

OUTCOME OF PREGNANCY IN WOMEN WITH MODERATE OR SEVERE RENAL INSUFFICIENCY

DAVID C. JONES, M.D., AND JOHN P. HAYSLITT, M.D.

ABSTRACT

Background Pregnant women with mild preexisting renal disease have relatively few complications of pregnancy, but the risks of maternal and obstetrical complications in women with moderate or severe renal insufficiency remain uncertain.

Methods We determined the frequency and types of maternal and obstetrical complications and the outcomes of pregnancy in 67 women with primary renal disease (82 pregnancies). All the women had initial serum creatinine concentrations of at least 1.4 mg per deciliter (124 μ mol per liter) and gestations that continued beyond the first trimester.

Results The mean (\pm SD) serum creatinine concentration increased from 1.9 ± 0.8 mg per deciliter (168 ± 71 μ mol per liter) in early pregnancy to 2.5 ± 1.3 mg per deciliter (221 ± 115 μ mol per liter) in the third trimester. The frequency of hypertension rose from 28 percent at base line to 48 percent in the third trimester, and that of high-grade proteinuria (urinary protein excretion, >3000 mg per liter) from 23 percent to 41 percent. For the 70 pregnancies (57 women) for which data were available during pregnancy and immediately post partum, pregnancy-related loss of maternal renal function occurred in 43 percent. Eight of these pregnancies (10 percent of the total) were associated with rapid acceleration of maternal renal insufficiency. Obstetrical complications included a high rate of preterm delivery (59 percent) and growth retardation (37 percent). The infant survival rate was 93 percent.

Conclusions Among pregnant women with moderate or severe renal insufficiency, the rates of complications due to worsening renal function, hypertension, and obstetrical complications are increased, but fetal survival is high. (N Engl J Med 1996;335:226-32.)

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THE editorial comment “children of women with renal disease used to be born dangerously or not at all — not at all if their doctors had their way,”¹ reflects an early view of the effect of kidney disease on pregnancy. However, among pregnant women with mild renal insufficiency (serum creatinine, <1.4 mg per deciliter [124 μ mol per liter]) fetal survival is only moderately reduced and the underlying disease is not irreversibly worsened.²⁻⁶ In contrast, the presence of moderate or severe renal insufficiency in pregnancy has been reported to accelerate the underlying disease and markedly reduce fetal survival.^{2,7}

This study was organized as a joint project among six medical centers to determine the frequency and types of complications during pregnancy in women with preexisting moderate or severe renal insufficiency and the types of fetal and neonatal complications.

METHODS

We analyzed the outcomes of 82 pregnancies in 67 women with preexisting primary renal disease who were seen at the six centers between 1971 and 1993 (13 women were followed during 2 pregnancies, and 1 during 3 pregnancies). All the women had a serum creatinine concentration of at least 1.4 mg per deciliter before pregnancy or at the first antepartum visit and a pregnancy that continued beyond the first trimester. In 74 pregnancies (90 percent) the women had renal disease before becoming pregnant. The renal disease was classified as either chronic glomerulonephritis or chronic tubulointerstitial disease, on the basis of a biopsy or the extent of proteinuria and characteristic changes on intravenous pyelography.

Serum creatinine was used as an index of the glomerular filtration rate. Renal insufficiency was regarded as moderate when the serum creatinine concentration was 1.4 to 2.4 mg per deciliter (124 to 220 μ mol per liter) and severe when it was 2.5 mg per deciliter (221 μ mol per liter) or more. Since the serum creatinine concentration is not linearly related to changes in the glomerular filtration rate, we used the equation $1/\text{serum creatinine}$ ($1/S_{Cr}$), whose result correlates linearly with the filtration rate,⁸ to estimate changes in the glomerular filtration rate during and after pregnancy as compared with the values at the initial antepartum visit in the first or second trimester. To account for nonspecific variations, a 25 percent change in the value of $1/S_{Cr}$ was judged to indicate a change in the glomerular filtration rate. End-stage renal disease was defined as a serum creatinine concentration above 6.0 mg per deciliter (530 μ mol per liter).

