Medical
Surgical and
Therapeutic

COMPLICATIONS OF PREGNANCY

Medical, Surgical, and Gynecological Complications of Pregnancy

by THE STAFF OF THE MOUNT SINAI HOSPITAL

NEW YORK CITY

EDITED BY

Alan F. Guttmacher, M.D.

Obstetrician and Gynecologist-in-Chief, The Mount Sinai Hospital, New York

AND

Joseph J. Rovinsky, M.D.

Assistant Attending Obstetrician and Gynecologist, The Mount Sinai Hospital, New York

CONTRIBUTORS

FROM THE

STAFF OF THE MOUNT SINAI HOSPITAL

ALBERT ALTCHEK, M.D., *Assistant Attending Obstetrician and Gynecologist*
STUART S. ASCH, M.D., *Assistant Attending Psychiatrist*
ARTHUR H. AUFSES, JR., M.D., *Assistant Attending Surgeon*
MORTIMER E. BADER, M.D., *Assistant Attending Physician*
RICHARD A. BADER, M.D., *Assistant Attending Physician*
IVAN D. BARONOFSKY, M.D., *Director, Department of Surgery, and Surgeon-in-Chief to The Mount Sinai Hospital*
MURRAY H. BASS, M.D., *Consulting Pediatrician to The Mount Sinai Hospital*
LESTER BLUM, M.D., *Assistant Attending Surgeon for Peripheral Vascular Diseases*
JOHN J. BOOKMAN, M.D., *Assistant Attending Physician for Metabolic Diseases*
MORTON S. BRYER, M.D., *Assistant Attending Physician*
MARTIN CLYMAN, JR., M.D., *Assistant Attending Obstetrician and Gynecologist*
SIMON DACK, M.D., *Associate Attending Physician for Cardiology and Chief of the Prenatal Cardiac Clinic*
ARTHUR M. DAVIDS, M.D., *Attending Obstetrician and Gynecologist*
HENRY DOLGER, M.D., *Associate Attending Physician for Metabolic Diseases and Chief of the Diabetes Clinic*
HENRY L. DORFMANN, M.D., *Senior Clinical Assistant Physician*
STANLEY EDELMAN, M.D., *Assistant Attending Surgeon*
LEON EISENBUD, D.D.S., *Associate Attending Pathologist for Oral Pathology*
ARNOLD N. FENTON, M.D., *Assistant Attending Obstetrician and Gynecologist (resigned)*
ALFRED E. FISCHER, M.D., *Attending Pediatrician and Chief of the Pediatric Diabetes Clinic*
EUGENE W. FRIEDMAN, M.D., *Associate Attending Surgeon for Head and Neck Surgery*
J. LESTER GABRILOVE, M.D., *Assistant Attending Physician*
JOSEPH A. GAINES, M.D., *Attending Obstetrician and Gynecologist*
STANLEY I. GLICKMAN, M.D., *Associate Attending Urologist*
ARNOLD GOLDENBERG, M.D., *Research Fellow in Medicine*
JOSEPH L. GOLDMAN, M.D., *Director, Department of Otolaryngology, and Otolaryngologist-in-Chief to The Mount Sinai Hospital*
EZRA M. GREENSPAN, M.D., *Assistant Attending Physician*
J. CONRAD GREENWALD, M.D., *Senior Clinical Assistant Obstetrician and Gynecologist*
ALAN F. GUTTMACHER, M.D., *Director, Department of Obstetrics and Gynecology, and Obstetrician and Gynecologist-in-Chief to The Mount Sinai Hospital*
BERNARD E. HERMAN, M.D., *Assistant Attending Surgeon*
WALTER H. JACOBS, M.D., *Resident in Gastroenterology*
HENRY D. JANOWITZ, M.D., *Associate Attending Physician for Gastroenterology and Chief of the Gastrointestinal Clinic*
ROBERT H. JOELSON, M.D., *Clinical Assistant Physician for Metabolic Diseases*
IRWIN KANTOR, M.D., *Assistant Attending Dermatologist*
EMANUEL KLEMPNER, M.D., *Associate Attending Obstetrician and Gynecologist*
ISADORE KREEL, M.D., *Fellow in Surgery*

