Idiopathic Myocardial Failure in the Last Trimester of Pregnancy and the Puerperium

By W. R. MEADOWS, M.D.

Instances of congestive heart failure due to unexplained myocardial disease are not infrequently discussed in the literature. The present study, an extension of a similar study reported by Hull in 1937, relates this type of case to the terminal aspects of pregnancy and the puerperium with an incidence of 1 in 1300 deliveries. Four case reports with necropsy findings, together with the clinical observations made on a total of 15 cases are presented. The frequency of recurrence of the syndrome with subsequent pregnancies and the predominance in the puerperium of this type of congestive failure over those of the usual etiologies suggests that the relationship to pregnancy is not fortuitous.

CONGESTIVE heart failure due to idiopathic myocardial disease appearing for the first time during the terminal aspects of pregnancy or the puerperium apparently was known to earlier writers, but it was not until 1937 that adequate clinical and pathologic descriptions appeared in the literature. In that year Hull described the clinical findings in 27 patients, and Gouley, McMillan, and Bellet presented 4 cases with autopsy findings. Since then a number of case reports have appeared, and reference to "postpartum heart disease" has been made by others, but as yet the syndrome is not generally recognized and is not described in most texts on cardiology.

This communication consists of a report of 4 cases with necropsy findings, the clinical observations made on a total of 15 that, with 1 exception, have been followed for a period of 5 to 9 years since their original hospitalization at Cook County Hospital, Chicago, and a brief comparison with the observations of others. The purpose of this report is to confirm and extend previous observations made on this syndrome and to stress the absence of previous heart disease or hypertension in most of these cases. These instances also add further basis for the belief of previous observers that the coincidental pregnancy is in some way related to the myocardiopathy and suggest that many of these cases may be misdiagnosed as rheumatic or hypertensive heart disease due to the lack of general recognition of the syndrome.

From the Department of Medicine, Cook County Hospital, Chicago, Ill.

CASE REPORTS

Case 1

G. J., a 30-year-old Negro with 10 previous births, entered the hospital on April 18, 1945. Three months after the birth of a female infant on December 10, 1944, she developed paroxysmal nocturnal dyspnea (initially in the absence of exertional dyspnea), occasional precordial pain, frequent emesis, and, later, upper abdominal pain. Physical examination revealed a blood pressure of 150/112, a heart rate of 108, a precordial systolic murmur, P2 louder than A2, but no peripheral signs of congestive failure. The ocular fundi were normal. One week following admission the blood pressure dropped to 116/80, and the heart rate to 88. After 9 days she was discharged much improved.

Five pregnancy records from 1930 to her last in 1949 revealed the finding of a precordial systolic murmur noted during 2 different pregnancies, 1 plus albuminuria in only 1 of 17 tests, and multiple blood pressure readings within normal limits except for occasional diastolic levels as high as 94 toward the end of the last 2 pregnancies.

During the next 2½ years her symptoms and signs persisted, and obvious signs of congestive failure appeared and became severe. Hemoptysis and right pleuritic chest pain were frequent. The diastolic blood pressure was frequently elevated on admission, but readings fell as low as 98/78 during her hospital stays. Gallop rhythm was noted on several admissions. In 1947 she was admitted with generalized anasarca, a pulsating liver, and mild icterus. Following paracentesis and 2 months of hospital bed rest she remained relatively asymptomatic for 2 years during which time the ascites gradually disappeared and pulsation of the liver was no longer detected. Blood pressures ranged between 120/80 and 172/110 during this period, and the systolic murmur and cardiac enlargement persisted. Six weeks after stopping digitalis in June 1949 she began a slowly deteriorating course during which
generalized anasarca, tricuspid insufficiency, and icterus again appeared. Death occurred on June 15, 1951. This patient’s hospital diagnoses usually were rheumatic heart disease, less often hypertensive heart disease.

Post Mortem. The heart weighed 525 Gm. All chambers were dilated, especially those of the right side. The myocardium was flabby. The right ventricular wall measured 4 mm.; and the left, 1.3 cm. In the subendocardial layers of all chambers were many yellow or fibrotic areas. A mural thrombus near the apex of the left ventricle overlay a larger area of subendocardial fibrosis. Valvular deformities were absent, and the coronary vessels were patent throughout. Microscopically, degeneration of muscle fibers was seen throughout the myocardium but particularly in the subendocardial and subepicardial zones. The fibers were swollen, pale staining, and vacuolated. Cross striations were indistinct or absent, and golden brown pigment was present at both poles of the nuclei. The interstitium was edematous but showed little cellular infiltration. Small focal areas of fibrosis were scattered throughout the myocardium. The endocardium was somewhat thickened, and in the deeper portion loose cellular granulation tissue extended into the underlying thrombus.