Hypertension was defined as a mean arterial blood pressure above 105 mm Hg, with the mean pressure calculated as $[\text{systolic pressure} + (\text{diastolic pressure} \times 2)]/3$.⁹ The criterion for the development of hypertension during pregnancy was the finding of a mean arterial blood pressure above 105 mm Hg in women whose mean arterial pressure had previously been 105 mm Hg or less. Exacerbation of preexisting hypertension was defined as an increase in the mean arterial blood pressure of 20 mm Hg.

Proteinuria was classified by quantitative or semiquantitative measurements as absent if testing with a dipstick showed no protein or trace levels or if the level was below 300 mg per liter, low grade if the dipstick showed a value of 1+ or 2+ or the level was 300 to 3000 mg per liter, and high grade if the dipstick showed a value of 3+ or 4+ or the level exceeded 3000 mg per liter. For the purposes of evaluating pregnancy-related changes in the glomerular filtration rate, blood pressure, and proteinuria, we used the first measurement of these indexes, when performed in the first or second trimester, as the base-line values.

From the Departments of Obstetrics and Gynecology (D.C.J.) and Internal Medicine (J.P.H.), Yale University, New Haven, Conn. Address reprint requests to Dr. Jones at Yale University School of Medicine, Department of Obstetrics and Gynecology, P.O. Box 208063, New Haven, CT 06520-8063.

Delivery before 37 weeks of gestation was classified as preterm. Neonatal death was defined as death before discharge from the hospital. An infant was considered small for gestational age if he or she was below the 10th percentile of size for gestational age at delivery, according to recently published tables.¹⁰

Treatment of neither renal disease nor hypertension was standardized. Management of these conditions was left to the discretion of the attending physicians.

Statistical analysis was performed with Statview¹¹ on a Macintosh Duo 280c computer (Apple Computer, Cupertino, Calif.). Student's t-test, Fisher's exact test, the chi-square test, and McNemar's test were used as appropriate. Tables showing more complete results are available from the National Auxiliary Publications Service (NAPS).*

RESULTS

The characteristics of the 67 women and their 82 pregnancies are shown in Table 1. Preconception data were available for 79 percent of the pregnancies, and data from one or more follow-up visits conducted within 12 months after delivery were available for 93 percent. Seventy-five percent of the deliveries occurred after 1984 (median, 1987; range, 1971 to 1993), when recent advances in obstetrical care and neonatal intensive care units were available. Seventy-four percent of the women were white, 12 percent were Asian, and 12 percent were black. The underlying renal disease was categorized as chronic glomerulonephritis in 34 women (51 percent) and chronic tubulointerstitial disease in 33 (49 percent). The severity of the women's renal insufficiency, classified as moderate during 59 pregnancies (72 percent) and severe during 15 pregnancies (18 percent), the incidence and severity of hypertension and proteinuria at the initial antepartum visit, and the number treated with antihypertensive medications are shown in Table 1.

Maternal Complications during and after Pregnancy

The effects of pregnancy on renal function, mean arterial blood pressure, and proteinuria are shown in Table 2. On the basis of data available from early and late in pregnancy, the mean (±SD) serum creatinine concentration increased from a base-line value of 1.9±0.8 mg per deciliter (168±71 μmol per liter) to 2.5±1.3 mg per deciliter (221±115 μmol per liter) in the third trimester (P<0.001). This increase was accounted for, at least in part, by an increase in the number of pregnancies in which renal insufficiency was severe in the third trimester (36 percent, vs. 17 percent at base line; P=0.035). In addition, the incidence of hypertension (48 percent vs. 28 percent, P=0.01) and high-grade proteinuria (41 percent vs. 23 percent, P=0.007) nearly doubled

