HEART DISEASE AND PREGNANCY.*

BY

J. CHALMERS CAMERON,

Montreal, Canada.

Professor of Obstetrics, McGill University.

THE mutual relationship between heart disease and pregnancy should interest every medical man, whether he is engaged chiefly in obstetric practice or whether he is devoting himself mainly to general medicine. The physician should be able to forecast the probable effects of pregnancy and labor upon those of his patients who are suffering from heart trouble, while the obstetrician should know how endocarditis and chronic valvular disease

*Read before the Canadian Medical Association, Section of Obstetrics and Gynecology, Ottawa, June 10, 1908.

may modify or derange the course of pregnancy, labor and the puerperium in patients whom he is expected to confine. Unfortunately, professional opinion regarding these matters, as a rule, is rather vague and uncertain, partly because of the persistence of certain venerable traditions and partly because teachers and text-books have devoted too little attention to the subject. They have not taught us clearly enough that the various forms of heart disease affect pregnant women in different ways and that it is fundamentally important to make an exact diagnosis before we attempt treatment. We cannot, even approximately, estimate the risks and dangers run by such patients or lay out a rational line of treatment for them until we have made out the condition of the heart and estimated the degree of compensation present, as well as the probability of its continuance. It used to be a common belief among the laity that heart troubles are rare in pregnancy, that such patients have a certain immunity from pregnancy and that even if they do become pregnant their cardiac troubles may improve or, at any rate, will not become worse. Routine examination of hospital cases shows that heart troubles exist in from 1 to 2½ per cent., while clinical experience demonstrates that cardiopaths are not unusually sterile, that they are not especially liable to abort, and that the majority of them may bear a living child safely, especially in their first pregnancy; but it is also certain that each recurring pregnancy aggravates their heart lesion and increases its dangerousness, and that disastrous results are most apt to occur in those women who have been weakened by several pregnancies occurring in rapid succession. Indeed, it is not uncommon for women to go through one pregnancy or even several without the existence of a heart lesion having even been suspected; this is particularly apt to occur in cases of mitral incompetence with good compensation. A routine examination of the heart in all pregnant women would not only make such an oversight impossible, but would also show how frequently cardiac complications do exist and how a little care will generally enable us to deliver such patients safely. Although it is undoubtedly true that endocarditis and valvular disease are serious complications of pregnancy, needing constant watchfulness and care, yet nevertheless it is equally true that the danger has been very much overrated and that the presence of heart disease is not a necessary nor even a very frequent indication for the induction of abortion or premature labor.

Causes and Course.—A cardiac lesion may have existed before pregnancy began; in such a case it may be said that pregnancy complicates the heart disease; or the cardiac trouble may have been latent, and by pregnancy it is developed and made known. Finally, it may begin during pregnancy or the puerperium, and then it must be considered a complication of pregnancy. In the latter case it is the result usually of rheumatism or some other intercurrent disease, or has been produced by toxemia or septic infection. But whatever may have been the cause, the heart lesion is always aggravated by pregnancy, especially after the fourth or fifth month, while, on the other hand, the course of pregnancy may be more or less seriously affected by it. There may be a miscarriage; or, if the patient reaches term, she may die during or after labor or in the puerperium; or even if she escapes with her life, she may be left with a crippled heart, more or less of an invalid for the rest of her days.

It is not hard to understand why pregnancy affects heart troubles injuriously. In the nonpregnant state, when an attack of endocarditis leaves a valve contracted or incompetent, the circulatory balance is restored and maintained by a compensatory hypertrophy. But when pregnancy occurs in such a case, it complicates matters by calling upon the heart for still more compensation while the conditions are becoming less and less favorable for such repair with the advance of pregnancy. Tension is increasing, the nutritive quality of the blood is impaired, since it must not only provide material for the growth of the fetus, but must also carry off the increasing amount of waste matter eliminated by it. The enlarging uterus and the increasing intraabdominal pressure crowd up the diaphragm, displacing the heart, preventing the full expansion of the lungs and consequently limiting their oxygenating power. It is easy to understand, therefore, how it becomes increasingly difficult to establish and maintain compensation as pregnancy proceeds. It may be urged that some observers deny the existence of cardiac hypertrophy in normal pregnancy, claiming that the increase in the area of cardiac dullness is due to upward displacement of the heart and not to hypertrophy. Other observers, too, assert, as the result of experiment and actual measurement, that the lung capacity remains constant in normal pregnancy. But it is hard to admit the validity of such claims, when every day we see for ourselves how easily breathlessness on exertion is produced and how quick and shallow the respira-

tion usually is in pregnant women. As the result of clinical observation, it seems only reasonable to infer that the capacity of the lungs is decreased and expansion becomes limited directly in proportion to the growth of the uterine tumor. It is also well established clinically that in cases of pregnancy complicated with heart disease, it is rare to find urgent dyspnea in primigravidæ, and that when it does occur in multigravidæ it begins usually about the fourth or fifth month and becomes more distressing as the abdomen enlarges, and that very slight causes may then produce heart failure.

Prognosis.—As regards their degree of dangerousness, cardiac lesions in pregnancy are usually arranged in the following order: *mitral stenosis, aortic insufficiency, mitral insufficiency* alone or complicated with stenosis or some aortic lesion.

Practically, however, such a classification must be accepted with caution, for it is difficult or impossible to base a prognosis merely upon the kind of valvular disease present in any given case. The primiparity or multiparity of the patient, her general health and nutrition, the condition of the kidneys, the amount of compensation present and the ease with which it is upset or restored, the patient's ability to remain quiet in bed free from exertion or excitement, and the way in which she responds to treatment—all these things are of more importance in estimating the probable result than the kind of anatomical lesion present.

Mitral Stenosis.—Since less blood than usual passes through the narrowed mitral orifice, such patients, as a rule are poorly nourished, weak and anemic. In labor, the first stage does not cause any particular distress; not until the bearing-down pains of the second stage begin, do symptoms of constitutional disturbance appear. Then, the glottis being closed, the lungs tense with air, the diaphragm depressed, the uterus and abdominal muscles in strong and frequently recurring contractions, an increased amount of blood is driven into the veins and forced along toward the already engorged right heart. Relief cannot come from the front, because the narrowed mitral allows but an insufficient quantity of blood to pass through into the left ventricle, and the blood is dammed back upon the left auricle which consequently distends, thus offering still greater resistance to the unloading of the lungs and engorged right heart. The symptoms, therefore, are decreased blood-pressure, an irregular, small, rapid, flickering pulse and cyanosis which tends to increase. The character of the pulse is explained by the fact that enough

blood does not enter the left ventricle for it to pump a full stream, and the cyanosis is explained by the increasing engorgement of the right heart. As the bearing-down efforts continue, the strain increases, and narcosis or death may occur if the tension is not relieved. At the close of the second stage, if free hemorrhage takes place from the uterus, the right heart may be somewhat relieved; but whether it is or not, a new danger must be faced presently. On account of the cessation of the placental circulation and the contraction of the uterus, a large quantity of blood is liberated and is forced into the veins. Under this new strain, the right heart may become so embarrassed that it may cease to contract and sudden death may occur.

