 1921, showed dyspnea, and edema of feet. There was obesity, the left ventricle was hypertrophied with signs of mitral insufficiency. Blood pressure was 180/110. Response to exercise was poor. Diagnosis: essential hypertension, obesity, relative mitral insufficiency. Outlook poor.

10. Age twenty-five. Gravida 3. Admitted August 28, 1920. History of tonsillitis and heart trouble. Eclampsia in 1917, since which, dyspnea, headaches and edema had been present. Admitted with signs of mitral insufficiency and toxemia of pregnancy. Blood pressure, 140/80; urine, albumin, 60 per cent by volume with casts. Five days later, delivery was normal after which the condition improved. Diagnosis: mitral insufficiency, toxemia of pregnancy.

11. Age thirty-nine. Gravida 7. Admitted October 12, 1920. History of heart trouble at 6 years and rheumatic fever in 1912. Admitted with edema, a trace of albumin in the urine, and signs of mitral insufficiency. Normal delivery on the day of admission. Diagnosis: mitral insufficiency, slight decompensation. Examination in postnatal clinic, June 14, 1921, showed moderate hypertrophy and signs of mitral insufficiency. No dyspnea except on unusual exertion or excitement. Outlook good.

12. Age thirty-two. Gravida 4. Admitted July 30, 1921. Moderate dyspnea, edema and cyanosis on admission. Very obese. In hospital nine weeks before labor, two weeks of which were spent in bed. Digitalis administered. Considerable hypertrophy but little dilatation; compensation fairly good. Normal delivery September 12. Condition improved. Diagnosis: mitral stenosis and insufficiency.

13. Age twenty-four. Primipara. Admitted February 3, 1920. Rheumatic fever at 12. At 21, was in the hospital because of heart condition. Admitted with marked decompensation, cardiac dilatation and hypertrophy, rate of 140, and signs of mitral stenosis and insufficiency. Moderate toxemia of pregnancy. De-

livery one week later at term by medium forceps. Child stillborn. Postnatal examination, May 26, 1921, poor response to exercise, marked hypertrophy, moderate dilatation. Diagnosis: mitral stenosis and insufficiency; toxemia of pregnancy. Outlook poor.

14. Age thirty-eight. Gravida 4. Admitted June 19, 1920. No antenatal care. Admitted in labor. Heart: hypertrophy and slight dilatation; some edema, infected teeth. Labor normal. June 28, 1921, at follow-up clinic, complained of dyspnea. The heart showed moderate hypertrophy and dilatation, mitral stenosis and insufficiency with impaired reserve power. Outlook only fair.

15. Age thirty-five. Gravida 2. Admitted July 12, 1921. Rheumatic fever as child. Latent cardiac trouble for years. No trouble with heart in labor nine years before. At sixth month of present pregnancy, had acute tonsillitis followed by palpitation, rapid, irregular heart action, dyspnea, nervousness. Slight hypertrophy, rate 130 with periods of grouped extrasystolic irregularities. Systolic and presystolic murmurs at apex. No stasis. Treatment: rest and digitalis with improvement. July 21, Cesarean section. Convalescence and eventual heart condition satisfactory. Diagnosis: mitral stenosis and insufficiency, acute infectious myocarditis following tonsillitis.

16. Age twenty-eight. Primipara. Admitted April 22, 1920. History of poliomyelitis at 2. Cardiac insufficiency for nine years, marked for past six months. Admitted with edema, albuminuria, and other evidence of pregnancy toxemia, mitral stenosis and insufficiency. Blood pressure 188/110. No improvement under treatment. Labor induced at the sixth month, four days after admission, after which, the heart became compensated. Diagnosis: mitral stenosis and insufficiency, secondary anemia, severe toxemia of pregnancy.

17. Age twenty-four. Primipara. Admitted February 17, 1921. Admitted with marked dyspnea, orthopnea, and edema. The heart was so over acting that no definite evidence of valvular lesion could be made out. There was albuminuria. Delivery, seventeen days later, was at term and normal, but during labor the pulse rate was 170 and the quality very poor. Compensation was restored soon after labor. Postnatal examination, June 14, 1921, showed cardiac hypertrophy of moderate grade. Diagnosis: mitral stenosis and insufficiency. Prognosis good.