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TABLE 1. CHARACTERISTICS OF 67 WOMEN WITH RENAL DISEASE AND THEIR PREGNANCIES.*

CHARACTERISTIC	VALUE	INDIVIDUAL VISITS	CUMULATIVE VISITS
Number of pregnancies†	82		
Age — yr	28±6		
Year of delivery			
Median	1987		
Range	1971-1993		
Type of renal disease — no. of women (no. of pregnancies)			
Glomerular	34 (39)		
Tubulointerstitial	33 (43)		
Preconception serum creatinine values available — no. of pregnancies (%)	65 (79)		
Time of measurement — mo before pregnancy			
Median	6		
Range	1-96		
Time of initial antepartum visit — no. of pregnancies (%)			
First trimester	62 (76)		
Second trimester	17 (21)		
Third trimester	3 (4)		
Follow-up ≥12 months — no. of women (%)	52 (78)		
Duration of contact after last pregnancy — mo			
Median	36		
Range	12-180		
Follow-up postpartum visit — no. of pregnancies (%)			
6 wk	61 (74)	61 (74)	
6 mo	58 (71)	72 (88)	
12 mo	47 (57)	76 (93)	
			VALUE AT INITIAL ANTEPARTUM VISIT
Serum creatinine — mg/dl‡	1.9±0.8		
Serum creatinine category — no. of pregnancies (%)‡			
1.1-1.3 mg/dl§	8 (10)		
1.4-2.4 mg/dl	59 (72)		
2.5-5.5 mg/dl	15 (18)		
Arterial blood pressure			
Mean — mm Hg	99±17		
≤105 mm Hg (mean, 91±10) — no. of pregnancies (%)	57 (70)		
Antihypertensive therapy — no. of women	18		
>105 mm Hg (mean, 118±14) — no. of pregnancies (%)	25 (30)		
Antihypertensive therapy — no. of women	11		
Proteinuria — no. of pregnancies (%)¶			
None	17 (22)		
Low grade (300-3000 mg/liter)	41 (52)		
High grade (>3000 mg/liter)	21 (27)		

*Because of rounding not all categories total 100 percent. Plus-minus values are means ±SD.

†Thirteen women were followed during two pregnancies, and one during three pregnancies.

‡To convert values for serum creatinine to micromoles per liter, multiply by 88.4.

§In these cases the women's serum creatinine concentrations were at least 1.4 mg per deciliter before conception and decreased early in pregnancy.

¶Data were obtained during the 79 pregnancies in which urinary protein excretion was measured at the first visit.

TABLE 2. EFFECT OF PREGNANCY ON RENAL DISEASE.*

VARIABLE†	1ST OR 2ND TRIMESTER	3RD TRIMESTER
Serum creatinine		
No. of pregnancies	76	76
Mean — mg/dl	1.9±0.8	2.5±1.3
<1.4 mg/dl — no. (%)	8 (11)	7 (9)
≥1.4–2.4 mg/dl — no. (%)	55 (72)	42 (55)
≥2.5 mg/dl — no. (%)	13 (17)	27 (36)
P value‡	0.035	
Arterial blood pressure		
No. of pregnancies	78	75
Mean — mm Hg	99±17	105±18
≤105 mm Hg — no. (%)	56 (72)	39 (52)
>105 mm Hg — no. (%)	22 (28)	36 (48)
P value‡	0.01	
Proteinuria		
No. of pregnancies	70	68
None — no. (%)	16 (23)	13 (19)
Low grade (300–3000 mg/liter) — no. (%)	38 (54)	27 (40)
High grade (>3000 mg/liter) — no. (%)	16 (23)	28 (41)
P value‡	0.07	
GESTATIONAL CHANGES§		
Glomerular filtration rate		
No. of pregnancies	70	
Change in serum creatinine (mg/dl)	+0.5¶	
Remained stable — no. (%)	55 (79)	
Decreased by 25% — no. (%)	14 (20)	
Increased by 25% — no. (%)	1 (1)	
Arterial blood pressure		
No. of pregnancies	74	
Change in mean arterial blood pressure (mm Hg)	+6.1**	
Persistent normotension — no. (%)	35 (47)	
Stable hypertension — no. (%)	17 (23)	
New-onset hypertension — no. (%)	19 (26)	
Exacerbation of hypertension — no. (%)	3 (4)	
Proteinuria		
No. of pregnancies	59	
Stable — no. (%)	36 (61)	
Decreased — no. (%)	6 (10)	
Increased — no. (%)	17 (29)††	