What, then, is the danger of labor in a case of mitral stenosis? Evidently it lies in overdistension of the right heart brought on by the frequent bearing-down pains of the second stage, and later by the blood from the uteroplacental vessels being suddenly poured into the engorged veins during the third stage.

And what is the remedy? Shortening the second stage, preventing or moderating the bearing-down pains by means of morphia, chloroform or ether and delivering artificially as quickly as possible. After the birth of the child, encouraging free bleeding from the uterus and if that does not relieve the cyanosis and strengthen the failing, shabby pulse, venesection (eight to sixteen ounces) will diminish the strain and tide the patient over the critical point of pressing danger. As bleeding from the uterus is to be encouraged, not checked, ergot should not be given and frictions to the fundus should not be used. After the urgent danger is over, perfect rest should be secured, by a small hypodermic of morphia if necessary, and the heart should be stimulated by hypodermic injections of strychnia and digitalin.

Mitral Insufficiency.—This is the commonest of the heart complications of pregnancy. In most of the cases there is good compensation and labor terminates safely, consequently this form of cardiac lesion is generally considered to be less dangerous than mitral stenosis or aortic disease. This hopeful view should not lead us to underestimate the danger, or to be less watchful and careful in the matter of treatment. When the insufficiency is great, especially if there is also tricuspid regurgitation, no form of heart disease is more desperate or has a gloomier outlook. When a woman with a damaged mitral is subjected to the strain

of rapidly recurring pregnancies, sudden and unexpected breakdown is apt to occur. She may have gone through two or three pregnancies safely and without any alarming symptoms, but each time it is becoming harder to get good compensation, until finally the limit is reached and the crash comes. In mitral insufficiency, the hypertrophied left ventricle throws the blood with great force back against the leaky valve, the left auricle dilates, the lungs become engorged and cough and hemoptysis are common symptoms. Unlike mitral stenosis, the symptoms of mitral insufficiency are not much relieved by the emptying of the uterus, for the left ventricle goes on pumping back the blood as before, keeping up the distension of the left auricle along with the consequent engorgement of the lungs and right heart. The bearing-down pains of the second stage increase the action of the left ventricle and intensify the symptoms. Cyanosis, passive pulmonary congestion, edema, and irregular pulse of low tension are the prominent features. These patients convalesce slowly and need careful watching and judicious treatment for weeks or months. Relief comes only from resting the heart and building up compensation. The patient may die in a few days, weeks or months after labor, generally as the result of some imprudence. The signs of danger during labor are an irregular, failing pulse, restlessness and dyspnea, along with increasing cyanosis.

What should be done in such cases? The first stage should be allowed to go on naturally, meanwhile securing free, watery movements and steadying the heart with digitalin. When the second stage begins, morphia, choroform or ether will relax the muscles, check or moderate the bearing-down pains, allow the vessels to dilate and the blood-pressure to fall and the strain on the right heart to moderate. Delivery should be effected as soon as possible by artificial means, free bleeding from the uterus should be promoted and hypodermic injections of strychnia and digitalin should be given as circumstances require. During convalescence, careful feeding, tonics and prolonged rest in bed are necessary. The patient should be warned against the danger of a future pregnancy.

Mitral stenosis and mitral insufficiency often coexist, and in such cases the symptoms of stenosis usually predominate, the prognosis is graver than when either lesion exists alone.

Aortic insufficiency is a comparatively rare complication of pregnancy. The infrequency of its occurrence is due to the

fact that the degenerative changes in the aorta and its valves which are the chief cause of this lesion do not usually occur until middle life or later when the child-bearing period is almost or wholly over. The danger is most marked after the fourth or fifth month when increase in the blood-mass and in blood-pressure aggravates the regurgitation and disturbs left ventricle compensation, even although hitherto it has been fairly well adjusted. The symptoms are edema, dyspnea, restlessness and insomnia. The second stage is disastrous; the bearing-down pains increase the blood-pressure, regurgitation becomes worse, the left ventricle must work harder to empty itself, and finally the end comes by syncope.

This is the form of heart disease which calls for prompt emptying of the uterus, no matter whether the child is viable or not, whenever symptoms of distress manifest themselves. Immediate interference is positively indicated when such symptoms appear early or persist in spite of rest and treatment.

Aortic stenosis is also rare, and is seldom found without the mitral valve being more or less involved also. It is remarkable how often aortic stenosis and mitral stenosis are found to coexist.

Treatment of Pregnancy Complicated with Heart Disease.—As soon as the lesion is discovered, treatment should begin. The patient should be kept under observation and efforts made to guard and maintain compensation. It is not wise to wait till a breakdown occurs, for it is much easier to prevent the loss of compensation than to restore it after it has been lost. Such patients should be kept from excitement, overexertion and fatigue; long walks, hill climbing, running up and down stairs, hot baths, alcoholic drinks, going to theaters and concerts or to meetings in overcrowded, ill ventilated halls may do serious damage. Gentle exercise and plenty of fresh air are helpful when the patient's condition will permit. The bowels should be kept free, not only for the purpose of relieving the circulation, but also to lessen the chances of toxemia developing. Upon the appearance of such symptoms as dyspnea, palpitation, a feeling of oppression, cough, hemoptysis or edema, the patient should be put to bed at once and kept absolutely at rest. An icebag or cold compresses over the heart may give relief when the pain is severe. Frequent dry cuppings, sinapisms or turpentine stupes are useful when there is lung engorgement with dyspnea and local pain. A small hypodermic of morphia acts

like a charm when dyspnea is urgent. Edema with quick, irregular pulse calls for digitalis.* The prolonged and free use of strychnia often enables us to tide the patient safely along to term, or at least to the period of the child's viability.

If the patient is seen early and compensation is good, if it is the first pregnancy, or if there is no exhaustion from rapid childbearing, she may be allowed to go on to term, the compensation meanwhile being closely watched. If the patient is not seen until the heart symptoms are marked, the first endeavor should be to build up compensation by absolute rest in bed, dieting etc. If that cannot be done, it will be best to induce labor as soon as the child is viable, in the thirty-fourth or thirty-fifth week; if possible; not earlier than the thirty-second, nor later than the thirty-sixth week.

If only moderate compensation exists and the patient is allowed to go on to term, the chances of failure are increased. Clinical experience goes to show that better results can be obtained in moderately severe cases by inducing labor when the child is small and can be delivered easily, than by allowing the patient to go on to term with the chances of a large child and a difficult forceps or version operation.