18. Age twenty-eight. Gravida 3. Admitted July 10, 1920. Influenza and pneumonia in 1919. Earlier in pregnancy was admitted with dyspnea and edema, moderate hypertrophy of heart and congestion of lungs and was treated for fifteen days with improvement. Normal delivery on day of second admission. Convalescence satisfactory and condition good when discharged. Diagnosis: mitral stenosis and insufficiency.

19. Age thirty. Primipara. Admitted September 21, 1920. Rheumatic fever at 5 and at 10. Known cardiac disease since 12. In the ninth month of pregnancy, partial decompensation with marked hypertrophy and signs of mitral stenosis and insufficiency. Marked improvement after rest. Normal delivery September 28, 1920. Diagnosis: mitral stenosis and insufficiency.

20. Age twenty-one. Primipara. Admitted February 10, 1920. History of measles only. Admitted with marked dyspnea and enlargement of the heart, a rate of 120, and signs of mitral stenosis and insufficiency. Version and breech delivery at term on day of admission. Postnatal examination, June 28, 1921. Dyspnea on effort, marked hypertrophy and moderate dilatation. Diagnosis: mitral stenosis and insufficiency. Outlook fair.

21. Age twenty-eight. Gravida 2. Admitted February 26, 1921. Dyspnea and edema for two years, worse during pregnancy. Hemoglobin, 35 per cent. Ova of

uncinaria in stool. Heart: moderate hypertrophy and slight dilatation. Systolic and presystolic murmurs at apex. Spleen enlarged. Wassermann, ∓. Treatment: transfusion, digitalization, neosalvarsan. Delivery normal, March 6th. Much improved on leaving hospital. Diagnosis: mitral stenosis and insufficiency.

22. Age twenty-seven. Gravida 4. Admitted August 9, 1920. History of positive Wassermann reaction. Mild cardiac insufficiency for two months. Admitted with the signs of mitral stenosis and insufficiency of moderate degree, and of healed pulmonary tuberculosis. Delivery normal. Diagnosis: mitral stenosis and insufficiency.

23. Age twenty-eight. Gravida 2. Admitted March 15, 1920. History of rheumatic fever at 18, of repeated quinsy, of diphtheria and scarlet fever. Heart trouble for two years. Admitted with hypertrophy and dilatation, signs of mitral stenosis and insufficiency. Marked improvement followed treatment. April 2, normal delivery at term. Postnatal examination May 26, 1921. No symptoms of cardiac insufficiency. Diagnosis: mitral stenosis and insufficiency.

24. Age twenty-six. Gravida 2. Admitted September 26, 1921. Repeated tonsillitis. In Sloane Hospital in April, 1920, with toxemia of pregnancy. Labor induced. On admission, decompensated, showing dyspnea, cough, edema. In December, 1921, in hospital three weeks. Because of decompensation, abortion advised but refused. September 26, heart hypertrophied and moderately dilated, rate 100, regular. Presystolic and systolic murmurs at apex, stasis at bases of lungs, massive edema of legs and feet. Advancing decompensation. Death, September 28, 1921, from pulmonary edema.

25. Age twenty-seven. Primipara. Admitted May 25, 1920. Influenza in 1918. Severe toxemia with blood pressure, 225/120; albuminuria, edema, signs of mitral stenosis and insufficiency. Delivery the day following admission by forceps. Convulsion during labor and two afterwards. Full-term stillbirth. Diagnosis: mitral stenosis and insufficiency, eclamptic toxemia of pregnancy.

26. Age twenty-nine. Gravida 3. Admitted April 17, 1920. Past history included influenza and five attacks of rheumatic fever; also, diphtheria, scarlet fever, tonsillitis, and chorea. Marked cardiac insufficiency, hypertrophy and dilatation. Delivery, April 29th, was normal at term. Following delivery for ten days had Graham-Steell murmur. Discharged in good condition. Outlook satisfactory. Diagnosis: mitral stenosis and insufficiency, transitory relative pulmonary insufficiency.

