

This PDF is a selection from an out-of-print volume from the National Bureau of Economic Research

Volume Title: Economic Aspects of Health

Volume Author/Editor: Victor R. Fuchs, editor

Volume Publisher: University of Chicago Press

Volume ISBN: 0-226-26785-7

Volume URL: <http://www.nber.org/books/fuch82-1>

Publication Date: 1982

Chapter Title: Prenatal Medical Care and Infant Mortality

Chapter Author: Jeffrey E. Harris

Chapter URL: <http://www.nber.org/chapters/c6544>

Chapter pages in book: (p. 13 - 52)

PART

1 Determinants of Health

This Page Intentionally Left Blank

1 Prenatal Medical Care and Infant Mortality

Jeffrey E. Harris, M.D.

In 1969, forty-three percent of expectant black mothers and seventy-two percent of expectant white mothers began prenatal medical care during their first trimester of pregnancy. By 1977, fifty-nine percent of black mothers and seventy seven percent of white mothers had begun prenatal care during their first trimester. Only 3% of expectant black mothers and 1% of expectant white mothers currently receive no medical attention before the onset of labor (U.S. National Center for Health Statistics 1978, Table A; 1980a, Table 20).

My main purpose in this paper is to inquire: How can we determine whether prenatal medical care has favorably influenced the outcome of pregnancy?

The role of prenatal care has been the subject of serious dispute in the obstetric and public health literature for nearly four decades. This dispute has been fomented in great part by the nonexperimental nature of the evidence. Virtually all studies of prenatal care analyze cross-section data on the uncontrolled experience of thousands of women and their pregnancies. The subjects under study are therefore self-selected. There are no randomized treatments. Possible confounding variables cannot be eliminated. Nor do the data reveal how the subjects actually made use of the medical services. This paper investigates in detail what inferences can and cannot be legitimately drawn from this type of evidence.

Jeffrey E. Harris, M.D., is at the Massachusetts Institute of Technology and the Massachusetts General Hospital. This research was supported in part by a grant from the Whitaker Health Sciences Fund. The author is currently a recipient of PHS Research Career Development Award DA-00072. Previously unpublished data presented in this paper were made available by the Office of Health Planning and Statistics, Massachusetts Department of Public Health. Valuable criticisms by W. DuMouchel, H. Farber, V. Fuchs, A. Garber, M. Grossman, J. Hausman, and D. McFadden are acknowledged. The opinions expressed in this paper are the author's sole responsibility.

Prenatal care is defined here as medical attention received from the time of conception up to, but not including, labor and delivery. The analysis of perinatal care, received during labor and delivery and in the neonatal period, is another matter.

This paper does not produce a definitive benefit-cost analysis of prenatal medical care. Nor does it pass final judgment on other determinants of pregnancy outcome. My main goal is a clear statement of the issues underlying this famous controversy. Along the way, some new methodological tools and new lines of investigation are suggested.

Prenatal Care and Infant Mortality: an Initial Examination

During 1975–76, there were 138,943 recorded live births in Massachusetts. Among live births, 1,229 infants (8.8 per 1000) died within 28 days of birth. Also reported were 1,335 fetal deaths. (In Massachusetts, reporting of fetal deaths beyond twenty weeks' gestation is legally required.)

The following analysis is based upon information encoded in the individual birth certificates and, where applicable, matched death certificates of these cases. Infant deaths beyond the neonatal period (28 days of age) were not analyzed. Similar cross-section data bases on linked birth and death records have been studied by Chase (1974, 1977), Chase et al. (1973), Cunningham et al. (1976), Gortmaker (1979), Kane (1964), Kessner et al. (1973), Kleinman et al. (1978), Lewit (1977), Mellin (1972), Morris et al. (1975), Niswander and Gordon (1972), Pakter and Nelson (1974), Russell and Burke (1975), Shah and Abbey (1971), Shwartz (1962), Shwartz and Vinyard (1965), Slesinger and Travis (1975), Susser et al. (1972), Taylor (1970), Terris and Glasser (1974), Terris and Gold (1969), Williams (1975), and others.