HOWARD P. KRIEGER, M.D., *Associate Attending Neurologist*
LOUIS S. LAPID, M.D., *Associate Attending Obstetrician and Gynecologist*
JOSEPH LAVAL, M.D., *Director, Department of Ophthalmic Surgery, and Ophthalmic Surgeon-in-Chief to The Mount Sinai Hospital*
WILLIAM LEIFER, M.D., *Associate Attending Dermatologist*
GERSON J. LESNICK, M.D., *Associate Attending Surgeon*
MARVIN F. LEVITT, M.D., *Assistant Attending Physician and Chief of the Renal Clinic*
ROBERT K. LIPPMANN, M.D., *Director, Department of Orthopedic Surgery, and Orthopedic Surgeon-in-Chief to The Mount Sinai Hospital*
ARTHUR W. LUDWIG, M.D., *Assistant Attending Physician*
RALPH E. MOLOSHOK, M.D., *Attending Pediatrician and Chief of the Pediatric Growth and Development Clinic*
ROBERT A. NABATOFF, M.D., *Senior Clinical Assistant Peripheral Vascular Surgeon*
HERBERT E. NIEBURGS, M.D., *Research Associate in Pathology and Director of the Cytopathology Laboratory*
GORDON D. OPPENHEIMER, M.D., *Director, Department of Urology, and Urologist-in-Chief to The Mount Sinai Hospital*
KERMIT E. OSSERMAN, M.D., *Assistant Attending Physician for Special Service and Chief of the Myasthenia Gravis Clinic*
SAMUEL M. PECK, M.D., *Director, Department of Dermatology, and Dermatologist-in-Chief to The Mount Sinai Hospital*
GISELLA PERL, M.D., *Senior Clinical Assistant Obstetrician and Gynecologist and Chief of the Vaginitis Clinic*
CHARLES M. PLOTZ, M.D., *Assistant Attending Physician for Special Service and Chief of the Arthritis Clinic*
COLEMAN B. RABIN, M.D., *Attending Physician for Thoracic Diseases, Associate Attending Radiologist, and Chief of the Chest Clinic*
ALEXANDER RICHMAN, M.D., *Associate Attending Physician for Gastroenterology and Chief of the Liver Clinic*
RICHARD E. ROSENFIELD, M.D., *Associate Attending Hematologist and Director of the Blood Bank*
HARRY ROSENWASSER, M.D., *Attending Otolaryngologist for Otologic Surgery*
JOSEPH J. ROVINSKY, M.D., *Assistant Attending Obstetrician and Gynecologist*
MARTIN SANDERS, M.D., *Clinical Assistant Hematologist*
IRVING J. SELIKOFF, M.D., *Associate Attending Physician for Thoracic Diseases and Chief of the Prenatal Chest Clinic*
SHEPPARD SIEGAL, M.D., *Associate Attending Physician for Allergy and Chief of the Allergy Clinic*
ROBERT S. SIFFERT, M.D., *Associate Attending Orthopedic Surgeon*
LOUIS E. SILTZBACH, M.D., *Associate Attending Physician and Chief of the Sarcoidosis Clinic*
SOLOMON SILVER, M.D., *Attending Physician and Chief of the Thyroid Clinic*
ARTHUR R. SOHVAL, M.D., *Associate Attending Physician for Special Service*
ALVIN S. TEIRSTEIN, M.D., *Clinical Assistant Physician and Dr. Daniel Poll Fellow in Medicine*
FREDERICK H. THEODORE, M.D., *Associate Attending Ophthalmic Surgeon*
ROBERT TURELL, M.D., *Associate Attending Surgeon for Rectal Diseases*
ROBERT I. WALTER, M.D., *Associate Attending Obstetrician and Gynecologist*
BERNARD S. WOLF, M.D., *Director, Department of Radiology, and Radiologist-in-Chief to The Mount Sinai Hospital*

PREFACE

The interplay between obstetrics and the other fields of medicine is more extensive and intimate than found in any other specialty. This stems from the fact that pregnancy represents a unique condition in the health cycle, unique in the first place because pregnancy creates a profound, temporary alteration of physiology which may rapidly deteriorate into a state of pathology; unique in the second place, because pregnancy represents a physiologic state which by the will of man can be prevented, or once begun, acutely terminated. In the third place, it is the only chapter of medicine in which the interests of two patients are so inextricably intertwined, interests occasionally antagonistic.