Section of the liver revealed distortion of the lobular architecture, the enlarged central fields frequently being connected by bridges. There was extensive recent and old central necrosis with hemorrhage. Fibrous septa with proliferated ductules frequently replaced the necrotic central and intermediate zones and sometimes reached the portal areas. This process was, in a few places, associated with formation of regenerative nodules, whereas in others the picture of subacute congestion predominated. Other findings were those of a biliary nephrosis, chronic pyelonephritis, and passive congestion of all organs.

Case 2

J. M., a 22-year-old Negro with 3 previous births, entered the hospital on March 14, 1949, complaining of severe left upper quadrant pain radiating into the left flank, which had started suddenly 8 hours earlier. Physical examination revealed a blood pressure of 120/100, a heart rate of 112, and a diffusely tender abdomen with rebound tenderness referred to the left flank. A tentative diagnosis of ruptured ectopic pregnancy was made, but 2 colpocenteses produced only straw-colored fluid. One specimen of urine obtained during the first 48 hours after admission was grossly bloody. On further inquiry a medical consultant found that 3½ months after the birth of a female infant on November 14, 1948, the patient had begun to vomit and soon after had a sensation of substernal fullness, paroxysmal nocturnal dyspnea, exertional dyspnea, and nocturia. He further noted Cheyne-Stokes respirations, a protodiastolic gallop rhythm, an apical systolic murmur, P2 louder than A2, but no peripheral signs of congestive failure.

The record of her last pregnancy revealed multiple blood pressure readings not exceeding 110/80, all urinalyses negative for albumin, and a negative cardiac examination. Delivery records for 1945 and 1947 were similarly negative except for a vague history of treatment for syphilis.

By the fifth hospital day abdominal symptoms had abated, blood pressure had fallen to 94/80, and several icterus was temporarily noted. Three blood transfusions had been given during the first 3 days in the hospital. The above symptoms continued to be troublesome, however, and signs and symptoms of advanced congestive failure appeared. Retrograde pyelography on March 24 was negative, and clear urine was seen coming from the left ureter, but she subsequently became febrile and developed pyuria. Several blood cultures were taken and subsequently reported negative. Fundoscopic examination on April 27 was negative. On June 9 the patient complained of tenderness of the left knee and calf muscles, and a positive Homans’ sign was elicited. One week later there was an episode of abdominal pain initially in the xiphoid area that later became diffuse. This incident was followed by substernal, left chest and left shoulder pain on June 20. She was discharged August 1.

Six months later she was seen in a debilitated state with generalized anasarca, and she described a recent sudden loss of consciousness. Blood pressure was 100/85 with alternans throughout the complete pulse range, and an electrocardiogram showed a 2:1 atrial flutter that persisted until her death (fig. 1). She reentered the hospital June 1, 1950, and died a day later. A loud precordial systolic murmur heard before her death continued a long standing controversy concerning a diagnosis of rheumatic heart disease on this patient.

Post Mortem. The heart weighed 350 Gm. All chambers were extremely dilated. The right atrium measured 1½ mm. in thickness, the right ventricle 4 mm., and the left ventricular wall from 6 mm. in the apical region to 14 mm. in the basal portion. Mural thrombi were found in the right atrial appendage and in both ventricular chambers adherent to the septal and anterior walls near the apex. In a large area around these adherent thrombi in the left ventricle the endocardium was thickened, light gray, and blended with the underlying myocardial fibrosis to include the inner one fourth of the wall; this endocardial thickening measured 5 cm. in diameter and extended to the lateral wall. There were no valvular deformities, and the coronary arteries were all patent and free of atherosclerosis. Microscopically, many of the muscle bundles were swollen, poorly staining, and contained in paraffin sections small empty vacuoles. Cross striations were
often indistinct and at times could not be made out. The interstitial tissue was edematous. Under the endocardium a few small fibrotic foci were seen, as well as several large foci of eosinophilic necrosis containing small round cells, histiocytes, and pigment-laden macrophages. In other areas the subendocardium was greatly thickened with granulation tissue (fig. 2).