Figure 1.1 depicts the crude relation between the total number of prenatal visits reported during pregnancy and the probability of neonatal death among all live births. Intervals of one standard error are shown around each point estimate of the neonatal mortality rate. The point at the extreme right of the figure, corresponding to “?” on the abscissa, represents the neonatal death rate among women with an unknown number of prenatal visits. Although fetal deaths were excluded from the results shown in Figure 1.1, their inclusion does not alter the qualitative relationship displayed here.

On its face, Figure 1.1 suggests that the quantity of prenatal care, as measured by the reported number of prenatal visits, has a substantial effect on pregnancy outcome. Beyond an apparent minimum of three prenatal visits, the neonatal mortality rate rapidly declines. After approximately ten visits, however, the returns to prenatal care apparently diminish. Although the neonatal mortality rates beyond twenty visits are

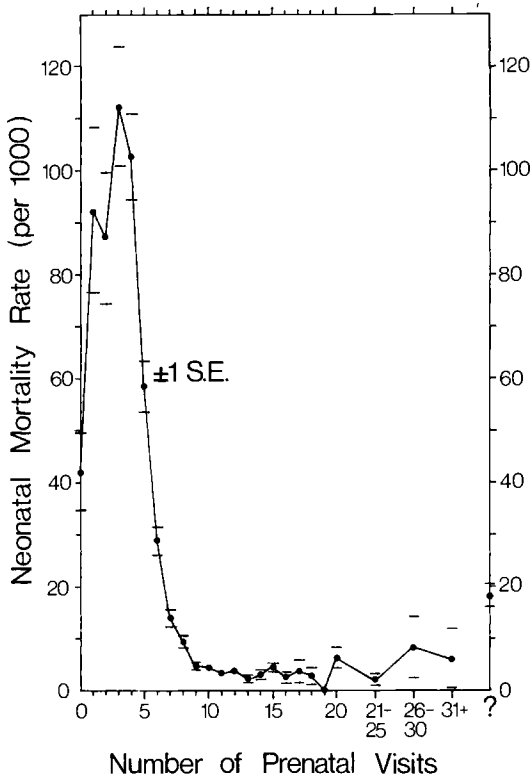


Fig. 1.1 Relation between number of prenatal visits and neonatal mortality rate per 1000 live births (Massachusetts, all races, 1975–76).

not very precise, the data suggest absolute decreasing returns to prenatal care.

In fact, not one of these conclusions is justified by the data. To see this, we must ask why some women report three prenatal visits, others report ten visits, and still others report twenty-five visits.

It is established obstetric practice in Massachusetts, and throughout the United States, for expectant mothers to follow a recommended schedule of visits—every four weeks for the first 28 weeks of pregnancy, every two weeks thereafter until the 36th week, then weekly until full term, and perhaps twice weekly if the baby is past due (U.S. National Center for Health Statistics, 1978; American College of Obstetricians and Gynecologists, 1974). The typical woman who recognizes her pregnancy at 6–8 weeks' gestation, follows the visit schedule recommended by her doctor, and delivers at 38–42 weeks will report about ten to fifteen visits. In fact, over two-thirds of the women in the sample reported a quantity of care in this range.

Those women reporting a quantity of prenatal care outside this range, however, constitute a much less homogeneous group. One important subpopulation of pregnant women, apparently concentrated among lower income and poorly educated groups, and among unmarried mothers and those of high parity, do not adhere to standard prenatal medical practice (Chase et al. 1973; Gortmaker 1979; Lewit 1977; U.S. National Center for Health Statistics 1978). These women may seek medical care only if they perceive some complication late in the course of pregnancy. Those mothers with no prenatal care may therefore represent a population with many fewer complications than those women with even one or two visits. In the range below ten prenatal visits, there is still another subpopulation of women who did follow the standard prenatal care schedule. As a result of placental insufficiency, infection, congenital anomalies, or other causes, their pregnancies—and therefore the course of prenatal care—were terminated prematurely. At the other end of the spectrum, women with previously established high risks (e.g., diabetes, rheumatic heart disease) or with increased risk detected during pregnancy (e.g., preeclampsia, intrapartum bleeding) seek care earlier and make more frequent prenatal visits. Among women with a large number of prenatal visits, however, there is also a group who were frequently monitored solely because they remained pregnant beyond their expected date of delivery.