Believing in the existence of an exceptional interplay of medicine and surgery with obstetrics, the Mt. Sinai Hospital departed from the usual pattern of organization when it opened its first department of obstetrics in November 1952. It created 10 specialty clinics within the framework of the department of obstetrics: cardiac, pulmonary, hypertensive-renal, diabetic, nematologic, neurologic, psychosomatic, obstetric-gynecologic endocrine, vaginitis, and varicose vein. Each of the 10 special clinics meets in the prenatal clinic area at a different time than the ordinary antepartum clinic. Each clinic is under the direction of one or more senior staff members from the department representing the particular medical discipline involved. Almost without exception the same senior staff physicians who initiated the special clinics continue to direct them 7 years later. Members of the obstetric-gynecologic resident staff attend the special clinics for the dual purpose of instruction and furnishing routine prenatal care. The less frequent complications of pregnancy which do not justify a separate obstetric clinic are ordinarily seen by a special consultant and once or twice a year he discusses the specific problems involved at an obstetric staff conference.

This type of organization has stimulated members from the other departments of the Hospital to develop particular interest in the pregnant women. It seemed to us that the invaluable knowledge accumulated about pregnancy by such a group of nonobstetric experts should be brought together in one book on the medical, surgical, and gynecologic problems of pregnancy. Each contributor is a member of the active staff of the Mt. Sinai Hospital. The authors hope that this book will have value not only for the obstetrician, whether specialist or general practitioner, and for the internist and surgeon, but also for residents, interns, and medical students. In an attempt to keep the material current we have largely excluded references published before 1950. Therapeutic measures have been stressed, not only specific drug therapy but the possible application of abortion and sterilization.

This volume is in no instance intended as a substitute for the several excellent, standard textbooks on obstetrics; it is intended, however, to be a valuable supplement to them. Readers may question the repetition of some of the material, such as the use and dosage of diuretics or antibiotics. We thought this was preferable to a distant page reference which is so apt to disturb continuity of thought.

This is a Mt. Sinai Staff Book and pays tribute to the high caliber of our medical and surgical colleagues. We thank them for their intense interest and the months required to prepare their contributions for this unique type of volume. The task of the editors has been relatively simple, due in great part to the cooperation of the publishers, The Williams & Wilkins Company.

ALAN F. GUTTMACHER, M.D.
JOSEPH J. ROVINSKY, M.D.

The Mount Sinai Hospital
New York City

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SECTION ONE

Cardiovascular-Renal System

1

HEART DISEASE

SIMON DACK, M.D., MORTIMER E. BADER, M.D., AND RICHARD A. BADER, M.D.

Cardiac disease in pregnant women has always presented a serious problem. With improvement in general medical care there has been a reduction in both the severity and number of other complications of pregnancy, so that at the present time cardiac disease, with its prevalence and gravity unchanged, has become statistically even more prominent. Indeed, in many obstetric institutions it represents the leading cause of maternal mortality. The fact that normal pregnancy produces numerous circulatory changes and symptoms which may simulate heart disease, *e.g.*, murmurs, dyspnea, edema, and weight gain, creates a further problem in differential diagnosis.

With increasing knowledge and awareness of the circulatory changes in normal pregnant women, better management of pregnant cardiacs has become possible. This has been manifested by a marked reduction in the number of therapeutic abortions for heart disease, and by a significant reduction in maternal mortality and morbidity from this cause. This is true in spite of the fact that a greater number of patients with heart disease are now carried to term rather than aborted.

Physiology of Circulation in Normal Pregnancy

The physiology of the maternal circulation in pregnant women has been studied intensively by others,[1-5] as well as in our own cardiopulmonary laboratory,[6] to determine the serial changes in blood flow and pressure in the cardiovascular system during pregnancy. An understanding of these circulatory changes underlies successful diagnosis and management of cardiac disease in pregnancy.

Heart Rate

In well controlled pregnant patients, serial observations reveal that the heart rate increases gradually, reaching a peak 1 to 2 months before delivery. It then returns toward the prepregnant control level at term. The maximal increase occurs at the seventh month and averages 10 beats per minute. Although a greater increase in rate occurs during physical exercise, even at rest there is a continually elevated rate throughout most of gestation.

Cardiac Output

The cardiac output is increased in pregnancy to levels approximately 30 to 50 per

cent above the nonpregnant control level. The peak increase occurs during the 25th to 32nd weeks of pregnancy and then the values gradually return toward normal prior to term. The mechanism for this increase in cardiac output may involve several factors, including increase in maternal oxygen consumption, hypervolemia, and an arteriovenous (A-V) shunt mechanism in the placenta.