There were multiple hemorrhagic infarcts in the right lung, some dark red and others brown. A large thrombus was found in the right pulmonary artery. Both fibrosing and miliary pulmonary tuberculosis was present with caseous tuberculosis of the tracheobronchial lymph nodes. The kidneys and spleen were also sites of tuberculosis and healed infarcts. The left renal artery was reduced to 2 mm. in diameter, and the left kidney was shrunken as the result of old infarcts. Passive congestion of all organs was noted.

Case 3

D. W., a 29-year-old Negro with 2 previous births, entered the hospital on December 2, 1949. Three months following the delivery of a male infant on September 2, 1949, she experienced an attack of paroxysmal nocturnal dyspnea with hemoptysis, and the third such attack precipitated hospital admission. A cough had been present since the seventh postpartum week. Physical examination revealed a blood pressure of 160/86, a "triple rhythm" with a rate of 115, no murmurs, and A2 equal to P2. Ocular fundi showed grade 1 generalized arteriolar spasm. As objective evidence of congestive failure was not noted, the patient was isolated temporarily because of a suspicion of pulmonary tuberculosis. She was kept at strict bed rest without any specific cardiac medication for 2 months, and symptoms, "triple rhythm," and tachycardia were never noted again. Blood pressure had fallen to 120/80 when discharged. Electrocardiographic abnormalities had disappeared by the time of discharge (fig. 3).

Her obstetrical record revealed blood pressures as high as 190/110 for 3 days prior to her last delivery, with readings of 120/60 on the eighth postpartum day and 136/90 on the ninth postpartum day. Readings as high as 170/100 had been obtained during her first delivery a year previously. No albuminuria or cardiac abnormalities were noted on either occasion.

This patient's blood pressure readings reached hypertensive levels again 1 year later but remained at normotensive levels following radical surgery for a pelvic malignancy in January 1953. She delivered a full-term infant on August 4, 1952, but never experienced further symptoms of cardiac disease. On April 30, 1956, she died from obstructive nephropathy with uremia due to metastatic carcinoma.

A postmortem examination was performed by the coroner. The heart weighed 375 Gm. On section, the myocardium was red-brown in color. The epicardial and endocardial surfaces were not remarkable. A diagnosis of eccentric hypertrophy of the myocardium was made. The remaining findings were those of her neoplastic disease.

Case 4

M. L., a 24-year-old Negro with 3 previous births, entered the hospital on July 28, 1950, with a history of sudden inability to lie flat that had begun about 7 weeks after the delivery of a female infant on April 8, 1950. It was difficult for the patient to describe the sensation that was relieved by sitting up, but at various times she mentioned restlessness, dyspnea, and a substernal oppression radiating through to the back also described as a "smothering pain." Night cough, hemoptysis, exertional dyspnea, and vomiting with occasional hematemesis soon followed. Physical examination revealed a blood pressure of 128/104, a gallop rhythm with a heart rate of 104, and P2 markedly accentuated. No murmurs were noted, and there were no peripheral signs of congestive failure. Eleven days later the blood pressure had fallen to 95/70, and the gallop had disappeared, but she continued to have occasional episodes of vomiting and substernal oppression during the remainder of her 2 months' hos-
pitalization. Recurrent lower abdominal pain and tenderness, which had been noted in the hospital, continued after discharge, but cardiac symptoms disappeared. In the year that followed blood pressures did not exceed 120/80, but gallop rhythm was occasionally heard, cardiac enlargement persisted, and electrocardiograms remained abnormal (fig. 4).

Available records of her 3 pregnancies revealed multiple blood pressure readings not exceeding 124/80, all urinalyses negative for albumin, and a negative cardiac examination on 4 different occasions. The last pregnancy had been eventful only for frequent headaches and abdominal pain.

In the last trimester of this patient’s subsequent pregnancy terminating on September 19, 1951, she again developed symptoms and signs of heart failure, which became intractable and resulted in her death on September 8, 1955. During this time she remained for the most part normotensive. It is of interest that in October 1953 an intern in another hospital wrote in response to an inquiry about her: “On each admission there is no agreement as to

Fig. 2. Microscopic sections of the myocardium of case 2. A. Progressive subendocardial fibrosis and zone of vacuolization of subjacent myocardial fibers. B. Increase of interstitial fibrous stroma isolating small islands of muscle fibers. C. and D. Subendocardial thickening due to fibrosis.
etiology—not even with regard to possible diastolic murmur."