Finally, 5.5% of live births of black women and 1.7% of live births of white women were recorded to have an unknown number of prenatal visits. Among those records with missing data on prenatal care, but with completed information on other characteristics, there was a disproportionate fraction of out-of-wedlock, higher order births, and teenage pregnancies. Since prenatal care information on live birth certificates is typically completed by hospital staff personnel and not by the mother, missing data are more likely to occur when the patient has no prior hospital record of the pregnancy (U.S. National Center for Health Statistics 1978, Technical Appendix). The unknown prenatal care category is therefore very likely to contain a disproportionate fraction of women with no prenatal care.

These facts seriously complicate the interpretation of Figure 1.1. Since early termination of pregnancy interrupts the normal course of prenatal care, the marked decline in neonatal mortality in the range of three to ten visits could mean that the extent of care is merely an indicator of fetal maturity. If the group with an unknown number of visits is composed primarily of women with no care, then the observed neonatal mortality for women who reported no care may be substantially overstated. Aside from this possible bias, the elevated neonatal mortality rate of the no care group could reflect poor socioeconomic status, illegitimacy, or other factors correlated with the demand for care. The increasing mortality rate

in the range from zero to three visits, moreover, could reflect the higher complication rate among mothers who seek care only late in gestation. The possibility of increased mortality in the range beyond fifteen visits could merely reflect the higher medical risks of some mothers in that group.

Prenatal Care and the Duration of Pregnancy

To unravel these difficulties, I first examine in detail the relation between prenatal care and gestational age.

Many investigators (e.g., Eastman 1947; Oppenheimer 1961; Pakter et al. 1961) have noted that mothers with little or no prenatal care have substantially higher rates of preterm delivery. It has not gone unnoticed, however, that shortened gestation may interrupt the standard prenatal care schedule, and therefore induce a spurious correlation between prematurity and the total number of visits (Drillien 1957; Hellman 1953; Kane 1964; Shwartz 1962; Shwartz and Vinyard 1965; Terris and Glasser 1974; Terris and Gold 1969). Terris and Glasser (1974) recognized that this spurious correlation also applied to the time of initiation of care, since the interval to the first prenatal visit might just as well be truncated by early termination of pregnancy. Statistically adjusted measures of prenatal care, such as the average number of visits per week of gestation, were similarly inappropriate because the frequency of visits on the standard schedule increased later in pregnancy.

Despite repeated recognition, this paradox remains unresolved. Studies of the effect of prenatal care on other dimensions of pregnancy outcome (such as birth weight and mortality) have merely capitulated that the quantity of prenatal care and the duration of pregnancy were confounded variables. Hence, measurement of prenatal care was somehow to be adjusted for gestational age. Kane (1964), for example, excluded cases delivered prior to 38 weeks, while Chase et al. (1973, Table 3.9) excluded cases delivered prior to 36 weeks. Lewit (1977), and Russell and Burke (1975), included gestational age as an additional explanatory variable in ordinary least squares regressions of prenatal care on birth weight and infant death. (The fact that their linear specifications failed to correct for the nonlinearly increasing frequency of visits at the end of pregnancy was overlooked.) Wells et al. (1958) similarly adjusted for length of gestation in an analysis of covariance of prenatal care and perinatal death. The frequently cited Institute of Medicine study of New York City births in 1968 (Kessner et al. 1973, p. 59) constructed an a priori index of prenatal care adequacy, determined by the number of prenatal visits adjusted for gestational age. A given schedule was deemed "adequate" in this study only if the mother had private obstetrical care. The same adequacy index, exclusive of the private obstetrical care re-

quirement, was subsequently used by Gortmaker (1979) in a multiple contingency table analysis. As in the Institute of Medicine study, this author assigned all observations with unknown care to the "inadequate" category (Gortmaker 1979, Appendix A).