Fetal or maternal oxygen consumption. The increased cardiac output is not closely correlated with an increase in the fetal or maternal oxygen consumption. The maternal oxygen consumption increases gradually so that just before term it is about 10 to 20 per cent over nonpregnant control levels, whereas the cardiac output reaches its peak about the seventh month, returning toward normal at term.

Hypervolemia of pregnancy. The second factor, hypervolemia of pregnancy, must be considered since there is an increase in plasma volume of about 50 to 65 per cent.[7] This increase begins early, reaches a peak about the 7th month, and then falls slightly, so that the plasma volume is still significantly elevated at the time of delivery. Therefore, if hypervolemia alone were to account for the serial changes in cardiac output, another factor, such as redistribution with pooling of blood in the lower extremities, would have to be invoked to explain the reduction in cardiac output in the last trimester.

Function of placenta as an A-V shunt. The third and most probable explanation of the curve of cardiac output is the function of the placenta as an A-V shunt. The presence of a bruit over the placenta, widening of the pulse pressure, high cardiac output, and decreased A-V oxygen difference all point to the dynamics of an A-V shunt. Such a shunt-like mechanism of the placenta would account for these circulatory changes, especially when one considers that the senescence of the placenta which occurs in the last trimester of pregnancy could be responsible for closing of these A-V anastomoses and return of cardiac output toward normal. Since there are anatomical and physiologic data to support the A-V shunt theory, it has become the most acceptable explanation for the changes in cardiac output.[2, 5, 8]

The cardiac output changes in pregnancy are of clinical importance: (1) The increase in output exists continuously for 24 hours a day (even at rest) and represents an increased burden which in the presence of cardiac disease could embarrass the circulation. Indeed, calculations in our laboratory[6] of left ventricular work during pregnancy revealed an increase at the time of the peak increase in cardiac work, with a parallel reduction prior to term. (2) The reduction in cardiac output and heart work in the last trimester of pregnancy allows for more cardiac reserve to be present during parturition. (3) The curve of cardiac output also explains why many patients have more difficulty in the midtrimester of pregnancy than during the last trimester, and why a patient who goes through the midtrimester without heart failure will usually have little difficulty in the third trimester.

Arterial Blood Pressure in the Systemic and Lesser Circulations

The systolic blood pressure in normal pregnant women varies little throughout gestation, but a small reduction in diastolic pressure has been observed and attributed to decrease in total systemic peripheral resistance.[2, 9] Hence, there is an increase in the pulse pressure which reaches a maximum during the seventh and eighth months, at the time when the maximal pulse rate and cardiac output levels prevail. The pulse pressure then returns toward normal. This is consistent with a placental shunt mechanism as described previously.

Although in our own studies[6] we found no significant changes in systolic, diastolic, and mean blood pressures taken on groups of patients during different periods of pregnancy, we did not make serial studies on the

same patients through gestation. In our laboratory the total peripheral resistance was found to be markedly reduced, reaching a minimum in the 25th and 27th weeks and gradually returning toward normal levels at term.

Measurements of blood pressure in the lesser circulation have also been made.[4, 6] The pulmonary artery and right ventricular systolic pressures show no significant changes. The right ventricular end diastolic pressure was increased in 25 per cent of the patients during the 25th to 35th weeks of pregnancy, and in one-third of these patients it was further increased during exercise. The pressure changes in the lesser circulation of these normal pregnant women are consistent with a congested circulatory state. Since this exists in the noncardiac pregnant woman, it is understandable why the pregnant cardiac patient is prone to develop heart failure, particularly at the end of the midtrimester. The mechanism for the increase in pressure in the right side of the heart in a few patients is probably related primarily to the hypervolemia of pregnancy, which is maximal at the time these pressure changes are noted. The fact that exercise causes a further increase in pressure explains why rest, with its decrease in venous return and cardiac work, represents an advantage to the cardiac patient in all periods of pregnancy, particularly during the 27th to 29th weeks.

Venous Pressure

The venous pressure is elevated in the lower extremities during pregnancy, reaching a maximum at term. This maximal elevation in the lower extremities may be 10 to 15 cm. of water greater than the level in the upper extremities.[10] The increased venous pressure is most probably due to increase in uterine vein flow into the inferior vena cava, and pressure of the gravid uterus on the pelvic veins. The significance of this finding is that in pregnant women it is not uncommon to encounter edema of the lower extremities, which may represent not cardiac decompensation but rather a physiologic finding consequent to the above factors. Although the mechanical factor of increased venous pressure is dominant in edema formation, changes in body water and hypervolemia contribute significantly (see below).

