

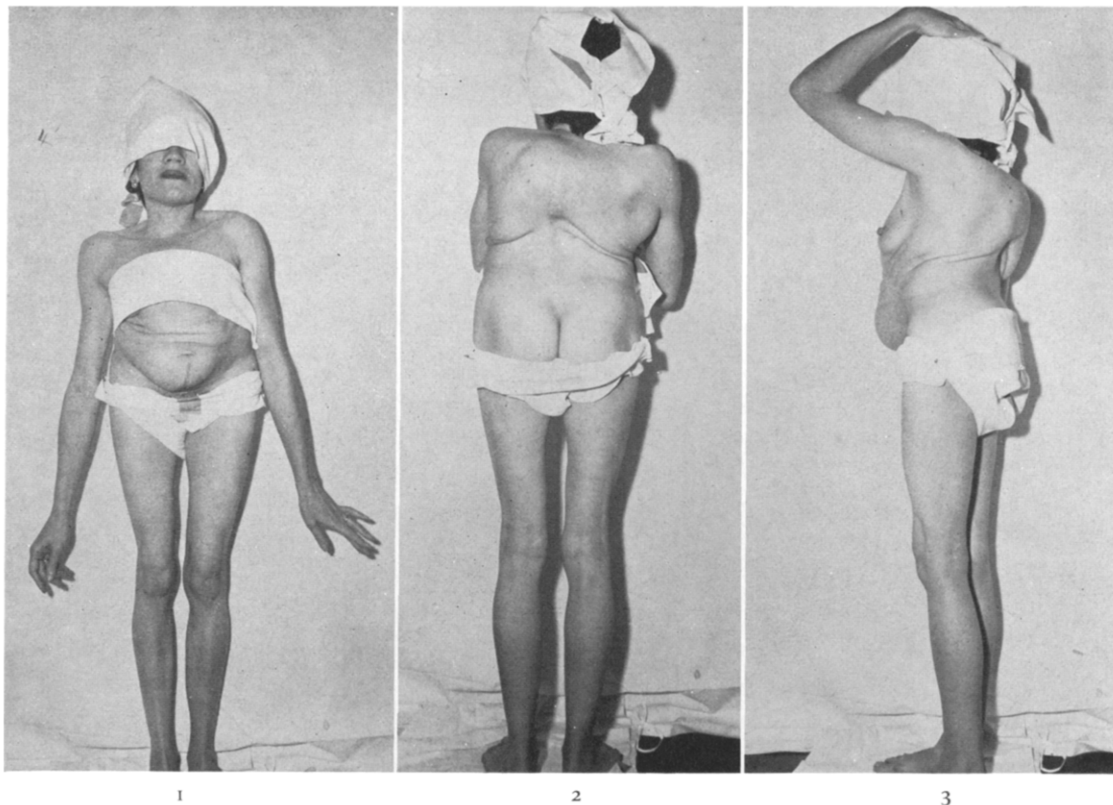
# PREGNANCY AND KYPHOSCOLIOSIS\*

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**M**ARKED deformity of the thoracic cage as a complication of pregnancy is relatively rare. Mendelson<sup>1</sup> reported

gravidity and because improvement in the vital capacity occurred as pregnancy approached term.



FIGS. 1 to 3.

an incidence of 0.01 per cent for this complication at the New York Lying-In Hospital. A similar incidence was reported by Fearl<sup>2</sup> and Higginbotham<sup>3</sup> (1 in 12,000 and 1 in 18,790 deliveries, respectively). Since 1948, at the United States Naval Hospital in Portsmouth, Virginia, this complication has occurred once in 15,067 deliveries.

This case is added to the scant literature on the subject because of the additional complicating factor of preeclampsia superimposed on essential hypertension in an elderly primi-

Mrs. E. W., an intelligent, forty-three year old, white primigravida, reported to this clinic in January, 1952, stating that she believed herself to be pregnant. Past history revealed that at the age of three she had been afflicted with Pott's disease of the spine and had been treated by bedrest, traction and braces until the age of fourteen. At the time of her marriage she was advised against pregnancy because of the residual deformity and for fourteen years practiced contraception successfully. In July of 1951 she was diagnosed as being menopausal

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and discontinued contraception. On September 15th she had her last normal menstrual period. In November the patient felt that she was pregnant, with the usual presumptive symptomatology. In December she was convinced and had protracted hyperemesis. A positive frog

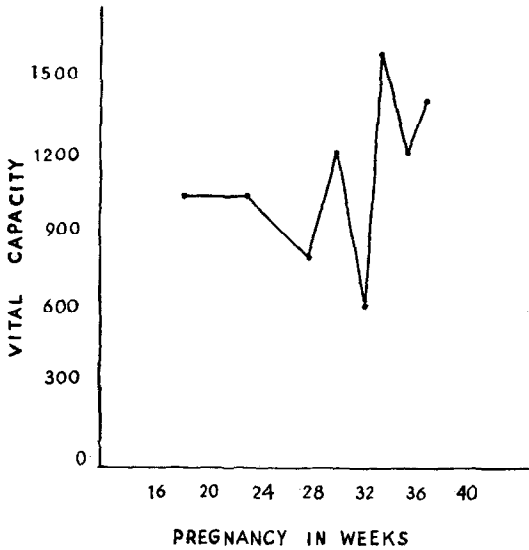


FIG. 4.

test was obtained and the patient was referred to this activity for possible therapeutic abortion. Additional history revealed that she had had an elevated systolic blood pressure (140 to 142) for years, the diastolic not being known, and that there had been a persistent tachycardia for at least six years for which quinidine had been prescribed. No history suggesting cardiac or renal failure was obtained.