When the lesion is grave, the patient exhausted and there is reason to believe that compensation will not be maintained, it is better to end pregnancy, whether the child is viable or not.

When labor comes on, the first stage should be allowed to terminate naturally, unless urgent symptoms arise. A free bowel movement may be obtained and digitalin may be given freely to steady and strengthen the heart. If this stage is prolonged, nutrition must be maintained and rest and sleep secured. When the os is fully dilated, the uterus should be emptied artificially under light anesthesia of some kind. A hypodermic of morphia at the beginning of the second stage will soothe the pains, and then only a few whiffs of chloroform or ether will be required for the easy and rapid termination of labor. If the os is undilated and rapid delivery is necessary, artificial dilatation of the os or multiple incisions of the cervix should be employed. After the birth of the child free bleeding from

*It has been shown experimentally that digitalis is an irritant to unstriped muscle and may excite uterine action. Consequently it has been urged that digitalis should not be given to pregnant women with heart trouble, for fear of bringing on abortion. According to our experience no such complication need be feared with moderate doses of the drug, and we are in the habit of giving it as freely as we give strychnin when it is indicated.

the uterus should be encouraged. Ergot and frictions of the uterus do harm by checking the loss of blood which might relieve the overburdened right heart. If cyanosis persists and the venous pressure is not relieved, venesection will give the promptest results. Some recommend nitrite of amyl or nitro glycerin immediately after delivery when there is great dyspnea and extreme high tension. I have no experience of these drugs.

It is better to allow the placenta to separate naturally and to come away without artificial assistance. If the strain of delivery has not been recovered from, the extra strain consequent upon the artificial separation and extraction of the placenta may prove the last straw, and the patient may collapse suddenly (Case No. 1273).

To obviate the ill effects of the rapid fall of intraabdominal pressure after the conclusion of the second stage, it has been recommended to apply a firm binder, or to put a sand bag on the abdomen immediately after delivery.

The puerperium needs careful management. Strychnia, digitalin, morphia, laxatives, tonics and careful feeding should be used as circumstances require.

The child should not be nursed, even in mild cases; the mother needs all her strength and all the nourishment and rest she can get to enable her to recover from the effects of pregnancy and labor. Prolonged rest in bed during the puerperium is imperative until involution is complete and compensation has become well established. It should not be forgotten that the heart needs to involute as well as the uterus.

Whether or not it is prudent for a young woman with a pronounced valvular lesion to marry is a question sometimes submitted to the physician. In such a case the risks of marriage are always great, for the occurrence of pregnancy is certain to aggravate the disease and shorten life. It is best always to discountenance marriage under such circumstances. Whether our advice is followed or not, it is our duty to give the warning and that with no uncertain voice.

Analysis of the Series of Thirteen Hospital Cases.—These thirteen cases occurred in a total of 1022. Several have not been included in the list because the heart lesion was moderate, the compensation good, the delivery spontaneous and normal, no special symptoms arose and no special treatment was required. If we include these milder cases, the proportion of pregnant women with cardiac lesions who were delivered in the Montreal

Maternity would be about 2 per cent. Of these thirteen, there were:

I-gravidæ, 3
II-gravidæ, 4
III-gravidæ, 2
VII-gravidæ, 2
IX-gravida, 1
XIV-gravida, 1

There were:

Under thirty years of age, 8.
Thirty years and over, 5.
The youngest was twenty-one; the oldest, forty.

A definite history of cardiac lesions previous to this pregnancy, 6.
Cardiac lesions arose probably during this pregnancy, 6.
Doubtful, 1

Labor at term,	8
Labor shortly before term,	2
Labor premature,	2
Undelivered,	1
	13
Labor spontaneous,	4
Labor, induced and high forceps,	1
Forceps deliveries:	
mid, 1	
low, 3	
	4
Version and extraction,	2
Extraction of breech,	1

Twins, 2 cases.

Anesthesia and narcosis during labor were used as follows:

Chloroform alone in	3 cases
Ether alone in	1 case
Morphia,	2 cases
Morphia and chloroform,	1 case
Morphia, chloroform and ether,	1 case
No anesthesia or narcosis,	4 cases

Results.—Mothers:

Discharged in fair or good condition 11 (1 undelivered).
Died, 2.

Children:

Born alive in 11 confinements out of 12, 12 (one case of twins).
Born dead in 1 confinement out of 12, 2 (twins).
If we exclude the two cases of myocarditis without valvular

lesion, the maternal mortality was one out of eleven (9.09 per cent.). In ten cases of valvular disease out of eleven, the mothers recovered and the children were born alive; in one case both mother and children (twins) died.

CASE NO. 859.—*Mitral stenosis with insufficiency.* Good compensation.

L. K., æt. twenty-five, II-gravida. Admitted, December 17, 1905; confined, December 17; discharged, December 30.

This patient entered the Maternity in labor, at term. From the first pain to the delivery of placenta, three and one-half hours. Labor spontaneous and rapid. Pulse after conclusion of third stage was ninety-six. Puerperium uneventful- discharged in good condition on the fourteenth day.

Clinical Note by Dr. Finley.—Slight choreic movements of face, hands and left foot. Cardiac impulse seen and felt near nipple, three and one-fourth inches from mid-sternal line. Marked presystolic thrill and strong, rather heaving impulse which extends up to third rib on left side. Marked diastolic shock at pulmonary cartilage. Dullness vertically to border of fourth rib; transversely from mid-sternal line, outward for three inches at fourth rib.

At apex, first sound replaced by systolic murmur; reduplication of second sound; systolic murmur transmitted into axilla and inward to lower end of sternum and upward to pulmonary cartilage. It is loud and blowing, best heard at apex.

At apex and just inside, there is an indistinct rumbling presystolic murmur, localized over a small area.

At aortic cartilage both sounds distinct; at pulmonary, faint systolic murmur and second sound enormously accentuated.

Pulse, low tension, normal volume, slightly collapsing, and slight capillary pulsation.

In neck is distinct pulsation in internal jugulars, more marked in left than in right. Liver and spleen not palpable.

CASE NO. 1405.—*Mitral stenosis with insufficiency.* Good compensation.

A. B., æt. thirty, II-gravida. Admitted, January 18, 1907; confined, January 25; discharged, February 7.

First pregnancy was normal, easy labor, good result for mother and child. In present pregnancy, general condition has been good, no dyspnea, can lie down without distress, some edema of left leg. Skin is pale; pulse small, regular and of fair tension; no capillary pulsation. Lungs clear; no precordial pain.

Apex beat visible in fifth interspace, five and one-fourth inches from mid-sternal line, palpable at same point, the impulse being forcible and fairly well localized. Cardiac dullness at level of fourth rib, extends transversely from right border of sternum to five inches to left of mid-sternal line.