27. Age twenty-seven. Gravida 2. Admitted March 9, 1920. History of rheumatic fever at sixteen years. Headache and slight cyanosis present but no dyspnea or edema. There were signs of mitral and aortic insufficiency with moderate enlargement of the left heart. Delivery was normal on the day of admission. Diagnosis: aortic and mitral insufficiency, mitral stenosis.

28. Age twenty-four. Gravida 1. Admitted March 21, 1920. History of tonsillitis, quinsy, and rheumatic fever. Cardiac symptoms for one year. Dyspnea, orthopnea, edema and some cyanosis on admission. Signs of aortic and mitral insufficiency with auricular fibrillation and a pulse rate averaging 100. There were a few coarse rales at the bases of the lungs. By medical treatment, compensation was restored in five days and the patient left the hospital returning in labor March 21st. Delivery was normal at term. Postnatal examination on June 14, 1920, revealed some cyanosis, a pulse rate of 88 and regular, marked hypertrophy and dilatation with general outlook poor. Diagnosis: mitral and aortic insufficiency.

29. Gravida 2. Admitted June 4, 1921. History of dyspnea, edema, and signs of mitral stenosis and insufficiency beginning with and continuing since first preg-

nancy. Labor, June 10, was terminated by forceps, eliminating the second stage. Postnatal examination, June 28, 1921, showed moderate hypertrophy and dilatation with excellent compensation. Diagnosis: mitral stenosis and aortic insufficiency.

30. Age twenty-one. Gravida 1. Admitted October 14, 1919. History of several attacks of rheumatic fever. Entered hospital with failing compensation, cough, and extreme edema. After six weeks of medical treatment and a trial labor of five hours, a cesarean section and sterilization were done. Convalescence was satisfactory. Diagnosis: mitral stenosis and insufficiency, possible aortic insufficiency, functional pulmonary leakage.

31. Age thirty-nine. Primipara. Admitted November 17, 1919. History of rheumatic fever nineteen years before. Admitted in labor with marked edema. Signs of mitral and aortic insufficiency. Labor was terminated by a medium forceps delivery November 18, 1919. Postnatal examination, May 26, 1921, showed marked hypertrophy, little dilatation and fairly good compensation. Diagnosis: mitral stenosis and insufficiency; aortic insufficiency.

32. Age thirty-nine. Gravida 6. Admitted March 17, 1920. History of measles. Slight cyanosis and edema on admission. The liver was palpable. Normal delivery at term ten days later. Condition satisfactory. Postnatal examination, May 26, 1921, revealed considerable hypertrophy but little dilatation. Diagnosis: mitral stenosis and aortic insufficiency.

33. Age thirty-eight. Gravida 10. Admitted April 17, 1920. History of subacute rheumatic fever. Dyspnea and marked edema for one month previous to admission. Signs of mitral stenosis and aortic insufficiency. Delivery was at term, April 8th, the child living about one hour. Diagnosis: mitral stenosis and insufficiency; aortic insufficiency.

34. Age twenty-eight. Primipara. Admitted September 27, 1920. History of diphtheria at two years, frequent colds and tonsillitis. No cardiac symptoms noted on admission. Delivery the day of admission was followed by cyanosis and signs of some enlargement of the right heart with auricular flutter. Eight hours later, the pulse was normal. Postnatal examination, May 26, 1921, revealed no heart lesion. Diagnosis: transitory dilatation of the heart with auricular flutter immediately postpartum.

35. Age twenty-four. Gravida 3. Admitted September 27, 1920. No history of previous illnesses. The physical examination was negative on admission. Immediately following delivery, breathing stopped and the heart became totally irregular with auricular fibrillation. In an hour the respirations were normal and the heart again regular. The patient left the hospital in good condition without evidence of valvular lesion. Diagnosis: transitory auricular fibrillation immediately postpartum.