*Because of rounding, not all categories total 100 percent. Plus-minus values are means ±SD.

†To convert values for serum creatinine to micromoles per liter, multiply by 88.4.

‡The P value is for the comparison of the distribution of values in the first or second trimester with that in the third trimester (by the chi-square test or Fisher's test).

§The results were based on paired data obtained in both early pregnancy and the third trimester. Changes in the glomerular filtration rate were estimated with the equation $1/S_{Cr}$.

¶P<0.001 by Student's t-test.

||One woman had hypertension in the first trimester with an exacerbation of hypertension and delivery in the second trimester.

**P=0.006 by Student's t-test.

††P<0.001 by the chi-square test.

during the third trimester as compared with the base-line values.

In the 70 pregnancies (57 women) with paired data on renal function, there was a gestational decline in the glomerular filtration rate in 14 (20 percent); the mean reduction in this subgroup was 44 ± 14 percent. In the 11 pregnancies for which estimates of the decline in renal function were available before the women became pregnant, the decline accelerated during pregnancy. Maternal renal function declined immediately after delivery in an additional 16 pregnancies (23 percent) in women whose glomerular filtration had been stable during gestation, so that the total rate of pregnancy-related decline in renal function was 43 percent.

Because hypertension has been reported to increase the risk of a pregnancy-related reduction in the glomerular filtration rate,^{4,12,13} we sought to determine whether hypertension during pregnancy affected renal function both during and after pregnancy in these women. In this analysis the presence of hypertension was correlated with both the mean value of $1/S_{Cr}$ and a significant decline in the glomerular filtration rate (defined as a 25 percent decrease in the $1/S_{Cr}$ value). The presence of hypertension at the first antepartum examination was associated with a significantly lower mean $1/S_{Cr}$ value in the third trimester than the absence of hypertension (25 percent decrease vs. 12 percent decrease, $P=0.01$), but not with a decline in the glomerular filtration rate (28 percent vs. 17 percent of pregnancies, $P=0.30$). However, by six months post partum, hypertension in early pregnancy was not significantly associated with either the mean $1/S_{Cr}$ value (14 percent decrease, vs. a 27 percent decrease in the absence of hypertension; $P=0.20$) or the incidence of a decline in the glomerular filtration rate (decline in 35 percent vs. decline in 45 percent, $P=0.60$).

In contrast, hypertension occurring in the third trimester was associated with both a lower $1/S_{Cr}$ value in late gestation than was normotension (25 percent decrease vs. 8 percent decrease, $P<0.001$) and a decline in the glomerular filtration rate (decrease in 32 percent vs. 11 percent, $P=0.004$). Furthermore, when the severity of hypertension in the third trimester was categorized as mild or moderate (mean arterial blood pressure, 106 to 123 mm Hg) or severe (>123 mm Hg), the respective mean reductions in the value for $1/S_{Cr}$ during the third trimester were two and five times that with pregnancies in which maternal blood pressure was normal (18 percent decline vs. 7 percent decline, $P=0.02$; and 37 percent decline vs. 7 percent decline, $P<0.001$). However, third-trimester hypertension had no adverse effect on renal function six months after delivery.