None of these studies has had any bearing on the causal relation between prenatal care and the duration of pregnancy. The possibilities that prenatal attention could suppress early labor, or identify overdue mothers requiring induced labor, or screen out fetuses that are subsequently ill-fated, remain untested.

Prenatal Care and Premature Delivery as Competing Risks

At any time during gestation, a woman is subject to some instantaneous risk of termination of pregnancy. This risk of termination will depend upon the duration of pregnancy thus far, as well as other maternal and infant characteristics, including the presence of prenatal medical attention. The timing of prenatal care also represents of type of risk. That is, at any time during gestation, there is some instantaneous probability that a woman will make a prenatal visit, and in particular that a woman thus far without care will initiate prenatal care. This risk of visiting the doctor will depend in turn upon various maternal and infant characteristics.

Our problem is that the risk of visiting the doctor and the risk of termination of pregnancy are in competition. Among women who received no prenatal care, the termination of pregnancy occurred, in effect, before the initial visit could take place. Among those who did receive care, the initial visit occurred before the termination of pregnancy. In this context, we may inquire whether the initiation of prenatal care (when it does occur prior to termination of pregnancy) modifies the subsequent risk of pregnancy termination.

Let $\lambda_V(v)$ and $\lambda_T(t)$, respectively, be the instantaneous risks (or hazard rates) for making an initial visit and for termination of pregnancy. The rate $\lambda_V(v)$ is the probability that prenatal care is initiated in the short interval $(v, v + dv)$, given that no care has been received prior to time v . The rate $\lambda_T(t)$ is the probability that pregnancy will terminate in the short interval $(t, t + dt)$, given that gestation has lasted until time t . The concept underlying the hazard $\lambda_V(v)$ has been mentioned only once in the literature (Terris and Glasser 1974). The hazard $\lambda_T(t)$ is the more familiar gestational age-specific force of exit in a fetal life table (Bakketeig et al. 1978; Mellin 1962; Taylor 1970).

Consider the event that pregnancy terminates without prenatal care at time t . (Time is measured from the point of conception.) Provided that the risks of initiation of care and termination of pregnancy are initially independent, the probability of this event is

$$(1) \quad \lambda_T(t) \exp\left[-\int_0^t \lambda_T(s) ds\right] \times \exp\left[-\int_0^t \lambda_V(s) ds\right]$$

The first expression in (1) is the probability that pregnancy terminated at time t . The second expression is the probability that prenatal care was not sought in the interval $[0, t]$. (See David and Moeschberger 1978; Lancaster 1979).

Let $\lambda_{TV}(t|v)$ be the risk of termination of pregnancy at time t , given that prenatal care was initiated at time $v \leq t$. The interdependence of hazard rates captured by this notation is a special case of the more general hypothesis that the number and timing of each prenatal visit affect the risk of termination of pregnancy. Now consider the event that care is initiated at time v and pregnancy subsequently terminates at time t . The probability of this event is

$$(2) \quad \lambda_V(v) \exp\left[-\int_0^v \lambda_V(s) ds\right] \exp\left[-\int_0^v \lambda_T(s) ds\right] \\ \times \lambda_{TV}(t|v) \exp\left[-\int_v^t \lambda_{TV}(s|v) ds\right]$$

The first expression in (2) is the probability that prenatal care is initiated at time v and pregnancy did not terminate in the interval $[0, v]$. The second expression is the probability of termination of pregnancy at time t given that prenatal care was initiated at time v and that the pregnancy was intact at time v .

The hypothesis that the presence of care affects the subsequent rate of termination of pregnancy means that $\lambda_{TV}(t|v) \neq \lambda_T(t)$. When $\lambda_{TV} < \lambda_T$, prenatal care slows down the rate of termination of pregnancy; that is, it prevents prematurity. When $\lambda_{TV} > \lambda_V$, prenatal care accelerates the termination of pregnancy.

An Illustrative Test

Figure 1.2 depicts the frequency distribution of length of gestation among mothers with and without prenatal care in Massachusetts during 1975–76. The results in Figure 1.2 confirm the association between prenatal care and full term gestation: 29% of mothers with no prenatal care, as opposed to 5% of mothers with some prenatal care, had gestations less than 36 weeks' duration. (Although Massachusetts requires reporting only of pregnancies of 20 weeks' duration, a small fraction of the sample included pregnancies of shorter duration.)