Post Mortem (San Francisco Hospital). The heart weighed 650 Gm. The left ventricle averaged 9 mm. in thickness; the right ventricle, 5 mm. The myocardium was soft, and there was dilatation of all chambers. The endocardium of both ventricles was diffusely thickened. On section of the left ventricular wall scattered areas of fibrosis, yellow-white in color, were found measuring up to 1 cm. in diameter. The endocardium in the left ventricle under the most involved area of myocardial scarring had a large mural thrombus attached that measured 3 by 5 cm. Organizing mural thrombi were also found in the right atrium. The chordae tendineae were slightly thickened, but valvular deformities were absent. The coronary arteries were thin, elastic, and patent throughout their lengths. Microscopically, the myocardial fibers appeared relatively healthy with cross striation visible in most places. A number of fibers were fractured with loose ends apparent and there were a few areas where myocardial fibers were degenerated and showed nuclear changes. The endocardium of both ventricles was markedly thickened with fibrosis and elastic tissue that showed a great infiltration of lymphocytes, plasma cells, macrophages, and a few eosinophils, which involved the superficial layers of the myocardium. A number of myocyte nests were found in the subendocardial region and scattered areas of fibrosis were also seen between the myocardial fibers. Perivascular inflammatory-cell infiltration was noted around the smaller vessels. The epicardium was noted in a few places and was slightly thickened with an indefinite lining (fig. 5).

There was passive congestion of the liver, kidneys, and lungs, and the last 2 organs were the sites of microscopic infarctions. Hydrothorax and abdominal ascites were noted.

**Clinical Observations of 15 Cases**

Some estimate of the incidence of this syndrome can be made by comparing the total of 6 cases, all of

![Fig. 3 Electrocardiograms in case 3.](image)

![Fig. 4. Electrocardiograms in case 4.](image)
which were Negroes, with the total of 7,519 deliveries that occurred at the Cook County Hospital in 1949. Thirteen of the 15 patients in this series were Negroes. This correlated closely with the proportion of Negro patients on the obstetrical wards but was greater than the 52 per cent given for the total patient population of the hospital. The ages of these patients ranged from 22 to 44 years, and the number of pregnancies from 1 to 10. In 2 instances the last pregnancy had resulted in twin births.

A review of past pregnancy records revealed that a diagnosis of pre-eclamptic toxemia had been made during the terminal aspects of the last pregnancy in 2 instances and that hypertension, i.e., readings exceeding 150 systolic or 90 diastolic, had been a feature of the labor period in 1 other. In no case was hypertension observed to be more than a transient phenomenon. An inconstant systolic murmur had been heard in 3 patients, one of whom eventually was proved to have no valvular lesion at necropsy.
but in no instance was there definite evidence of preexisting organic heart disease.

The past history revealed syphilis in 6 cases and an episode of polyarthritis at age 17 in 1.

**Symptoms.** The onset of the initial symptoms of congestive heart failure occurred within the first 3½ postpartum months in 11 patients but was as late as 5 months postpartum in 2 and as early as the last trimester of pregnancy in 2. In the latter instances symptoms of congestive failure disappeared at the time of delivery only to reappear again 4 to 8 weeks later. The earliest and most consistent symptoms were cough becoming worse at night and recurrent bouts of paroxysmal nocturnal dyspnea, each symptom being noted by 11 patients. These symptoms usually preceded the onset of exertional dyspnea, orthopnea, and hemoptysis, each of which was noted by 7 patients.

Chest pain noted by 8 patients consisted of recurrent precordial distress in 3 and substernal oppression in 5, 3 of whom had radiation of pain through to the back. One patient described an associated sensation of numbness in both arms.

Gastrointestinal complaints consisted of nausea and vomiting in 7 and hematemesis in 3. Abdominal pain was reported by 6, and in 1 patient there was postmortem evidence that this had been due to splenic and renal infarction. In 2 instances periumbilical pain had been present since delivery and had preceded the first symptom of postpartum congestive failure by 1 to 2 months.