Circulation Time

As one would expect, there is a rapid circulation time during pregnancy which parallels the increased cardiac output and increase in pulse rate.[1] The arm-to-tongue time generally averages 13 seconds or less, compared to the usual normal figures of 15 to 16 seconds. The increased blood velocity reaches a maximum during the 34th week of pregnancy, gradually returning toward a more normal value at term. These changes parallel those in cardiac output.

Blood Volume and Body Water Changes

There is an increased plasma volume in pregnant women. Plasma volume levels are variably reported[2-5, 7] as being 50 to 65 per cent greater during the 25th to 35th weeks of pregnancy than are normal nonpregnant levels, decreasing to approximately 50 per cent at term. There is also an increase in the red blood cell volume.[11] However, the increase in plasma volume is relatively greater than that of the red cell volume, producing a relative reduction in the red blood cell count and hematocrit.

The total blood volume is increased, reaching a peak during the 30th to 36th week, with a slight decrease from peak values at term. The total body water increases gradually up to term, the increase being approximately 20 per cent above normal.[12, 13] The effect of these changes on normal circulatory dynamics has already been discussed. Such hypervolemia obviously presents a major threat to the pregnant woman with impaired cardiac reserve.

Electrocardiogram

The electrocardiogram in a normal pregnant woman may reveal nonpathologic

changes.[14] The electrical axis is deviated to the left by approximately 15 degrees during gestation and returns to normal just before or after delivery. In some patients, the development of a Q wave and inverted T wave in lead 3 may be noted as well as a small Q in aVF. In addition, in our experience, it is not uncommon to find an inverted T wave in leads V_1, V_2, and even occasionally in V_3, and lowering of T in V_4. All these changes are probably positional, because of elevation of the diaphragm and a more transverse position and rotation of the heart.

Chest X-ray

During pregnancy the roentgenogram of the chest reveals increase in the transverse diameter of the heart, with straightening of the left upper cardiac border.[14] In about 15 per cent of the patients the pulmonary artery segment may become prominent because of increased pulmonary blood flow. The changes in cardiac size and shape are due principally to elevation of the diaphragm and should not be erroneously regarded as evidence of cardiomegaly. Another factor responsible for the apparent increase in heart size is increase in the volume of the heart as a result of hypervolemia.

Respiratory Function

No discussion of the circulatory changes in pregnancy can be complete without consideration of the problem of respiratory function in pregnancy. Dyspnea is a common symptom in the normal pregnant woman, its incidence being approximately 60 to 65 per cent.[15, 16] It may occur at rest and is aggravated by exercise or the recumbent position. This dyspnea occurs in the absence of any demonstrable cardiac or pulmonary lesion or insufficiency. Indeed, conventional pulmonary function tests reveal no changes other than moderate hyperventilation at rest and after exercise as pregnancy progresses.[16] In the latter part of pregnancy the oxygen consumption is increased to approximately 10 to 20 per cent above normal. The vital capacity may be normal, slightly reduced, or even slightly increased.

Elucidation of the mechanism of dyspnea in pregnancy has received impetus from the employment of newer techniques in the study of respiratory physiology. Studies in our laboratory[17] have demonstrated that (1) in normal pregnant women there is an increase in the amount of oxygen consumed for given levels of overbreathing, as compared to the nonpregnant control; and (2) this increment in oxygen cost of breathing in pregnancy increases gradually toward term. Similar findings of increased oxygen cost of breathing has been observed in patients with cardiac failure, pulmonary granulomatosis, and emphysema. This finding in normal pregnant women suggests that one of the possible mechanisms for the dyspnea may be related to increased oxygen cost of breathing. Further studies in our laboratory[18] of the mechanical work of breathing have demonstrated that there is no change in transpulmonic compliance and pulmonary airway resistance, suggesting that extrapulmonic factors, *e.g.*, increased abdominal burden with elevation of the diaphragm and engorgement of the breasts, may be the cause of the increased work.

Whatever the mechanism, the increased dyspnea in normal pregnant women creates a problem in evaluating cardiac symptomatology, particularly in patients with minor or borderline lesions who have no objective evidence of failure but may have subjective sensations of dyspnea. Psychologic factors are of considerable importance and may frequently result in hyperventilation and complaints of breathing difficulty. This phenomenon does not differ from the emotional hyperventilation found in the nonpregnant state.