To construct a statistical test of the hypothesis that prenatal care affects the duration of gestation, I need to impose some additional restrictions on the data and the model. First, I exclude cases with unknown prenatal care and unknown gestational age. (These are omitted in Figure 1.2 and constitute 4% of the entire sample.) In this illustration, the problem of

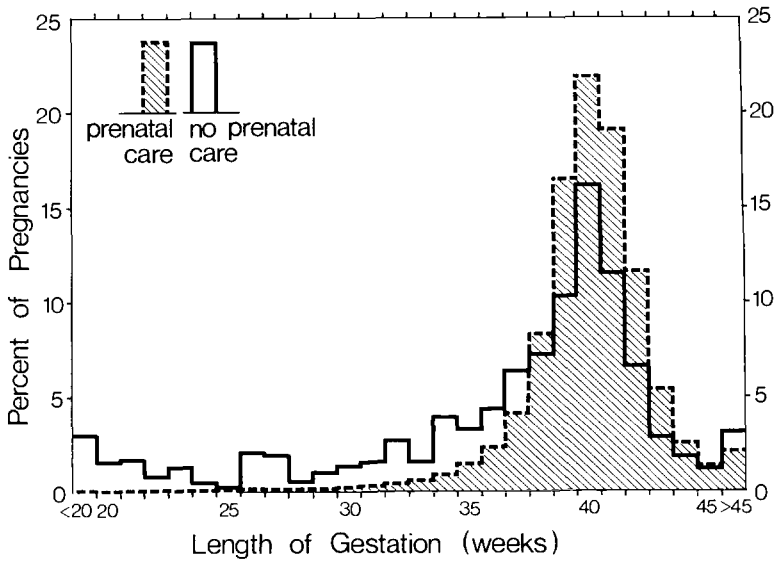


Fig. 1.2 Frequency distribution of length of gestation among women with and without prenatal care. Live births and fetal deaths (Massachusetts, all races, 1975–76).

nonrandomly missing observations is therefore not addressed. Second, I consider both live births and fetal deaths. Inclusion of fetal deaths admits the possibility that prenatal care prevents spontaneous abortion or other causes of premature delivery resulting in death during labor. Third, I examine only a subsample of 6,736 black women’s pregnancies. The alternative of analyzing the pregnancies of women of all races, with indicator variables for each race, does not appear warranted at this stage. The effects of prenatal care among black mothers may differ considerably from the corresponding effects among other races.

I further restrict the model to the proportional hazards form

$$(3) \quad \lambda_{TV}(t|v) = (1 + \alpha)\lambda_T(t)$$

where $\alpha > -1$ is a constant, independent of t and v . Although the instantaneous effect of prenatal care on the rate of termination of pregnancy is assumed to be time-independent, the total effect of prenatal care on the duration of pregnancy will nevertheless depend upon the time of initiation of care.

To complete the statistical model, I need to specify how the hazards λ_T and λ_V depend upon time and other observed characteristics. Let $\underline{X} = (X_1, \dots, X_K)$ be a vector of explanatory variables. I assume that λ_T and λ_V depend upon t and \underline{X} in the following way:

$$(4) \quad \lambda_T(t|\underline{X}) = (\rho_T \omega_T) (\rho_T t)^{\omega_T - 1} \prod_{k=1}^K (1 + \theta_{Tk} X_k)$$

$$\lambda_V(v|\underline{X}) = (\rho_V \omega_V) (\rho_V v)^{\omega_V - 1} \prod_{k=1}^K (1 + \theta_{Vk} X_k)$$