At apex, both sounds are blurred; first sound replaced by loud, blowing systolic murmur, which is transmitted to axilla. There is

also a rough presystolic murmur, beginning right after second sound and leading up to the first. At the base, a soft, blowing systolic and diastolic murmur, heard best over pulmonary area, but also over aortic, P^2 sharp.

Labor at term was rapid. In one and one-half hours after the beginning of labor pains, the os was fully dilated. A hypodermic of morphia, gr. $\frac{1}{8}$, was given and the membranes were ruptured. The head descended slowly to the perineum, but in spite of strong pains, it was not expelled. As the pulse was growing very feeble, a hypodermic of strychnin, gr. $\frac{1}{30}$, was given, low forceps applied without anesthesia and the head easily delivered. Placenta separated in ten minutes and was pressed off. Another hypodermic of strychnin, gr. $\frac{1}{30}$, was then given. She was in good condition and made an uninterrupted recovery. Highest temperature was 98.8°. Child living and well.

A question might arise as to the significance of the basic diastolic murmur in this case. Such murmurs are so rarely functional that it is quite possible that the aortic orifice was involved as well as the mitral.

CASE NO. 2037.—*Mitral insufficiency.* Fair compensation.

C. G., æt twenty-one. I-gravida. Admitted, February 20, 1908; confined, March 18; discharged, April 1.

Patient has some edema, and more or less bronchitis at times. There is a moderate amount of albumin in the urine, with some casts.

Cardiac dullness did not extend beyond the nipple line. At the apex, a faint systolic murmur was heard, transmitted to the axilla. At times this murmur almost disappears; some days it is heard but faintly, other days very distinctly. At no time during pregnancy or while in hospital was there loss of compensation.

Labor.—First stage tedious; in second stage pains were regular and strong and advance was rapid. To moderate the severity of the pains, a little chloroform was given, to the obstetrical degree only. Suddenly toward the close of the second stage, the mask being over her face, she became extremely cyanosed, her breathing became rapid and shallow and then stopped altogether. Artificial respiration was begun at once and she gradually recovered. The placenta separated naturally and came away in fifty minutes; it was pale, firm and nearly half fibroid. The puerperium was uneventful. The highest temperature was 101.4°.

This case shows how suddenly dangerous symptoms may develop in the second stage. Misled by her good condition and the rapid course of her labor, it was thought that she might be allowed to deliver herself. Had forceps been applied early in the second stage, in all probability no difficulty would have been experienced. Her recovery was no doubt due mainly to the fact of her youth and her being a primipara.

CASE NO. 1272.—*Mitral insufficiency.* Fair compensation.

Heart Disease in Pregnancy, Labor and the Puerperium

Case No.	Heart Lesion	Lesion began this preg.	Began before	Age	Gravida	Labor at or before term	Pregnancy	Labor	Puerperium
859	Mitral Stenosis with Insufficiency	?	?	25	II	Term	? Admitted in Labor.	Spontaneous and Rapid	Uneventful
1405	Mitral Stenosis with Insufficiency	+	—	30	II	Term	Slight Edema	Rapid—Low Forceps	Uneventful
2037	Mitral Insufficiency	+	—	21	I	Term	Alb. Casts, Bronchitis	Sponta. Rapid Collapse	Uneventful
1272	Mitral Insufficiency	—	+	30	II	Term	Under observation throughout preg. for mitral lesion, alb. casts. Bronch.	Mid forceps	Uneventful
1273	Mitral Stenosis with Insufficiency	—	+	24	III	Prem.	Edema—dyspnea, cough	Version and Extraction—Twins	—
1290	Mitral Stenosis with Insufficiency	—	+	26	II	Term	Headache—Palpitation, dizziness, dyspnea	Low Forceps	Dyspnea, Cardiac Pain—Irregular Pulse
1444	Mitral Stenosis with Insufficiency	—	+	35	VII	Term	Cyanosis, dsypnea cough, hemoptysis, edema general	Twins—Breech presentations, extraction	Uneventful, recovery
1750	Aortic Stenosis	+	—	25	II	Term	Slight edema	Forceps failed—Version and Extraction	Irregular, Intermittent Pulse for a few days
3010	Aortic Stenosis	+	—	40	VII	Near Term	Slight dyspnea, edema, cyanosis, poor compensation	Low Forceps	Uneventful
1458	Aortic Stenosis and Mitral Insufficiency	—	+	35	IX	Prem.	Edema, cyanosis, dyspnea, rapid breathing, palpitation, cough, hemoptysis	Induction of labor in 35th Week (Krause) High Forceps	Uneventful
1807	Acute Dilatation of Heart with Endocarditis involving Aortic and Mitral Valves, the latter prob. secondary	—	+	25	III	Before Term	Edema, dyspnea, pulm. edema	Spontaneous, very rapid	Dyspnea for a week—then good recovery
1719	Myocarditis acute	+	—	24	I	—	General weakness and abdominal pain after a fall. Dyspnea, frequent micturition—Headache	Undelivered	—
2095	Myocarditis chronic	—	Probably	40	XIV	Term	Alcoholism—occasional fainting fits	Retained Placenta, collapsed 15 minutes after birth of child	—

Abbreviations: M = Morphia.
+ = in 3d and 4th columns, the appearance of heart lesions; in columns of results, *favorable* for mother
— in columns of results, *death* for mother and *born dead* for child

Cases Treated in the Montreal Maternity.

Anesthesia	Compensation	Highest Temp.	Results		Subsequent History	Remarks
			Mother	Child		
None	Good	—	+	+		Admitted in labor. History hard to get and unsatisfactory.
M. gr. 1-6 Chlorof.	Good	98.8°	+	+	Good Health	
(Obstet. degree)	Mod.	101.4°	+	+	Good Health	Towards end of second stage, during chloroform, patient became suddenly cyanosed, breathing became bad and stopped. Artificial respiration restored breathing. Afterwards good and steady recovery.
Chlorof. (light)	Good	—	+	+	Good Health	Membs. were ruptured and forceps applied to head in cavity to save the strain of second stage. Made a good recovery; discharged on 18th day. No special treatment required for heart trouble.
M. gr. ½ Chlorof. (light)	Poor	—	—	— —	—	After 4 days' treatment in hospital felt so much better that she insisted upon going home; 3 days later she returned in labor, in desperate plight, having walked ½ mile, mostly up hill. Both children presented by breech; chloroform was necessary to permit the hand being passed through O. I. Death occurred suddenly after manual extraction of the placentæ.
Chlorof. (light)	Mod.	99°	+	+	Feeling so much better she insisted upon going home on 11th day. A few months later, she died at home.	— —
M. gr. ½	Poor	99.6°	+	+ +	In good condition a year afterward	Heart trouble began after her first confinement.
M. gr. ½ Chlorof. Ether	Fair	99.8°	+	+	A few months later was in good condition. Just recently aborted	Albuminuria, with plentiful granular casts.
None	Poor	100°	+	+	No report	The history of previous pregnancies not satisfactory.
Ether	Poor	98.6°	+	+	Insisted upon going home on 14th day. Was then in fair condition. Thirteen days afterward was admitted to M. G. H. and died in 3 days of acute dilatation of heart	The heart trouble began 7 years ago and for 6 years she has been unable to do any hard work. Has been laid up several times with severe attacks, lasting 5 weeks. Her legs were never swollen till a few months ago. Was confined to bed for months before entering the Maternity.
None	Poor	101.°	+	+	Discharged much improved. Three months later entered W. G. H. Treated two weeks for chronic interstitial nephritis.	— —
—	Much improved	—	+	?		Was discharged much improved in health. In a few weeks was so well that she was able to go home to the West Indies, where she would be confined.
None	Poor	—	—	+		Was an alcoholic, lived a hard, rough life—drinking freely. The child steadily lost weight, seemed incapable of digesting anything. It died on the 8th day, having lost 640 grams.