Summary of Normal Circulatory and Ventilatory Changes in Pregnancy

The normal hemodynamic changes in pregnancy can be summarized as follows:

1. Increased cardiac output, beginning

early in pregnancy, reaching a peak of about 40 per cent above normal at about the seventh month, and falling toward normal at term.

2. Increased blood velocity and shortened circulation time to values at the lower limit of normal (average, 12 seconds).

3. Increased plasma and blood volume to 50 to 60 per cent above normal, particularly during the second half of pregnancy.

4. Increased heart rate in the second and third trimester, averaging 10 beats per minute.

5. Small increase in arterial pulse pressure due to slightly lowered diastolic pressure; decrease in systemic peripheral resistance simultaneous with the peak changes in cardiac output, with return toward normal at term.

6. A slight rise in pulmonary artery pressure but a significant increase in right ventricular end diastolic pressure at rest in 25 per cent of patients at the end of the second trimester, accentuated by exercise.

7. Disproportionate increase in venous pressure in the lower extremities, as compared with the upper, predisposing to dependent edema.

8. Slight increase in oxygen consumption, reaching 10 to 20 per cent above normal.

9. Increase in oxygen cost of breathing without significant change in transpulmonic compliance or airway resistance.

Cardiopulmonary Symptoms and Signs in Normal Pregnancy

Physiologic adjustments in the maternal circulation may produce symptoms and findings in the normal pregnant woman which simulate organic heart disease.[19] These effects are such that when an otherwise asymptomatic or mild heart lesion is present a false impression of congestive heart failure may arise. Furthermore, cardiopulmonary symptoms and signs will often make evaluation of the severity of preexisting organic heart disease difficult.

The following cardiopulmonary symptoms and signs may occur in the absence of organic heart disease in the normal pregnant woman:

1. Shortness of breath on effort, associated with increased ventilation rate and elevated diaphragm. There may be dyspnea on recumbency simulating the orthopnea of heart failure. Some patients may have sighing respirations.

2. Tachycardia and overactive heart action associated with palpitations at rest or on mild exertion.

3. Apparent cardiac enlargement.

4. Edema of the lower extremities.

5. Functional heart murmurs, associated with the overactive heart, increased blood velocity, and anemia. Such murmurs are generally soft and blowing in character, rarely louder than grade 2 intensity and often best heard in the pulmonic area. A functional or hemic murmur which has been present before pregnancy usually becomes accentuated as pregnancy progresses and may simulate an organic murmur attributable to valvular disease. Occasionally a systolic or a to-and-fro murmur which arises from branches of the mammary artery or veins of the breast may be heard in the pulmonic area and upper left sternal margin.[20-23] The murmur results from increased blood flow through these vessels during the latter part of pregnancy. In one of our patients it simulated a patent ductus arteriosus. It can be differentiated by noting its disappearance during strong pressure of the stethoscope on the chest wall, obliterating the dilated vessel. The murmur generally disappears after pregnancy.

6. Physiologic changes in the heart sounds. P_2 is often accentuated because of increased pulmonary blood flow. Not infrequently it becomes ringing or amphoric in character and it may be difficult to exclude the presence of a pulmonic diastolic murmur. The third sound at the mitral area becomes accentuated as a result of increased ventricular filling and the fourth, or auricular, sound may become prominent as a result

of increased auricular filling and contraction. The latter two physiologic effects may lead to the erroneous diagnosis of diastolic or presystolic murmurs. In the presence of an overactive heart action the heart sounds may be transmitted to the neck vessels and may simulate transmitted aortic murmurs or thrills.

Aids to Differential Diagnosis of Physiologic and Abnormal Cardiac Signs

When difficulty arises in differentiating between functional symptoms or physiologic changes and those due to organic heart disease, several diagnostic procedures may have clinical value. Careful examination of the lungs for evidence of pulmonary congestion, examination of the neck veins for evidence of venous distention, and radiologic examination of the chest to determine the presence of cardiac enlargement and pulmonary congestion are indicated. In addition, measurement of the venous pressure and circulation time may help exclude congestive heart failure. Thus, edema of the legs in the presence of a normal venous pressure in the arms suggests local venous hypertension and stasis rather than congestive failure. A normal circulation time would suggest that dyspnea is due to functional factors rather than to pulmonary congestion. It should be remembered that a circulation time ordinarily considered to be the upper limit of normal (18 seconds) may actually represent significant slowing of the pulmonary blood flow, since normally the circulation time in pregnancy may be moderately shortened to 12 seconds.