The expressions $(\rho_T \omega_T) (\rho_T t)^{\omega_T - 1}$ and $(\rho_V \omega_V) (\rho_V v)^{\omega_V - 1}$ are Weibull hazard functions. The parameters ω_T and ω_V , in particular, incorporate the possibility that the rates of termination of pregnancy and initiation of prenatal care are time dependent. The hazard rate increases monotonically for $\omega > 1$, decreases monotonically for $\omega < 1$, and remains constant for $\omega = 1$. In the expressions $\prod_k (1 + \theta_{Tk} X_k)$ and $\prod_k (1 + \theta_{Vk} X_k)$, each parameter θ corresponds to the incremental effect of a given explanatory variable on one of the hazard rates. Each multiplicand $(1 + \theta X)$ represents the contribution of a specific explanatory variable to the proportional risks of termination of pregnancy and initiation of care. Under the restrictions (4), these proportional risks are assumed to be independent of gestational age. Similarly, under the restriction (3), the expression $(1 + \alpha)$ represents the contribution of prenatal care to the relative risk of termination of pregnancy.

Suppose that we have independent observations $\{t_i, \underline{X}_i: i = 1, \dots, N\}$ on the durations of pregnancy and other explanatory variables for mothers with no prenatal care, and independent observations $\{t_j, v_j, \underline{X}_j: j = 1, \dots, M\}$ on the durations of pregnancy, times of initiation of care and other explanatory variables among mothers with prenatal care. If the data $\{t_i, t_j, v_j\}$ are observed in continuous time, then the joint likelihood of these $N + M$ observations is

$$(5) \quad L^1 = \prod_{i=1}^N \lambda_T(t_i|\underline{X}_i) \exp[-\Lambda_T(t_i|\underline{X}_i)] \exp[-\Lambda_V(t_i|\underline{X}_i)]$$

$$\times \prod_{j=1}^M \lambda_V(v_j|\underline{X}_j) \exp[-\Lambda_V(v_j|\underline{X}_j)] (1 + \alpha) \lambda_T(t_j|\underline{X}_j) \exp[-\Lambda_{TV}(t_j, v_j|\underline{X}_j)]$$

where $\lambda_T(t|\underline{X})$ and $\lambda_V(v|\underline{X})$ are defined in (4), and where $\Lambda_T(t|\underline{X}) = (\rho_T \omega_T)^{\omega_T} \prod_k (1 + \theta_{Tk} X_k)$, and $\Lambda_V(v|\underline{X}) = (\rho_V \omega_V)^{\omega_V} \prod_k (1 + \theta_{Vk} X_k)$, and $\Lambda_{TV}(t, v|\underline{X}) = (1 + \alpha) \Lambda_T(t|\underline{X}) - \alpha \Lambda_T(v|\underline{X})$. This likelihood function, which I have superscripted with the numeral "1" to distinguish it from others used below, can be rewritten in the form

$$(6) \quad L^1 = \prod_{i=1}^N \lambda_T(t_i|\underline{X}_i) \exp[-\Lambda_T(t_i|\underline{X}_i)] (1 + \alpha)^M \prod_{j=1}^M \lambda_T(t_j|\underline{X}_j) \exp[-\Lambda_{TV}(t_j, v_j|\underline{X}_j)]$$

$$\times \prod_{i=1}^N \exp[-\Lambda_V(t_i|\underline{X}_i)] \prod_{j=1}^M \lambda_V(v_j|\underline{X}_j) \exp[-\Lambda_V(v_j|\underline{X}_j)]$$

$$= L_T^1 \times L_V^1$$

where L_T^I and L_V^I are multiplicatively separable in the parameters $\{\alpha, \omega_T, \rho_T, \theta_{Tk}\}$ and $\{\omega_V, \rho_V, \theta_{Vk}\}$ respectively. Hence, the maximum likelihood estimates of these two sets of parameters can be obtained separately without bias.

Table 1.1 displays the main characteristics of the subsample of black women's pregnancies. There were 82 neonatal deaths (12.3 per 1000 live births) and 43 fetal deaths. Among observations excluded from this sample because of missing information on birth weight, gestational age, initiation of prenatal care, or other explanatory variables, there were 29 additional neonatal deaths and 38 additional fetal deaths.