1d *born alive* for child.

S. G., æt. twenty-six, I-gravida. Admitted, October 8, 1906; confined, October 8; discharged, October 25.

There was a history of rheumatism from early childhood, with frequent attacks of cardiac difficulty, loss of compensation, edema of legs, palpitation, etc. At the fourth month, she came under the care of Dr. Gordon, and then was found to have loss of compensation, with edema of legs, dyspnea, palpitation, hemoptysis, and signs of pulmonary infarction in the right base posteriorly. Under rest and treatment, all the symptoms disappeared except those of infarction, and she went along to term. On admission to hospital at the beginning of labor, it was found that the cardiac dullness extended transversely from the right sternal border to one-half inch beyond the nipple line, and a loud blowing systolic murmur was heard at apex, transmitted to axilla.

Labor pains began at 8 A. M. and were strong all morning: at 4 P.M. the pulse had become rapid and irregular, and as the os was fully dilated and the head well down, the membranes were ruptured artifically and forceps applied under light chloroform anesthesia. She stood the operation well and made a good recovery. A perineal tear, which was sutured unsuccessfully after labor, was repaired successfully six months later in hospital. She had at that time again signs of incompetence and pulmonary infarction, but she took ether well and made a good recovery. She is now living in New York and seems to be in good condition. During her stay in hospital on the two occasions it was not found necessary to give her any drug treatment for her cardiac trouble.

CASE NO. 1273.—*Mitral stenosis with insufficiency.* Poor compensation.

R. R., æt. twenty-four, III-gravida. Admitted, October 8, 1906; discharged, October 12; readmitted in labor, October 15; confined October 15, and died.

The patient on admission was poorly nourished, cyanosed, with dry, glossy skin and difficulty in breathing. The lungs were clear and there was no cough. Pulse was rapid, small and compressible; venous pulsation in vessels of neck. There was no precordial bulging, no visible pulsation. A systolic thrill was felt over apex, heart enlarged to left, a systolic and presystolic murmur heard at apex. Systolic murmur was rough and transmitted to axilla. At base, P^2 was sharp and accentuated, no murmur. Abdomen very tense, liver and spleen not palpable; no ascites.

She said that she had always been strong and well and that her first pregnancy and labor were normal, that her second pregnancy was normal up to the end of the third month when she fell down some steps and aborted. After that she was ill in bed for a month, with pain over the precordia, a hacking cough and expectoration. Since then she has been very short of breath, especially on exertion, and finds it hard to walk upstairs.

Never had swelling of the legs until two months ago, since which time there has been marked edema of both legs.

The patient was kept quiet in bed on liquid diet, the urine was collected and examined, the bowels regulated, and hypodermics were given of strychnia, gr. $\frac{1}{80}$, bis in die and digitalin, gr. $\frac{1}{100}$, 4 q. h. She improved markedly and, on October 11, Dr. Finley made the following clinical note:

Patient has slight orthopnea, but can lie down for a few minutes. Respiration hurried, thirty-two per minute. Cardiac impulse rather feeble and diffuse, about four inches from mid-sternal line; a distinct fine thrill. The dullness at fourth rib is one inch to right and three inches to left of mid sternal line. At apex first sound is sharp and accentuated and preceded by a rough, rumbling, presystolic murmur; second sound absent. At base P^2 moderately accentuated with reduplication. Pulse 114, rather small and compressible. There is short cough, but lungs are normal.

Under rest and treatment, patient began to feel so much better that she insisted upon going home on October 12, in spite of our warnings and remonstrances. Three days later (October 15), she came back again in labor, having walked part of the way from her home, a distance of about half a mile and mostly uphill. Her pulse was small and rapid, she was gasping for breath, severe pains were coming every two minutes, and she was in a very serious condition. On examination the os was found to be nearly dilated, and a hand was presenting. A hypodermic of morph. sulph., gr. $\frac{1}{8}$, and one of strychnin, gr. $\frac{1}{80}$, were given. The membranes were then ruptured and a hand came down, but almost immediately another bag of membranes presented. When it, too, was ruptured, another hand appeared. When the operator tried to pass his hand into the uterus, so much resistance was offered by the cervix that it was found necessary to give a little chloroform. When at last the hand was passed into the uterus, the first child was found to be lying obliquely, and the second vertically presenting by the head. With some difficulty the children were, one after the other, turned and delivered, both being premature and dead. The patient was then in such poor condition that she had to be stimulated with strychnia and saline infusions. In half an hour she had rallied fairly well. The placentæ could not be pressed off; so the hand was passed again into the vagina and one placenta which was completely detached was carefully removed by traction upon the cord; the other placenta had to be partially detached and then was easily delivered by traction upon the cord along with pressure from above. Almost immediately after the second placenta came away, the patient turned blue, gave a couple of gasps and died.

The following is the report of the autopsy held at the Royal Victoria Hospital by Dr. Klotz, pathologist to the Maternity:

Anatomical diagnosis:

Heart disease and pregnancy.

Chronic sclerotic mitral endocarditis.

Fibrosis of heart.
Hypertrophy of heart (right side).
Mitral stenosis with incompetence.
Hydrothorax.
Hydropericardium.
Edema of lungs.
Cloudy swelling and fatty degeneration of liver.
Enlarged spleen (cardiac spleen?)
Chronic intersitial nephritis.
Hypertrophy (compensatory) of kidney.
Puerperal state.

NOTE.—Death was due in this case to the condition of the mitral valve; its sudden appearance after delivery is likely a result of the sudden altered relation of the blood quantity to the vessels (combined, of course, with the extra strain of delivery).

The condition of the kidneys, an extreme grade of chronic interstitial nephritis with unilateral partial hypertrophy, is interesting.

CASE NO. 1290.—*Mitral stenosis with insufficiency.* Moderate compensation.