36. Age forty-two. Gravida 13. Admitted May 3, 1921. Rheumatic fever seventeen years before. Moderate decompensation for two years. Heart showed dilatation and hypertrophy, rate 92, totally irregular with pulse deficit of 40. Systolic and diastolic murmurs at apex. Liver and lungs congested. Spent one month in bed with disappearance of cyanosis and edema and pulse deficit. July 14, reentered hospital in labor. Version under ether. Mother's condition good. Child died. July 21, 1921, compensation was good and patient's condition was much better than on May 3rd. Diagnosis: mitral stenosis and insufficiency; auricular fibrillation.

37. Age thirty-nine. Gravida 9. Admitted April 13, 1920. Rheumatic fever and long standing cardiac disease. Cerebral embolism one month before admission. Heart: moderate hypertrophy and dilatation, auricular fibrillation, congestion of

TABLE I.—SUMMARY OF CASES

LESIONS	NUMBER OF CASES	ANTECEDENT HISTORY				COMPLICATION		TREATMENT						RESULT		
		RHEUMATIC FEVER	TONSILLITIS	CHOREA	SCARLET FEVER	SECONDARY ANEMIA	TOXEMIA OF PREGNANCY	REST AND DIGITALIS	NORMAL LABOR	INSTRUMENTAL DELIVERY	THERAPEUTIC ABORTION	CESAREAN SECTION	STERILIZATION	AVERAGE WEIGHT OF CHILD	MATERNAL DEATHS	FETAL DEATHS
Mitral Stenosis, pure	4	2	1				3	3	2	2				6 lb. 10 oz.	0	1
Mitral Insufficiency, pure	7	3	1	1	1		3	3	5	2				6 lb. 14 oz.	0	0
Mitral Stenosis and Insufficiency	14	4	3		1	6	4	6	9	2	1	1	1	6 lb. 12½ oz.	1	4
Mitral Stenosis and Insufficiency and relative Pulmonary insufficiency	1	1	1	1	1			1	1					6 lb. 9 oz.	0	0
Aortic and Mitral Lesions	7	5	1					3	4	2		1	1	7 lb. 3 oz.	0	1
Auricular Flutter	1		1							1					0	0
Auricular Fibrillation No Valve Lesion	1								1						0	0
Auricular Fibrillation with Valve Lesion	3	2		1		1	2	3			3				0	3
Aortitis	1							1	1					6 lb. 11 oz.	0	0
Post-Infectious Cardiac Decompensation	1						1					1			1	1
TOTAL	40	17	8	3	3	7	13	20	23	9	4	3	2	6 lb. 10 oz.	2	10

liver and lungs. Incomplete left hemiplegia. Therapeutic abortion in third month. Diagnosis: mitral stenosis and insufficiency, auricular fibrillation, cerebral embolism. Postnatal examination, May 26, 1921, moderate dyspnea, slight awkwardness of left side, moderate hypertrophy of heart, with fibrillating auricle.

38. Age thirty-seven. Primipara. Admitted July 24, 1920. History of chorea and malaria. Cardiac insufficiency for six months becoming severe the last ten days. Therapeutic abortion, August 5, 1920, in the second month of pregnancy. Diagnosis: mitral stenosis and insufficiency, auricular fibrillation.

39. Age thirty-five. Gravida 2. Admitted March 25, 1920. Wassermann reaction, four plus. Moderate hypertrophy of heart with systolic murmur at base. No decompensation. Normal delivery, March 25th, at term. Postnatal examination, May 26, 1921: exertional dyspnea, edema, evidence of dilatation of the aortic arch and a systolic murmur at apex and over aortic area. Diagnosis: syphilitic aortitis. Outlook poor.

40. Age twenty-one. Primipara. Admitted March 11, 1921. Influenzal pneumonia three weeks before. Moribund on admission with extreme cardiac decompensation. Died shortly after admission. Postpartum cesarean section, child died. Diagnosis: postinfectious cardiac decompensation.

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(*For discussion, see page* 83)