The simple measurement of vital capacity may be of some help, but only if serial measurements are made.[16, 24] A progressive decrease of vital capacity in serial examinations would suggest pulmonary congestion due to failure.[24]

Liver enlargement is not encountered ordinarily in noncardiac pregnant patients. Palpation of this organ may be quite difficult because of the enlarged uterus and elevated diaphragm.

It is apparent from the above discussion that a careful diagnostic examination of the cardiovascular system is important in all pregnant women. The confirmation of the functional nature of any cardiopulmonary finding would permit reassurance of the patient that her heart is normal and thus prevent an anxiety state; and the discovery of an organic lesion would initiate proper supervision and management, preventing possible cardiac failure.

Effects of Anemia on Cardiovascular Dynamics

Nutritional anemia which has existed before the pregnancy or which has developed during its course will accentuate all of the physiologic cardiovascular effects of pregnancy and often produce a difficult problem in differential diagnosis.[15] In the nonpregnant woman a mild to moderate anemia (hemoglobin, 9 to 11 gm.) may produce little effect on the cardiovascular system. In pregnancy, however, the combined cardiodynamic effects of both pregnancy and anemia may produce dyspnea, fatigue, tachycardia, overactive heart action, and increased blood velocity at rest and particularly after effort. A preexisting innocuous murmur may become exaggerated. In such a patient it will be difficult to exclude the presence of organic heart disease.

A severe anemia (8 gm. or less) may produce all of the signs simulating congestive heart failure, especially if a functional heart murmur is present.[19] However, in such a patient the circulation time is normal or shortened. As discussed later in the section on treatment of anemia, p. 21, the cardiac functional capacity of the anemic pregnant patient may be restored to normal only if measures to correct the anemia are employed in conjunction with therapy directed at the congestive failure itself.

Fluid and Electrolyte Retention in Pregnancy

It is well known that pregnant women show a tendency to edema and weight gain

even in the absence of cardiac decompensation or toxemia. This is due to a gain in extracellular fluid which may be disproportionate to the gain in lean body tissue and which occurs irrespective of the increase in circulatory blood volume.[13] There is increased retention of water, sodium, and chlorides, probably as a result of increased tubular absorption of these electrolytes by the kidneys.

Recent studies[25, 26] on urinary excretion of aldosterone, the adrenocortical salt-retaining hormone, have demonstrated that there is increased secretion of this hormone in normal pregnancy. This begins sharply after the third month, reaching its peak just before term.[26] After delivery there is a rapid fall to prepregnancy levels. The rise in aldosterone secretion in normal pregnant women may approach the increased levels observed in toxemia of pregnancy and in congestive heart failure.

Such fluid and electrolyte retention in normal pregnancy may lead to "edema of pregnancy." As discussed previously in the section on venous pressure, p. 3, this is most striking in the lower extremities because of the increased venous pressure resulting from pressure of the enlarging uterus on the pelvic veins. However, patients with this type of edema almost never develop congestive heart failure unless there is underlying cardiac disease, particularly rheumatic valvular disease. It is of interest that, even in toxemia of pregnancy with marked edema and hypertension, congestive heart failure and cardiac death are uncommon except in the presence of antecedent rheumatic or hypertensive heart disease.

On the other hand, pregnant patients with heart disease are more prone to develop congestive heart failure because of the increased fluid and electrolyte retention normal for pregnancy. When toxemia is superimposed on congestive failure, the added fluid retention and hypertension often result in intractable failure and maternal mortality.[27]

Rheumatic Heart Disease in Pregnancy

CLINICAL FEATURES

Incidence

The reported incidence of cardiac disease in pregnancy varies with the criteria utilized in different clinics and with the index of suspicion of the referring physicians in the prenatal clinics. Various authors[5, 28-32] report an incidence ranging between 1.2 and 3.7 per cent of all pregnant women. During the first 5 years of the obstetric service at the Mt. Sinai Hospital there were 203 patients with heart disease (1 per cent) in 21,169 deliveries.[33]*

The most common form of heart disease encountered in pregnancy is rheumatic heart disease, which accounts for at least 90 per cent of all cases. The remainder are divided among the arteriosclerotic, hypertensive, and congenital types. Congenital heart disease comprises only 1 to 3 per cent of all cardiac disease in pregnancy, although an incidence as high as 6 per cent has been reported.[34] The incidence in our clinic has been approximately 2 per cent. Hypertensive disease is the causative factor in 1 to 2 per cent of the cases. The fact that childbearing is rare beyond the age of 40 naturally tends to minimize arteriosclerotic and luetic heart disease as major factors in the etiologic types of disease in pregnancy. Not only age but sex is a factor, since arteriosclerosis is less common in women. In our clinic during a 6-year period there have been only three patients with arteriosclerotic heart disease (two afflicted with familial hypercholesteremia, coronary insufficiency, and angina pectoris).