J. M., æt. twenty-six, II-gravida. Admitted, October 19, 1906; confined, October 19; discharged, October 30.

Patient was admitted in labor, at term, complaining of weakness, headache, dizziness, palpitation and dyspnea. There is a history of measles in childhood, but none of rheumatism or scarlatina. The first pregnancy, labor and puerperium seemed to have been normal. During the following summer she began to feel weak, short of breath on exertion, and had to rest when going upstairs. During the following winter she was fairly well, but when she became pregnant, the weakness increased, and as pregnancy went on she began to have the headache, palpitation and dyspnea more marked than before, increasing gradually till the present time. Always feels worse after eating; palpitation increases so much that she is afraid to eat and therefore takes but two meals a day. No precordial pain, no edema of legs.

Present condition.—Face flushed, skin and mucous membranes rather cyanosed, orthopnea well marked. Lungs clear, no cough or expectoration. Pulse small, compressible, eighty-eight per minute; no venous pulsation or capillary pulse.

Heart.—Visible pulsation in second left interspace none visible at apex. Marked systolic thrill palpable over whole precordia. Dullness vertically to second rib, transversely at level of fourth rib, two and one-half inches to right and four inches to left of mid-sternal line. At apex, first sound sharp, second sound faint. Loud, rough systolic murmur transmitted to axilla, and a blowing presystolic murmur, which is also heard at base. At base P^2 very loud, sharp and reduplicated; A^1 normal; A^2 clearly heard.

Patient was admitted about 10 A.M. in labor at term; at 1 P.M. the bag of membranes appeared at vulva. It was ruptured artificially, a few whiffs of chloroform were administered,

forceps applied and the child easily and quickly delivered. The pulse then became imperceptible at the wrist; hypodermics of strychnin, gr. $\frac{1}{80}$, and digitalin, gr. $\frac{1}{100}$, were given. The placenta was allowed to separate and ultimately was carefully pressed off. Patient rallied well and passed a good night. The next day she had an attack of dyspnea with severe pain over heart and the pulse became rapid and irregular. She was given morphia, gr. $\frac{1}{8}$, and digitalin, gr. $\frac{1}{100}$, and a little later strychnin, gr. $\frac{1}{80}$, hypodermically. For a week digitalin and strychnin were kept up regularly, with an occasional injection of morphia to relieve dyspnea and precordial pain. At the end of a week, a few fine crepitant râles were heard over front of chest, but no dullness. Dr. Finley made the following note:

Pulse 60, regular, slightly diminished tension. Thrill slightly marked at apex; impulse strong and jet-like, felt four and one-half inches from mid-sternal line. Relative cardiac dullness vertically at third rib, transversely one inch to right and four and one-fourth inches to left of mid-sternal line at fourth rib. Well marked systolic and diastolic shock over second left space, best felt two and one-half inches from mid-sternal line, but also felt in third space. This impulse is also visible in second space. At apex, first sound loud and thumping, second sound not heard. A prolonged rumbling, presystolic murmur transmitted to posterior axillary line and inward toward lower end of sternum. At base P^2 enormously accentuated and also reduplicated. Soft systolic murmur heard in this region. Lungs clear.

The dullness diminished somewhat, the cyanosis disappeared and soon she was able to rest comfortably in a semirecumbent position. Feeling herself so much better, she insisted upon going home. A few months later she died at home, her heart troubles having been aggravated by want of care.

CASE NO. 1444.—*Mitral stenosis with insufficiency.* Poor compensation.

J. R., æt. 35, VII-gravida. Admitted, February 9, 1907; Confined, April 30; discharged, May 15.

Patient has had five living children and one miscarriage; the oldest child is ten, the youngest three years old. Previous labors normal. After first labor, she had inflammation of lungs and ever since has had heart trouble. During the four succeeding pregnancies, she felt well for the first four months, and then began to complain of weakness and cough with spitting of blood at times. She was treated for three weeks in the Royal Victoria Hospital for mitral endocarditis until compensation was fairly good. She left on January 6, but compensation was soon lost, and she was readmitted on January 20, suffering from severe dyspnea, cough and frequent hemoptysis. She was transferred to the Maternity on February 9. She was then markedly cyanosed, with visible pulsation of vessels of neck; there was dry, hacking cough, but no pain. Respiration not labored, twenty-four per minute; good expansion equal on both sides. Vocal fremitus normal. A

few coarse crepitant râles in the right supra and infraclavicular fossæ, and numerous fine crepitations over both bases. Pulse 120, of small volume and fair tension; no capillary pulse. Over precordia pulsation not visible, but quite palpable and diffuse. Apex beat palpable in fifth interspace, four and one-half inches from mid-sternal line. Over this area a fine systolic thrill is felt. Cardiac dullness, vertical at upper border of third rib; transverse from right border of sternum to four inches to left of mid-sternal line at level of fourth rib. At apex both sounds blurred, and a rough presystolic murmur transmitted upward to pulmonary area and axilla. Outside nipple line, both sounds sharp and distinct. Presystolic murmur lasts though systole. At base P^2 is accentuated.

A slight trace of albumin in urine, with some pus cells and urates.

Patient was kept in bed for upward of six weeks, and then allowed up for part of the day. Digitalis was given steadily for a month, then discontinued for a week and given again for another week; after that, only an occasional dose was required.

Labor.—First stage prolonged. Slight pains only. When os was fully dilated, a breech was found presenting with membranes intact. On account of the cardiac condition, it was thought best to deliver without anesthesia. A hypodermic of morphia, gr. $\frac{1}{4}$, was given and the membranes ruptured artificially. A hypodermic of digitalin, gr. $\frac{1}{100}$, was given and the child was easily extracted. A second sac was then found presenting; it was ruptured and the second child, also presenting by the breech, was easily extracted. Both children were alive and well developed (term). Patient's pulse was not much affected by the delivery. A hypodermic of strychnin, gr. $\frac{1}{30}$, was given as a precautionary measure, and the placenta was pressed off in ten minutes. During the puerperium, her condition improved markedly. She was up on the eleventh day and discharged on the fifteenth. She was seen a year after and was in good condition. Quite recently she entered the maternity again and aborted at the seventh month. She was discharged in good condition.

CASE NO. 1750.—*Aortic stenosis.* Fair compensation.

A. R., æt. 25, II-gravida. Admitted, August 9, 1907; confined, August 13; discharged, August 29. Slight generally contracted pelvis. Previous pregnancy, labor and puerperium normal.

A pale, delicate woman; lungs clear on percussion and auscultation. No visible pulsation over precordia. At apex is felt a very slight systolic thrill at times, no special shock. Over base is a very well defined long-drawn systolic thrill, but no special diastolic shock. Cardiac dullness vertically at second interspace; transversely one-half inch to right and four and one-fourth inches to left of mid-sternal line. Percussion note is impaired over manubrium. At apex, both sounds audible. First sound accompan-

ied by soft systolic murmur, which can be heard in axilla and over entire thorax, but becomes of maximum intensity as the aortic cartilage is reached; here it is very long in duration, extending up to second sound and has a distinctly rough, musical quality. Pulses, equal and synchronous, 72, regular, but small in volume and low in tension. Vessel wall not sclerosed. Neither liver nor spleen palpable; no edema of shins; albumin present, granular casts plentiful.