Physical examination revealed an emotionally upset, dehydrated, frightened woman with the marked deformity of the thoracic cage. (Figs. 1 to 3.) Evaluation of the cardio-respiratory system revealed no gross impairment other than a blood pressure of 140/90 and a pulse rate of 90. The response to exercise was essentially normal. Mensuration revealed the patient to weigh 76 pounds, to stand 51 inches and to have a decreased trunk/lower extremity ratio. The right leg was  $1\frac{1}{2}$  inches shorter than the left and the pubic symphysis to zyphoid diameter measured 6 inches. There was a decided tilt to the pelvis, but clinically it was believed to be adequate for vaginal delivery (later confirmed by x-ray pelvimetry). The existence of a normal intrauterine gestation was confirmed.

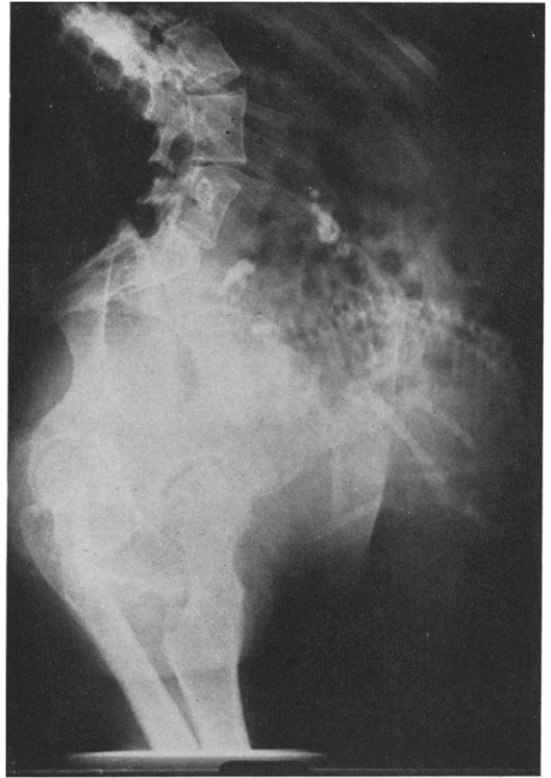


FIG. 5.

The patient was referred to the medical service for consultation where photofluorographic and electrocardiographic studies, vital capacity, exercise tolerance, venous pressure, circulation time and kidney function studies were obtained. It was their conclusion that she was a class 1 cardiac and that interruption of the pregnancy was not mandatory.

A thorough explanation of the problem was presented to the patient and the decision to accept the risks involved in order to achieve motherhood was made.

The patient was rigidly followed by the medical and obstetric services, with weight restriction, salt poor diet, ample rest and mild sedation prescribed. The prenatal course was essentially uneventful except for a persistent tachycardia and blood pressure of 140/90 until the twenty-eighth week. At that time the blood pressure was 160/110. A trace of albuminuria and edema was present, and the vital capacity was 800 cc. The patient was immediately hospitalized. On complete bedrest, increased sedation and ammonium chloride the albuminuria and edema rapidly cleared; the

blood pressure stabilized at 140/90 to 160/100 and the pulse rate at 90 to 110; the vital capacity increased to 1,200 cc. Blood urea nitrogen, uric acid  $\text{CO}_2$  combining power, phenolsulfonphthalein, Mosenthal, total protein with A/G ratio, red blood count, hemoglobin and hematocrit tests were all within limits considered normal for pregnancy. The urinary output consistently ranged over 1,000 cc. per day. The patient's condition remained essentially static except for mild fluctuations in blood pressure and pulse until the thirty-sixth week, at which time the blood pressure rose to 160/110 to 170/120, the patient gained three pounds in forty-eight hours and peripheral edema returned. It was decided to terminate the pregnancy.

As the cervix was not ripe, a classic cesarian section with bilateral tubal ligation was performed under local anesthesia supplemented by cyclopropane for closure. The infant, a girl, presented by the breech weighed 3 pounds 8 ounces and survived. The placenta was small and fibrotic.

The postpartum course was uneventful and the patient was discharged on the ninth postoperative day. Seven days later the blood pressure was 150/100 and the pulse rate 80. The patient was asymptomatic but stated that she

had returned to the emergency room on her eleventh postoperative day where, because of cough and shortness of breath, she had been digitalized by the internist on duty. The patient was followed closely as an outpatient and within three months the blood pressure and pulse had returned to the prepregnant state. At six months laboratory evaluation revealed no residual cardiovascular renal impairment. (Figs. 4 and 5.)

In Figure 4 the vital capacities at the various periods are depicted. It is believed that the impairment in vital capacity was due to the upward displacement and splinting of the diaphragm by the pregnant uterus. However, in the latter weeks of pregnancy improvement in the vital capacity occurred. This was believed due to the shifting of the gravid uterus from an upright to a horizontal plane, which allowed increased diaphragmatic excursion.

#### REFERENCES

1. MENDELSON, C. L. Pregnancy and kyphoscoliotic heart disease. *Am. J. Obst. & Gynec.* 56: 457, 1948.
2. FEARL, C. S. Kyphoscoliosis and pregnancy; its cardiorespiratory implications. *West. J. Surg.*, 59: 411, 1951.
3. HIGGINBOTHAM, W. H. Kyphoscoliotic heart disease complicating pregnancy. *Am. J. Obst. & Gynec.*, 65: 424, 1953.