Relative importance of heart disease. The number of pregnant women who have cardiac disease and are carried to term has

* There were 91 patients with cardiac disease in 14,089 private deliveries (0.65 per cent) and 112 such patients in 7080 ward deliveries (1.6 per cent), the latter rate being higher because of both the lower socioeconomic status of the patients and the more rigorous evaluation afforded service cases.

increased because of several factors: (1) decrease in incidence of therapeutic abortions; (2) increase in survival rates of rheumatic fever patients because of modern medical management; (3) increase in the number of patients who survive subacute bacterial endocarditis because of antibiotic therapy; and (4) relative increase in the incidence of cardiac disease compared to the total group of medical complications, as a result of decreased incidence and better control of infections, anemia, and toxemia. As a result, heart disease is now one of the leading causes of maternal morbidity and mortality in many clinics.[27, 35, 36]

Maternal Mortality

Maternal mortality from heart disease, *i.e.*, the mortality during pregnancy, labor, and the first 6 postpartum weeks (rather than the long term mortality), has declined over the past half century as a result of improved medical management. For example, Hamilton and Thompson[37] recorded an overall maternal mortality rate of 3.9 per cent for a 25-year period: 15 per cent during 1921 to 1924, 5 per cent during 1925 to 1927, and only 2 to 3 per cent during 1927 to 1937. Other workers report mortality rates varying from 0 to 3 per cent (Vander Veer and Kuo,[28] 3.4 per cent; Mendelson,[38] 1 per cent; Gorenberg and Chesley,[29] 0.4 per cent in 500 consecutive patients; and MacLeod,[39] 0 per cent). Gordon[40] reports a decrease in the maternal mortality from heart disease from a rate of 30.6 per 100,000 pregnancies in 1941 to 15.5 per 100,000 in 1951. One must recall that over 60 per cent of Angus McDonald's pregnant cardiac patients who were managed before 1878 died during pregnancy of congestive failure and other complications of cardiac disease (cited by Burwell and Metcalfe[5], p. 6).

This increase in maternal survival is even greater than the actual figures would indicate, since many women who would have undergone therapeutic abortions are being carried to term, thus increasing the number of serious risk patients who are being carried through pregnancy. When the data of Gorenberg and others are considered, one is impressed with the striking ability of such patients to survive pregnancy and delivery.

The figures in our clinic indicate that the over-all rate for mortality from cardiac disease in our pregnant population has been 0 per cent. Not one registered patient died from heart disease. One patient who was not registered came in as an "urgent admission" and died in congestive failure. If one includes this unregistered case, we have had one cardiac death on our obstetric service in 6 years, an incidence of 0.5 per cent among pregnant patients with organic heart disease.

Factors Affecting Maternal Mortality

Adequate medical supervision. Probably the most important factor affecting maternal mortality is good diagnosis and careful medical supervision of the cardiac patient to prevent congestive heart failure. For example, Gorenberg and Chesley[29] found that in closely supervised clinic patients there was a mortality rate of 0.4 per cent; in private patients, 10 per cent; and in nonregistered patients, 20 per cent. The cause of the maternal mortality in these groups was almost always heart failure.

A recent survey[27] of 1000 consecutive maternal deaths revealed that 45 were caused by cardiac disease. Thirty of these mothers died in congestive failure which in most instances had been inadequately treated. Similarly, Fitzgerald *et al.*[41] showed that adequate antepartum care had been given to only three patients of their series of 24 maternal deaths in 11 years. In Bramwell's series,[30] 10 of 15 patients who died were unregistered cases admitted in cardiac failure. The sole fatality due to heart disease on our own service occurred in a patient with severe hypertensive and rheumatic heart disease who had never been seen by us previously and who died in heart failure in the 33rd week of gestation, 44 hours after emergency admission.

Adequate medical care implies not only