Pains were strong and severe. Labor was allowed to go on till onset of second stage, when the membranes were ruptured. Head was not engaged: a hypodermic of morphia, gr. $\frac{1}{8}$, was given and repeated. An attempt to apply forceps without anesthesia failed. Anesthesia was started with chloroform and soon changed to ether. The head was at the brim in L. O. A. Another fruitless attempt was made to deliver with forceps, then version was performed and delivery effected with some difficulty. The child was deeply asphyxiated, but respiration was finally established. Patient bore the operation well, the pulse not changing markedly in character. During the rest of the day the pulse was irregular and intermittent, ranging from 64 to 72. The irregularity continued for several days, the lowest rate recorded was 56. By the ninth day the range was from 70 to 85. Patient sat up with a headrest on the twelfth day, in a chair on the fourteenth, and went home on the seventeenth day. No special medicinal treatment was required. A few months later she was seen and was in good condition.

CASE NO. 3010.—*Aortic stenosis.* Poor compensation.

M. T., æt. VII-gravida. Admitted March 31, 1908; confined, April 6; discharged, April 20.

Patient has had five living children and one abortion. Pregnancies and labor normal; puerperium normal, except after first child, when she was ill for several months. She now complains of dyspnea, cyanosis and edema of legs.

Note by Dr. Howard: There is marked visible pulsation in neck, but no special fullness of superficial veins. Pulsation seems to be systolic and to originate in the carotids where a definite systolic thrill can be felt. Lungs clear on percussion and auscultation. There is fairly forcible impulse over entire precordia, being marked in second, third, fourth and fifth interspace. On palpation, a well-marked purring, prolonged systolic thrill is felt over whole precordia to right as far as the mid-clavicular line and to left to anterior axillary line, the point of maximum impulse being in second right interspace, where it is extremely superficial and occupies almost the entire cycle. There is no special shock. At apex both sounds are heard, although first sound is almost obliterated by a loud systolic murmur; second sound is loud and sharp. This systolic murmur can be traced into axilla and upward into body of the heart. At base P^1 is loud; over aortic cartilage the systolic murmur is of extreme intensity, rather high-pitched and almost sawing in character; it is very

long-drawn, occupying most of the cycle. It can be heard everywhere over both backs. Pulse is small, sustained and of fair pressure; the vessel wall cannot be felt.

Labor.—The first stage was slow and tedious, but patient showed no ill effects. The membranes were ruptured as soon as the os was fully dilated, and the head came down well. Low forceps applied and delivery effected easily. Second stage lasted ten minutes. No anesthesia. Placenta pressed off in twenty-five minutes. Puerperium uneventful. Discharged on fourteenth day.

CASE NO. 1458.—*Aortic stenosis with mitral insufficiency.* Poor compensation.

L. A., æt. thirty-five, IX-gravida. Admitted, February 20, 1907; confined, March 28; discharged, April 10.

Patient has had six living children and two miscarriages. Oldest child is now fifteen, the youngest six years old; last miscarriage was in 1905 at three months. In previous pregnancies she suffered much from vomiting; labors were normal, the puerperium also; was always able to resume household duties soon afterward. Last menstruation began July 1, 1906. She entered the Royal Victoria Hospital on February 5, 1907, complaining of swelling of feet, palpitation and a sensation of smothering and choking. She was transferred to the Maternity on February 20.

Palpitation and breathlessness were first noticed seven years ago and lasted five months (three at home, two in the hospital). A year subsequently she was confined in the Maternity. Since then she has not been able to do any hard work. Had a severe attack three years ago and was again confined to bed for five months (at home). After that attack she was able to be about until last November when she took to bed and has not been up since, being troubled a good deal with cough and hemoptysis.

The legs were never swollen until this last attack.

She is poorly nourished; the skin and mucous membranes pale, with a cyanotic tinge. She assumes the orthopneic position; breathing is quiet: there is distressing cough, with blood-tinged expectoration; the lungs are clear.

There is pain over the precordia. Pulse, 100, soft, collapsing, of small volume and low tension. No capillary or venous pulse. The vessels are somewhat thickened. Diffuse pulsation is palpable over precordia Apex beat is palpable in sixth interspace four and one-half inches from mid-sternal line. A distinct systolic shock is felt at apex with a fine systolic thrill when patient sits up. Slight diastolic shock best felt over pulmonary area. Vertical dullness at second intercostal space: transverse four and three-fourth inches to left and one and one-fourth inches to right of mid-sternal line, at level of fourth rib. At apex, first sound is sharp, second sound accentuated, with loud, blowing systolic murmur transmitted to axilla. At base both sounds are accentuated; pulmonary very sharp. Over both pulmonary and aortic areas is

heard a rough, systolic murmur, most distinctly over pulmonary. Over third left costal cartilage there is a soft, blowing systolic murmur transmitted up and down the sternum. No ascites.

It was decided to induce labor as soon as the child was fairly viable. A bougie was inserted at 3 P.M. on March 27. Next morning the pains were strong and regular; at 4 P.M. the os was fully dilated. The membranes were then ruptured. A few whiffs of chloroform were given, forceps applied to the head at the brim and a living child easily extracted. The placenta separated in six minutes and was easily pressed off. A hypodermic of strychnin, gr. $\frac{1}{30}$, was then given. The puerperium was uneventful and both mother and child were discharged in fair condition on the fourteenth day. No special treatment was given for the heart condition, other than rest in bed, careful dieting and attention to the functions of bowels, kidneys and skin.

Contrary to advice, she insisted upon going home on the fourteenth day (April 10th). She was admitted to the M. G. Hospital on April 23, and died there on the twenty-sixth of acute dilatation of the heart.

CASE NO. 1807.—*Acute dilatation and hypertrophy of heart with endocarditis involving both aortic and mitral orifices. Mitral probably secondary.* Poor compensation.

K. D., æt. twenty-five, III-gravida. Admitted, September 11, 1907; confined, September 12; discharged, September 30.

Patient was married July 4, 1902, and has had two living children, the elder is now three and one-half and the younger one year old. Both times pregnancy, labor and puerperium were normal.

Clinical Note by Dr. Hamilton.—Patient very anemic, no dyspnea, lies comfortably in bed.

Pulse fairly full. Slightly collapsing. Very faint capillary pulse in fingers. Right radial fuller than left; carotid pulsation seen in neck. There is no definite venous overfilling, but along line of internal jugular veins is seen a diffuse wave; external jugulars negative.