Complications were analyzed separately in the 15 women (15 pregnancies) with severe renal insuffi-

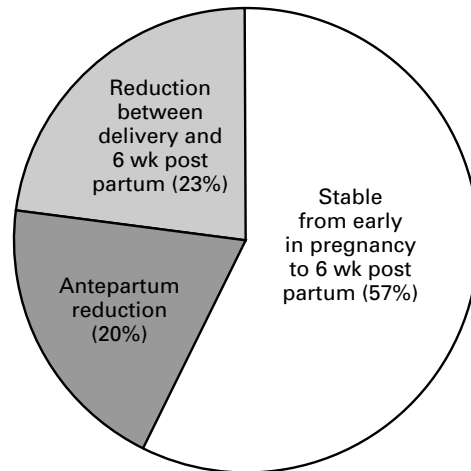
ciency at the first antepartum examination. The initial mean serum creatinine concentration was 3.4 ± 0.1 mg per deciliter (301 ± 9 μ mol per liter), and in three pregnancies the values exceeded 4.0 mg per deciliter (354 μ mol per liter). The incidence of hypertension at the onset of pregnancy was similar in the group with severe renal insufficiency and the group with only moderate renal insufficiency (47 percent vs. 27 percent, respectively; $P = 0.21$), as was the incidence of hypertension in the third trimester (64 percent vs. 44 percent, $P = 0.24$) and of exacerbation of hypertension during pregnancy (62 percent vs. 76 percent, $P = 0.31$). Moreover, the two groups were similar with respect to the reduction in the glomerular filtration rate during pregnancy and six months post partum.

Follow-up Studies after Pregnancy

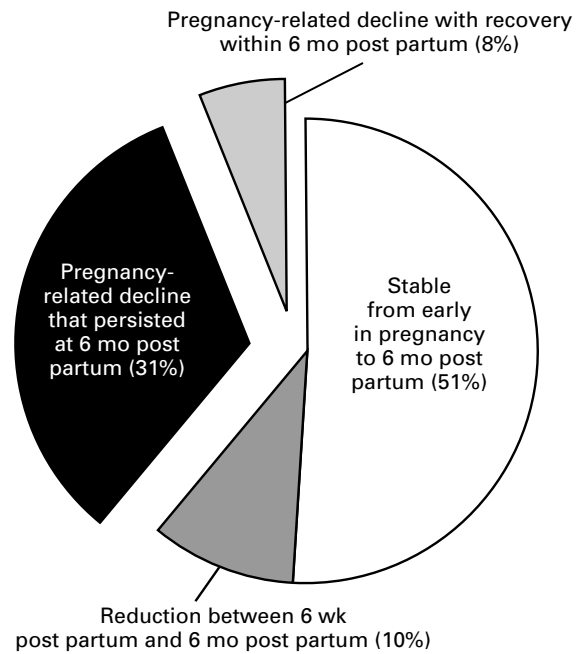
The results of follow-up studies of the maternal glomerular filtration rate are shown in Figure 1. In 51 percent of pregnancies the glomerular filtration rate measured six months after delivery was similar to the initial antepartum value. In 8 percent of pregnancies, renal function declined during pregnancy or within six weeks after delivery but recovered within six months. However, in 10 percent of the 70 pregnancies for which data were available, renal function decreased between six weeks and six months after delivery. A pregnancy-related loss of renal function persisted for six months post partum in 31 percent of pregnancies.

By 12 months post partum, eight women had had progression to end-stage renal failure, including seven who had a pregnancy-related loss of renal function and one with stable renal function during and immediately after pregnancy. In the remaining women (62 pregnancies, 39 with an initially stable course and 23 in which there was a pregnancy-related decline in the glomerular filtration rate), renal function declined slightly but progressively, such that the serum creatinine concentrations were 2.4 ± 1.1 mg per deciliter (212 ± 97 μ mol per liter) and 2.9 ± 0.9 mg per deciliter (256 ± 80 μ mol per liter), respectively ($P = 0.17$). These results suggest that the contribution of the postpartum loss of renal function resulting from a pregnancy-related decline in the glomerular filtration rate was relatively small in most women.