Cerebral embolism with hemiplegia occurred in 2 patients, and pulmonary embolism occurred in 1 other. These clinical embolic episodes occurred early in the course of the disease, the longest interval following the onset of symptoms being 3½ months. In 2 instances symptoms resulting from systemic embolization were the presenting complaint, and it was only learned later that symptoms of congestive failure had been present for a period of 2 to 5 weeks. Profuse sweating was noted in 2 instances.

In every instance except 1, patients sought hospital admission within 6 weeks of the onset of symptoms.

**Physical Findings.** At the time of admission to the hospital a labile and predominantly diastolic hypertension was present in every patient except 1. Nine of the 13 patients with diastolic readings between 100 and 116 had systolic readings that did not exceed 150. The remaining 4 had systolic readings between 170 and 190. When this hypertension had appeared in the puerperium was not determined by this study, but in 1 instance it was known to have been present for at least 8 days before the first sign or symptom of the exacerbation of congestive failure that followed a subsequent pregnancy. Pulsus alternans was noted in 3 and later in a total of 6. In 2 instances this sign extended throughout the entire pulse range.

Auscultation of the heart revealed a gallop or "triple" rhythm and a sinus tachycardia in all 15 patients, a precordial systolic murmur of grade I to III intensity in 9, and an accentuated P2 in 12. Cyanosis was prominent in the 2 white patients, and peripheral findings of congestive failure were present in 8.

The ocular fundi were examined in 13 patients and found to be abnormal in 4. This abnormality consisted of narrowing or spasm of the arterioles, with faint cotton-wool-like patches in one and a transient hemorrhage in another.

**Laboratory Findings.** Albuminuria, present in the 2 patients with a diagnosis of pre-eclamptic toxemia during the last trimester of pregnancy, was found in 11 during the period of their congestive failure postpartum. In only 1 did it fail to clear following cardiac compensation. Hemoglobin values were below 10 Gm. per 100 ml. in 1 case with a hypochromic anemia and a level of 6 Gm. per 100 ml., which was promptly corrected with ferrous sulfate. Persistent neutrophilic leukopenia was noted in 2, but was never explained. Significant eosinophilia was never found. Sedimentation rates were, for the most part, within normal limits. Kahn tests were negative in all cases.

Basal metabolism rates, urea clearance tests, various tests of liver function, total serum protein, serum uric acid, sodium, chloride, phosphorus, cholesterol, and glucose tolerance tests done on a number of patients were within normal limits. Serum potassium determined on 3 patients was initially low in case 4, ranging from 14 to 16.7 mg. per 100 ml. Attempts to demonstrate a virus in the serum of case 3 by animal inoculation proved unsuccessful.

**Electrocardiography.** Serial electrocardiograms were obtained in 10 patients. The admission electrocardiogram in each instance showed T-wave inversion in multiple limb and precordial leads. In one half the patients this inversion was asymmetric, as is seen in heart strain or digitalis effect, and in the other half the symmetry and depth of the inversion resembled that seen with myocardial infarction. Significant Q waves or conduction defects were not observed at any time, and the only arrhythmia occurred in case 2 many months after onset of the disease.

Tracings were interpreted as being within normal limits from 1 to 7½ months later in 5 patients; although improvement was noted in the graphs of another 4, some degree of abnormality persisted at the time of the last tracing 9 to 19 months later. One was not followed closely, but a normal tracing was obtained 2½ years later.

**Roentgenology.** Teleröntgenograms of the chest taken upon admission revealed generalized enlargement of the cardiac shadow in all patients. This enlargement was persistent in the 5 who eventually died in congestive failure. In 7 patients the transverse diameter of the heart returned to within normal limits in 3 to 16 weeks, while in another it did not
return to normal until 15 months later. Two patients were not followed closely, but 2½ years later their hearts were found to be within normal limits.

**Differential Diagnosis.** Three patients who did not have peripheral evidence of congestive failure and in whom the significance of the gallop rhythm was not recognized were given a diagnosis suggested by the most prominent symptom. One admitted with sudden, severe abdominal pain (renal embolus?) was initially considered to have a ruptured ectopic pregnancy; another who had a cerebral embolism subsequently underwent a craniotomy because of the suspicion of a brain tumor; and a third was initially isolated as a case of pulmonary tuberculosis because of the presenting symptom of hemoptysis.