Table 1.2 displays the maximum likelihood estimates of the parameters of L_T^I and L_V^I in (6). The estimate of the parameter α is -0.293 ; that is, prenatal care reduces the risk of termination of pregnancy by 29.3%. The estimate of ω_T far exceeds 1; the risk of termination of pregnancy rises very rapidly with increasing gestational age. For the Weibull hazard function (4), the ratio of the mean gestational age of black mothers without prenatal care to the mean gestational age of black mothers with care throughout pregnancy is $(1 + \hat{\alpha})^{1/\hat{\omega}_T} = 0.978$ (approximate standard error 0.007). That is, for a 40-week pregnancy, the absence of care

Table 1.1 Sample Characteristics of Pregnancies
(6,736 Black Women, Massachusetts, 1975-76)

Number of neonatal deaths	82	
Number of fetal deaths	43	
Percent initiated care in first trimester	74.5	
Percent initiated care in second trimester	20.9	
Percent initiated care in third trimester	3.6	
Percent with prior perinatal loss ^a	17.3	
Percent primagravida	39.7	
Percent recorded illegitimate	48.8	
Percent aged over 30 years	12.6	
Percent aged under 20 years	25.3	
Mean gestational age (weeks)	39.2	(s.d. 3.12)
Mean duration of prenatal care (weeks) ^b	28.5	(s.d. 7.90)
Mean birth weight (grams)	3123	(s.d. 619.)
Mean attained education (years)	11.6	(s.d. 0.22)
Mean annual volume of deliveries at hospital of birth (thousands)	3.1	(s.d. 2.09)

^aIncludes prior neonatal death or prior fetal death of at least 20 weeks' duration.

^bData on initiation of prenatal care was recorded by month of pregnancy. Calculation of weeks of care assumed that prenatal care was initiated at the midpoint of the recorded month of pregnancy.

(s.d. = standard deviation)

Table 1.2 Maximum Likelihood Estimates of the Effect of Prenatal Care on the Rate of Termination of Pregnancy: Model I. (6,736 Black Women, Massachusetts, 1975–76.)

		Parameter Estimates	
		L_T^1	L_V^1
Effect of prenatal care	α	-0.293 (0.075)	—
Weibull hazard parameters	ω	15.631 (0.114)	1.626 (0.053)
	ρ	0.026 (0.0001)	0.060 (0.004)
Parameters of Explanatory Variables			
Years of education	θ_1	-0.013 (0.004)	0.086 (0.020)
Years of age over 30	θ_2	-0.003 (0.006)	-0.166 (0.006)
Years of age under 20	θ_3	0.005 (0.010)	-0.076 (0.008)
Illegitimacy	θ_4	-0.071 (0.022)	-0.260 (0.017)
Prior perinatal loss	θ_5	0.091 (0.031)	-0.037 (0.029)
Primagravida	θ_6	-0.001 (0.028)	0.254 (0.032)
Log likelihood		-16734.3	-21371.9

Standard errors in parentheses.

reduces the mean gestational age by about 0.88 weeks (approximate standard error 0.28).

Table 1.2 also reveals statistically significant effects of attained education and prior fetal loss on the hazard of pregnancy termination. For black women with sixteen years of education, the risk of pregnancy termination is reduced by an estimated 12%, relative to black women with eight years of education (i.e., $(1 + 16\hat{\theta}_{T1}) \div (1 + 8\hat{\theta}_{T1}) = 0.88$, standard error 0.04). The interpretation of the statistically significant effect of illegitimacy (X_4) is more complicated. Since potentially confounding factors such as teenage pregnancy (X_3), first pregnancy (X_6), and reduced education (X_1) are held constant, the estimated reduced risk of early

termination of pregnancy for illegitimate births may reflect the experience of relatively older black women of higher parity. I shall return to this puzzling observation later.