The changes in serum creatinine concentrations during and after pregnancy are shown in Figure 2. Women with higher base-line serum creatinine concentrations (≥ 2.0 mg per deciliter) more often had severe renal insufficiency within six months after delivery than women with lower initial serum creatinine concentrations. An accelerated decline in the glomerular filtration rate occurred during only 1 of 49 pregnancies (2 percent) in which the mother's initial serum creatinine value was below 2.0 mg per



Glomerular Filtration Rate during Pregnancy



Glomerular Filtration Rate 6 mo Post Partum

Figure 1. Changes in the Glomerular Filtration Rate in Women with Primary Renal Disease before and after Delivery.

The segments pulled away from the circle represent pregnancies complicated by a pregnancy-related decline in the glomerular filtration rate, as described in the Methods section. A measurable reduction in the glomerular filtration rate was defined as a reduction of at least 25 percent in the $1/S_{Cr}$ value.

deciliter, 3 of 9 pregnancies (33 percent) in which serum creatinine values were 2.0 to 2.4 mg per deciliter, and 4 of 12 pregnancies (33 percent) in which serum creatinine values were 2.5 mg per deciliter or higher.

Pregnancy Outcome and Neonatal Complications

The frequency of preterm delivery and cesarean section was high (59 percent for both) (Table 3), as compared with the rates expected in the general population (10 percent and 20 percent, respectively). Fetal growth was reduced. The mean birth weight was 2239±839 g, and the birth weight was below the 10th percentile in 37 percent of the infants, nearly four times the expected rate. In addition to overt growth retardation, the majority of infants had birth weights below the 50th percentile for gestational age (Fig. 3). Four infants were stillborn at 23, 27, 27, and

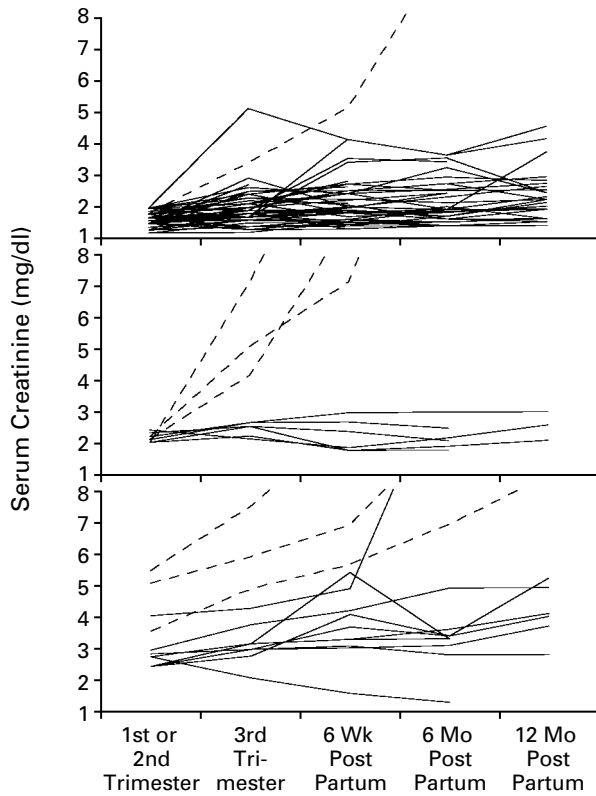


Figure 2. Serum Creatinine Concentrations in Women with Primary Renal Disease during and after Pregnancy, According to the Concentration Measured Early in Gestation.

Dashed lines represent women who had a pregnancy-related decline in renal function and subsequent progression to end-stage renal disease within one year post partum. Data are stratified according to the serum creatinine concentration at the onset of gestation: <2.0 mg per deciliter in the top panel, 2.0 to 2.4 mg per deciliter in the middle panel, and ≥2.5 mg per deciliter in the bottom panel. To convert values for serum creatinine to micromoles per liter, multiply by 88.4.

TABLE 3. OUTCOMES OF 82 PREGNANCIES AND NEONATAL COMPLICATIONS.