When the congestive failure was recognized, the most frequent etiologic considerations by the staff were rheumatic or hypertensive heart disease. These diagnoses were ruled out on lack of confirmatory evidence in the past hospital records and the evolution of the T-wave changes in the electrocardiogram. One patient with a faint early diastolic murmur at the base and a history of polyarthritis 16 years previously probably had rheumatic heart disease in addition, but the changes in the electrocardiogram were considered to support acute myocardial damage as the cause of the congestive failure. Anemia was an additional etiologic consideration in another, but the cerebral embolism, the absence of murmurs, and the vasoconstriction reflected by the hypertension were not considered compatible with this sole etiologic diagnosis.

It is apparent from the literature25, 26 and the experience with these cases that considerable division of opinion as to the nature of the cardiac lesion frequently occurs when this type of case is presented clinically. Not only pericarditis with effusion, but most of the rare causes of heart failure were considered at one time or another in these patients.

**Clinical Course.** With the exception of case 2, the symptoms of congestive heart failure of all patients were much improved following a period of hospitalization varying from 8 to 60 days. This improvement occurred only in response to digitalization in 1 patient whose severe failure persisted after 26 days of bed rest and treatment with mercurial diuretics and was temporarily interrupted by embolic episodes in 2 others.

Blood pressure readings were repeated on 12 patients before hospital discharge and were found to have fallen markedly in each instance. This fall to normotensive levels occurred within a few days to 3 weeks in 10 patients, but was delayed from 1 to 2 months in the remaining 2, 1 of whom had been toxemic at term. This observation was independent of the status of the congestive failure, the heart size, or the electrocardiogram and could only be correlated with the bed rest the patient was receiving. During the first year following the initial cardiac episode the blood pressures of 5 patients were found to be once again at hypertensive levels. In 3 of the 5 elevated blood pressures were associated with continuing congestive failure and were observed to fall markedly again during periods of bed rest in the hospital. By the end of the fourth year an additional 3 patients were noted to be hypertensive.

The systolic murmur noted in 9 patients on admission diminished in intensity in 2 and totally disappeared in another 2 as compensation was restored and cardiac enlargement decreased. In the other 5, 2 of whom were proved to have no valvular lesion on necropsy, the murmur persisted; and in 2 patients it was not heard until after discharge from the hospital.

A remission of signs and symptoms of heart disease occurred in the 10 patients whose cardiac transverse diameter returned to within normal limits. In 4 this was without any other therapy than 2 to 8 weeks of bed rest, whereas in 1 it occurred many months later without relation to therapy. Except for the exacerbations associated with the subsequent pregnancies of 4 of these 10 patients, 1 of whom has failed to make a complete recovery 5 years later, a complete remission has continued in the 3- to 9-year period that has followed. Four of the 5 patients with persistent cardiomegaly had symptomatic remissions lasting from 9 to 28 months, but all 5 eventually became chronic cardiac invalids and died 1 to 8 years after the onset of symptoms.

Ten patients have had 1 or more subsequent full-term pregnancies, and in 6, 1 of these pregnancies has been associated with an exacerbation resembling the original episode. In 2 instances 2 pregnancies with cardiac involvement either alternated with or were followed by another free of such complication. A further remission has occurred in 3 of the 6, but the 2 patients whose heart size had never returned to normal following the initial episode eventually died in congestive failure.

**Comparisons with Previously Reported Cases**

With certain exceptions this description is in general agreement with the original given by Hull. He observed the syndrome most frequently in primiparae while in the present series it occurred most frequently in association with the third pregnancy. Seventy-four per cent of his cases as compared with 40 per cent of the present series were symptomatic by the end of the first postpartum month; however, 73 per cent of the present series had become symptomatic by the end of the seventh postpartum week. Generalized anasarca and a small pulse, prominent features of his cases, were usually
late findings when they occurred in the present series. Hematemesis occurred in 1 of his patients and in 3 of the present series, but has not been reported by other observers. Eventual mortality appeared to be similar in both series.9

Chest and abdominal pain were not mentioned in Hull's description but have been reported by a number of subsequent observers.7, 9, 11, 14, 16, 17 In many instances chest pain has resembled that produced by coronary insufficiency, but it has not always been possible definitely to differentiate it from that due to pulmonary embolism. Sudden severe attacks of abdominal pain have been explained by the necropsy findings of mesenteric thrombosis and splenic and renal infarctions;7 but chronic, less severe abdominal pain associated with other gastrointestinal symptoms have not received adequate explanation except for the finding of pancreatitis in 1 patient at necropsy.11 In 1 instance9 sudden, but transient, excruciating periumbilical pain was the first symptom and preceded the onset of paroxysmal dyspnea by several hours. Chest and abdominal pain have been noted in cases of endocardial fibrosis and idiopathic myocardiopathy unrelated to pregnancy27-30 and thus are not unique for the type of case under discussion.