Heart.—Inspection difficult on acconnt of size of breasts. Diffuse impulse is felt over precordia. Slight pulsation of sternum. Over aortic area, there is a suggestion of a systolic thrill. No palpable accentuated sound. Marked sensitiveness over sternum.

Deep percussion is forbidden by pain, but the following is on record: deep dullness six and three-fourths inches transversely from a point one and one-fourth inches to right of M. S. L.

At apex, first sound weak, accompanied by a systolic murmur; second sound somewhat sharp.

Pulmonary Area.—P^1 weak, accompanied by a very similar systolic murmur; P^2 accentuated.

Aortic Area.—A^2 sharp, very comparable to P^2. Systolic murmur.

Tricuspid Area.—Systolic murmur, maximum intensity at second left space. The same murmur heard well at first right space and at first right I. C. C., but not quite so loud here. The murmur is heard in vessels of both sides of neck; second sound is also audible here. In left axilla systolic murmur is faintly audible. On auscultating from left axilla over apex to pulmonary area this murmur is heard with increasing loudness. No diastolic murmur is heard, the second sound being clear-cut and not followed by diastolic murmur.

Diagnosis.—Acute dilatation and hypertrophy of heart. Endocarditis involving both aortic and mitral orifices—the mitral involvement being likely secondary.

Labor began at 4 A.M. and was very rapid. Pains came on suddenly and forcibly; the membranes soon ruptured (5.15 A.M.), the head descended rapidly and emerged transversely, occiput to right.; one coil of cord about neck (5.25 A.M.). Placenta detached in ten minutes and came away spontaneously. On the second day postpartum, patient complained of great difficulty in breathing. The distress and smothering were relieved by hypodermic of morphia, gr. ¼, and sleep was secured. Morphia was continued daily for a week. When discharged (September 30), she was found to have improved markedly since admission: the edema and dyspnea have disappeared, the heart is somewhat smaller in size and the sounds are clearer.

On December 26, she was admitted to the M. G. Hospital with chronic interstitial nephritis and was discharged improved in two weeks.

CASE NO. 1719.—*Myocarditis, acute.*

E. P., æt. twenty-four, V-gravida. Admitted, July 21, 1907; discharged, August 23, undelivered.

Patient is a dressmaker; entered hospital on account of general weakness and pain in back and abdomen. She has had four living children and one miscarriage; the eldest child is now seven; the youngest nearly two years of age. There is nothing special in the history of previous pregnancies. There is no history of rheumatism, chorea or alcoholism and she seems to have been a very healthy woman. When she came to Montreal, about two months ago, she was in good health, but a month later she fell downstairs and hurt her back. For a few days she suffered from distressing frontal headache and from frequent micturition (especially at night.) She continued working for a couple of days after the fall, but fainted twice and had to stop on account of great general weakness and breathlessness on the least exertion. When lying in bed there is no dyspnea and no cyanosis. Lungs clear on percussion and auscultation.

Heart.—Rather forcible impulse, especially in nipple-line. Vessels of neck pulsating. Cardiac impulse not forcible on palpation, no thrill; point of maximum intensity in fifth left interspace 11 cm. from mid-sternal line. Cardiac dullness, vertical

at third rib; transverse 2 cm. to right and 11 cm. to left of mid-sternal line at level of fourth rib.

Apex.—Heart-sounds irregular, due to occasional reduplication of second sound. A soft systolic murmur of varying intensity, transmitted to axilla. Second sound audible at apex, rather short.

Tricuspid.—Systolic murmur louder, becoming still louder on being followed up sternum. Maximum intensity in third left interspace.

Base.—P^2 accentuated; A^2 sounds valvular in quality. The irregularity due to reduplication of second sound is best heard at base and is heard every third to fourth beat.

Pulse 72, small in volume and tension, irregular in force and rhythm.

Patient was kept quiet in bed, a hypodermic of morphia, gr. $\frac{1}{8}$, given 4 q. h. for a few days and an ice bag put over the precordia. When the symptoms were relieved. Pil Blaud was given and improvement was rapid. She sat up in a chair for two hours on August 11, and was discharged in good condition August 23, promising to return at the time of confinement. However, two months later, she had to go home to West Indies, but was feeling well and quite able to undertake the journey.

CASE NO. 2095.—*Myocarditis chronic.*

S. McH., æt. forty, XIV-gravida. Admitted, March 13, 1908; discharged, March 16; readmitted, March 22; confined and died, March 24.

No history could be got of trouble in previous pregnancies, except that there was an adherent placenta after the last labor. Patient showed some signs of mental disturbance and was evidently somewhat under the influence of liquor. Subsequently she gave a history of chronic alcoholism and of occasional fainting fits during this pregnancy. As the pains had entirely disappeared and there were no signs of labor, at her own urgent request she was allowed to go home on March 16. She returned on the 22d with pains coming at long intervals. Temperature normal; pulse, 120, irregular in rhythm, no murmurs, no enlargement of heart on percussion. Urine showed a faint trace of albumin, no casts. Fetal heart-beat, 160 to 180. Complained of nothing besides occasional labor pains. Definite pains set in March 24, at 7 A. M., but soon disappeared; they began again between 12 and 1 P.M., the membranes ruptured spontaneously at 1.15. The maternal pulse was then 118, fetal heart, 162. The second stage was short, four or five pains in all, and a living child was born; no anesthesia was used. After the birth of the child, the mother's condition seemed good and the pulse fell to 96. But the uterus did not contract well and considerable blood came away. About fifteen minutes after birth of child, the patient blanched suddenly and the pulse became weak. A hypodermic of strychnin, gr. $\frac{1}{30}$, was given, but as there was no response, a hypodermic of camphor oil,

mv. (camphor, gr. i) was administered and she rallied a little. Next, a submammary infusion of 600 c.c. normal saline solution was given and she seemed to get much better. As the placenta had not come away in three-fourths of an hour and there was still a little trickle of blood, it was decided to remove the placenta manually. It was found to be closely adherent along the right side of the uterus and in the right cornu. After the removal of the placenta another hypodermic of strychnia, gr. $\frac{1}{60}$, was given, and as her condition did not improve another hypodermic of camphor oil, mv., was administered. The condition, however, grew rapidly worse. Artificial respiration was tried, but in spite of all efforts she sank and died at 3.05 P.M.

At the autopsy, the following anatomical diagnosis was made: Chronic myocarditis; chronic nephritis; chronic pleuritis; chronic endometritis; pulmonary edema. The child (full-term) weighed at birth 3055 grams, but lost weight steadily and died on the eighth day, weight 2415 grams. It was nursed for a time by a patient in the ward whose child was thriving well, and then, when it became weaker, breast milk was pumped out and fed to it; whey mixture and modified milk were tried, but it seemed to be unable to digest anything.

605 Dorchester Street, W.