VARIABLE	VALUE
Preterm delivery (<37 wks) — no. (%)	48 (59)
Induction of labor — no. (%)	8 (10)
Delivery by cesarean section — no. (%)	48 (59)
Indication	
Fetal distress	4
Intrauterine growth retardation	11
Macrosomia	1
Dystocia	2
Breech position	2
Maternal renal deterioration	5
Maternal hypertension or preeclampsia	11
None reported	12
Mean (±SD) birth weight — g*	2239±839
Small for gestational age (<10th percentile) — no. (%)*	28 (37)
Death — no. (%)	6 (7)
Stillbirth — rate per 1000 births	49
Neonatal death — rate per 1000 births	24
Admission to neonatal intensive care unit — no. (% of liveborn infants)	28 (37)
Indication	
Prematurity, intrauterine growth retardation, or both	25
Sepsis	1
Unknown	2

*Data on birth weight were available for only 76 of the 82 pregnancies.

34 weeks, the last two born to one woman. Despite maternal complications, a high frequency of preterm deliveries, and an increase in growth retardation, the overall fetal survival rate was 93 percent. There were two neonatal deaths due to prematurity: one was delivered at 20 weeks because of the development of the HELLP syndrome (hemolysis, elevated liver-enzyme levels, and low platelet counts),¹⁴ and the other at 23 weeks because of severe preeclampsia. Twenty-eight neonates were admitted to an intensive care nursery, most because of prematurity.

We compared the 15 pregnancies (15 women) in which the base-line serum creatinine values were at least 2.5 mg per deciliter with the remaining 67 pregnancies (52 women) with lower values. The frequency of preterm deliveries was 73 percent and that of intrauterine growth retardation 57 percent in the pregnancies associated with severe renal insufficiency, as compared with 55 percent and 31 percent, respectively, in pregnancies associated with moderate renal insufficiency (P=0.25 and P=0.12, respectively). Fetal and neonatal survival in the subgroup with severe renal insufficiency was 100 percent. The six perinatal deaths were associated with pregnancies characterized by moderate renal insufficiency at the onset of pregnancy; in five of these pregnancies the serum creatinine concentration was below 2.0 mg per deciliter.

A gestational decrease in renal function was associated with a lower birth weight than was stable re-

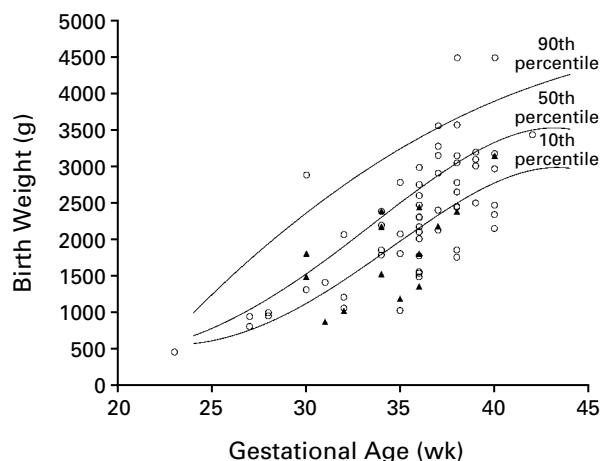


Figure 3. Birth Weights of Infants Born to Women with Moderate (○) or Severe (▲) Renal Insufficiency.

The curves for the normal-birth-weight percentiles were obtained from Amini et al.¹⁰

nal function (1883 vs. 2373 g, $P=0.05$), but not with increased intrauterine growth retardation or reduced fetal survival. The presence of hypertension at the onset of gestation was not correlated with fetal survival, preterm delivery, or growth retardation. When present in the third trimester, hypertension was associated with a higher rate of preterm delivery (72 percent vs. 46 percent, $P=0.02$) but not with increased intrauterine growth retardation (36 percent vs. 38 percent, $P=0.86$) or reduced fetal survival. The presence of high-grade proteinuria at any time during pregnancy had no effect on the outcome of pregnancy.