Abnormal electrocardiograms, especially shallow T-wave inversion, have been noted by all observers, but the magnitude and evolution of the changes described here are reported for the first time. It seems probable that this discrepancy is a result of the greater number of chest leads taken in recent years.

Hull's series of 27 cases included 2 patients who had had a similar episode following a previous pregnancy and another 2 who had subsequent full-term pregnancies, with 1 again developing congestive failure in the postpartum period. Two of Woolford's17 5 cases had subsequent pregnancies, and both again developed congestive failure in the postpartum period. In 1, cardiac involvement followed 3 nonconsecutive pregnancies, the first, third, and fifth.

The only previous observer to report on the frequency of this syndrome was Woolford, who found an incidence of 1 out of every 4,000 obstetrical admissions to the Cincinnati General Hospital.

**Pathology**

Fifteen necropsies have been reported since 1937.6-8, 11, 12, 16 With the exception of case 3 these have shown a soft, flabby, and dilated myocardium with ventricular mural thrombi found especially near the apex. Weights have varied from 260 to 650 Gm. with 6 of the 10 reported weights being between 470 and 525 Gm. No significant disease of the coronary arteries has been described, and in only 1 case7 has a valvular deformity been found. Microscopic features reported include focal and diffuse areas of disintegration of the muscle fibers with occasional hemorrhages, a minimal to moderate cellular response of lymphocytes and macrophages, and myocardial fibrosis; fat droplets in the muscle fibers were an additional finding in 1.11 These changes have been most intense and diffuse in the subendocardial portion of the myocardium, especially in that portion of the myocardium underlying thrombus formation. Focal endocardial swelling with disruption of the overlying endothelial layer and fibrin deposition as found in 1 early case7 may be the basis of subsequent thrombus formation as well as the diffuse endocardial thickening found in long-standing cases.29, 31, 32 It does not appear that these observations differ in any important respect from those found in similar cases unrelated to pregnancy.29, 33

**Etiology**

The cause of the endomyocardial lesions is unknown. Theories as to etiology and pathogenesis of similar lesions unrelated to pregnancy have received consideration elsewhere.34 In cases associated with pregnancy 2 lines of evidence suggest that the activation of the lesion at that particular time was not fortuitous but occurred as the result of unknown factors operating during and shortly after the preceding pregnancy. These were the frequency of recurrence of the syndrome in relation to a subsequent pregnancy, especially where these pregnancies were not consecutive, and Hull's observation that two thirds of cases of heart failure occurring in the postpartum period were due to heart disease of other than the usual etiologic types.

Two further remarkable features were the
significant increase in the incidence of twin births at the time of the last parturition noted here and by others\textsuperscript{6, 9, 17} and the initial acute hypertension associated with the congestive failure. Although the latter has not been an invariable finding, it appears that anything approaching a comparable frequency in similar cases unrelated to pregnancy is to be found only infrequently.\textsuperscript{32, 35} The phenomenon of an initially high blood pressure that returns to normal when the congestive failure accompanying hypertension is improved with treatment is not a new observation.\textsuperscript{36-39} Unexplained, however, is the constancy of the blood pressure phenomena observed in this series.

While neither the clinical nor the pathologic findings of these cases were those of hypertensive heart disease in the usual sense, several features were suggestive of effects usually attributed to coronary insufficiency. These included the previously described pathologic findings that are compatible with anoxia,\textsuperscript{39, 40} the character and radiation of the substernal pain that occurred in at least 5 patients, the configuration and evolution of the T waves in the electrocardiograms, and the results of an oxygen deprivation test in 1 patient. The mechanism of production of such a postulated anoxia is speculative, since minimal vascular changes in cases associated with pregnancy, when described, have been entirely out of relationship to the vast amount of destruction present.\textsuperscript{7}

In any consideration of the etiology of cases of this type the question of beriberi or malnutrition almost invariably arises.\textsuperscript{41- 42} The lack of definite evidence of dietary deficiency in the history, the absence of any physical stigmata, and the favorable course without vitamin therapy leave little basis for consideration of this etiology in the cases described here. Hull concurred in this opinion and stated that early suspicions that it might represent a nutritional deficiency disease have long since been disproven.\textsuperscript{43}

Upper respiratory infections, influenza, and grippe are occasional etiologic considerations when cases of idiopathic myocardial failure occurring in young individuals are reported in the literature, and 3 of these patients did interpret their initial respiratory symptoms as due to "a cold." In the present series of cases it appears that the symptoms of night cough, hemoptysis, and paroxysmal nocturnal dyspnea were the earliest clinical manifestations of myocardial failure rather than its cause.

Marked similarities between this syndrome and the congestive failure produced by desoxycorticosterone suggest that endocrine investigations might be fruitful. Virologic studies are also needed for the further advancement of knowledge concerning these idiologic disorders of the myocardium.

**TREATMENT**

Any consideration of treatment should emphasize that these patients have degenerative heart disease of unknown etiology but that early it is potentially reversible. Thus therapeutic efforts should not rest with the control of the symptoms of congestive failure but should include prolongation of bed rest until after the heart size has returned to within normal limits, when this objective is attainable. While bed rest alone is sufficient treatment in many cases, in others the need for digitalization becomes apparent early. Anticoagulants have not been used in this series. Since embolization, when it occurs, is frequently a very early event in the course of this condition, effective use of these agents would depend on their institution at the earliest possible moment.

**SUMMARY**

The syndrome of idiopathic myocardial failure occurring in the last trimester of pregnancy or the puerperium is characterized by symptoms of congestive failure, which, early, is predominantly left sided, chest and abdominal pain, embolic phenomenon in 25 to 40 per cent, acute hypertension, gallop rhythm, generalized dilatation of the heart, and T-wave inversion. Pathologic findings, which resemble similar cases unrelated to pregnancy, consist of a soft, flabby, dilated myocardium, ventricular mural thrombi, endocardial thickening of variable extent, and focal and diffuse areas of myocardial necrosis especially in the subendocardial portion of the ventricular wall. Approximately two thirds of the cases make complete clinical recoveries, but the syndrome tends to recur with subsequent
pregnancies. Experience with the present series indicates that these cases are usually considered clinically to have either rheumatic or hypertensive heart disease, one of the rarer recognized forms of myocardial disease, or a disease suggested by symptoms coming from organ embolization.

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SUMMARIO IN INTERLINGUA

Le syndrome de idiopathic disfallimento myocardic occurrente durante le trimestre final del pregnancy o durante le puerperio es characterizate per symptomasy de disfallimento congestive (que in le phases initial occurre predominantemente al latere sinistre), dolores thoracic e abdominal, phenomenos embolic (occurrente in inter 25 e 40 pro cento del cases), hypertension acute, rhythmo de galopo, dilatation generalisate del corde, e inversion del unda T. Le constataatione pathologic, que resimila cases comparabile sed non connectite con pregnantias, include un molle e flaccide myocardio dilatate, thrombos de pariete ventricular, spissification del endocardio de varie grados, e areas focal e diffuse de necrose myocardial, specialmente in le portion sub-endocardial del pariete ventricular. Circa duo tertio del patientes effectua un restablimento complete ab le puncto de vista clinic, sed le syndrome tende a recurrer con pregnantias subsequente. Observationes colligite in le presente serie indica que iste cases es generalmente considerate per le clinicos como cases de morbo cardiac rheumatic o hypertensive, de un del formas minus frequente de morbo myocardial, o de un morbo suggerite per symptomas causate per embolisation de organos.

REFERENCES

1. VIRCHOW, R.: Sitzung der Berliner Geburtshilflicher Gesellschaft, 1870 (Quoted by Porak).
MYOCARDIAL FAILURE IN LAST TRIMESTER OF PREGNANCY AND PUERPERIUM


43 HULL, E.: Personal communication.


These experiments were initiated in response to recent reports by others that baroreceptors are incapable of limiting the blood pressure rise of epinephrine and norepinephrine. The present experiments have shown that the pressor response to a given dose of epinephrine was enhanced after elimination of the buffering activity of the carotid sinus baroreceptors, provided the systemic blood pressure level before and after denervation was kept constant. The well-known function of the carotid sinuses was reemphasized. 

Aviado
Idiopathic Myocardial Failure in the Last Trimester of Pregnancy and the Puerperium

W. R. MEADOWS

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