DISCUSSION

This study was performed to determine the maternal complications, immediate and long-term effects of gestation on underlying disease, and outcome of pregnancy in women with moderate or severe renal insufficiency at the onset of pregnancy. Maternal complications were frequent and included an increase in hypertension and high-grade proteinuria. In addition, there was a pregnancy-related loss of renal function (a loss occurring during pregnancy or within six weeks after delivery) in nearly half of all cases, and in 23 percent of this subgroup (10 percent of the total series), there was a rapid progression to end-stage renal failure within six months after delivery. However, after the exclusion of the women with rapid progression to end-stage renal failure, the postpartum serum creatinine concentrations at 12 months were approximately the same in this subgroup of women as in women with stable renal function during pregnancy. The risk of accelerated progression to end-stage renal failure was highest

when the serum creatinine concentration was above 2.0 mg per deciliter at the beginning of pregnancy.

Although fetal survival was only moderately reduced, there were obstetrical complications in more than half the pregnancies. Growth retardation, prematurity, and maternal complications were the factors most often responsible for delivery by cesarean section and admission to a neonatal intensive care unit. The effect of renal disease on the growth and development of the surviving infants was not further evaluated.

It is useful to compare the present study with studies of pregnancy in women with primary renal disease characterized by normal or nearly normal function (serum creatinine, <1.4 mg per deciliter). Katz and associates reported the outcome of 121 pregnancies in 89 women.³ Hypertension was present in 23 percent of pregnancies, and a small and usually reversible decrease of renal function occurred in 16 percent. The authors concluded that pregnancy did not substantially alter the natural course of renal disease. Obstetrical complications included preterm delivery in 20 percent and intrauterine growth retardation in 24 percent. Infant survival was 89 percent. The results of most subsequent studies were similar.^{2,4-6} Therefore, except for infant survival, the incidences of maternal and obstetrical complications are approximately twice as high in women with at least moderate renal insufficiency as in women with normal or nearly normal renal function. In addition, pregnancy-related loss of renal function is an important complication in women with moderate or severe renal insufficiency.

In early small studies of pregnancy in women with moderate renal insufficiency, fetal survival rates were 50 percent or less.^{2,7,13,15,16} However, in recent studies of women with primary renal disease and moderate or severe renal insufficiency (studies involving 19 to 37 pregnancies),^{12,17,18} the fetal survival rates were 76 to 80 percent, and 16 to 50 percent of women had a gestational loss of renal function. We have extended those findings by providing estimates of the incidence of maternal and fetal complications in a larger series of pregnant women. In addition, follow-up on nearly all women up to 12 months after delivery permitted an assessment of the long-term effects of pregnancy on the natural course of renal disease. This analysis should provide useful guidelines for counseling women with preexisting renal insufficiency about their prospects for a successful pregnancy and the effect of pregnancy on their underlying disease.

APPENDIX

The following persons also participated in the study: P. Jungers, Hôpital Necker, Paris; A. Johenning and M.D. Lindheimer, University of Chicago, Chicago; E. Imbasciati, Ospedale Maggiore, Lodi, Italy, for the Study Group on Renal Disease and Pregnancy;

S. Abe, Keio University School of Medicine, Tokyo, Japan; and S.N. Sturgiss and J.M. Davison, Princess Mary Maternity Hospital, Newcastle upon Tyne, United Kingdom.

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CORRECTION

Outcome of Pregnancy in Women with Moderate or Severe Renal Insufficiency

Outcome of Pregnancy in Women with Moderate or Severe Renal Insufficiency . On page 229, in lines 11 through 13 of the left-hand column, the comparison of the incidence of exacerbation of hypertension during pregnancy in the group with severe renal insufficiency and in the group with only moderate renal insufficiency should have been 38 percent versus 24 percent, not 62 percent versus 76 percent, as printed.