Table 1.2 shows that the hazard rate for the initiation of prenatal care also increases with gestational age (i.e., $\hat{\omega}_V = 1.63 > 1$.) The rate of initiation of care for black women with sixteen years of attained education is 41% greater than the rate of initiation of care for black women with eight years of attained education (i.e., $(1 + 16\hat{\theta}_{V1}) \div (1 + 8\hat{\theta}_{V1}) = 1.41$, standard error 0.06). This estimate corresponds to a 19% reduction in the mean time to initiate care (standard error 2.1%). For an expectant black mother who seeks care at 12 weeks' gestation, this represents an average reduction of 2.3 weeks in the mean time to the first visit (standard error 0.26). The combined effect of illegitimacy, advanced maternal age, and previous pregnancies is substantial. A 35-year-old multiparous woman delivering an illegitimate child has a rate of initiation of care one-tenth that of a primagravida in her 20s delivering a legitimate child (i.e., $(1 + 5\hat{\theta}_{V2}) \cdot (1 + \hat{\theta}_{V4}) \div (1 + \hat{\theta}_{V6}) = 0.1$). This corresponds to an estimated fourfold increase in the mean time to initiation of care (standard error 0.23).

These estimates were derived from a selected data base in the context of a specific parametric model. The conclusion that prenatal care reduces preterm delivery may not withstand alternative data bases, or a formulation other than the proportional hazards model of (3) and (4). It is noteworthy, however, that the estimate and standard error of the parameter α changed only minimally when I included other explanatory variables, such as type of care (private versus ward), the percentage of rental housing, the median income in the census tract corresponding to the mother's residence, or when I tried alternative specifications of the effect of maternal education and age. The results did not change substantially when fetal deaths were excluded from the sample. Although I assumed that the week of initiation of care corresponded to the midpoint of the reported month of initiation of care, the use of a more complicated likelihood function that incorporated the interval characteristics of these data also did not substantially alter the results. Finally, when I included observations with unknown care in the analysis, assuming that these women in fact received no care, the estimate of α was reduced in absolute value to -0.20 .

Unobserved Characteristics and Fetal Selection

My analysis of the relation between prenatal care and the duration of pregnancy has thus far overlooked one serious problem of interpretation. Figure 1.3 depicts the relation between the month of initiation of care and the proportion of births of less than 36 weeks' gestation for white and

black mothers. (Intervals of one standard error are shown for blacks. The corresponding standard errors for whites were considerably smaller, and are omitted for clarity.) Figure 1.3 shows that the increasing relation between late care and preterm delivery is interrupted during the third trimester. Since the 36th week of gestation occurs during the ninth calendar month, one can assume that this finding is not simply an artifact of the 36-week cutoff used in the figure.

In any cohort of pregnant women, the initial fetal population is likely to be extremely heterogeneous in its health characteristics. If this heterogeneity is reflected in their risks of termination of pregnancy—with the least fit infants having the highest hazard rates—then the phenomenon of fetal loss can play a powerful selective role. In comparison to the fetal population at the time of conception, those infants that have remained in utero up to the third trimester will necessarily contain a smaller fraction of ill-fated fetuses. One distinctive characteristic of mothers who initiate care in the third trimester is that their infants have remained in utero just that long. Hence, for no reason other than natural selection, late initiators of care may have infants with lower rates of pregnancy termination than earlier initiators of care. But this selection effect need not apply to mothers without care, whose infants may have been delivered at any time during gestation. These phenomena are exactly reflected in Figure 1.3.

If we could ascertain all the relevant determinants of variation in the risks of termination of pregnancy, then in principle we could fully account for this selection phenomenon. The difficulty with this solution is not merely its cost. Even if we could assemble detailed data on fetal ultrasound measurements, urinary estriol levels, maternal weight gain, and other factors for a large cross-section of women, there might still be substantial unobserved variation in fetal robustness. These unobserved characteristics would then be subject to selection. The inverse relation between late care and the duration of pregnancy might not be eliminated by conditioning on the observable characteristics.

Moreover, if the phenomenon of fetal loss selects out the least fit infants, then any factor that slows the rate of termination of pregnancy will also retard this selective process. If prenatal care, in particular, reduces the risk of termination of pregnancy, then at any given week of gestation, those mothers who had early care will tend to have a higher proportion of less fit infants. This possibility is also consistent with the data in Figure 1.3.

The problem is further complicated if the mothers under study could ascertain those health characteristics of their infants that are not revealed to the analyst in the data. Mothers who perceive their babies to be less fit, or potentially less fit, may initiate care earlier, while those with uneventful pregnancies may delay care. This hypothesis would account not only

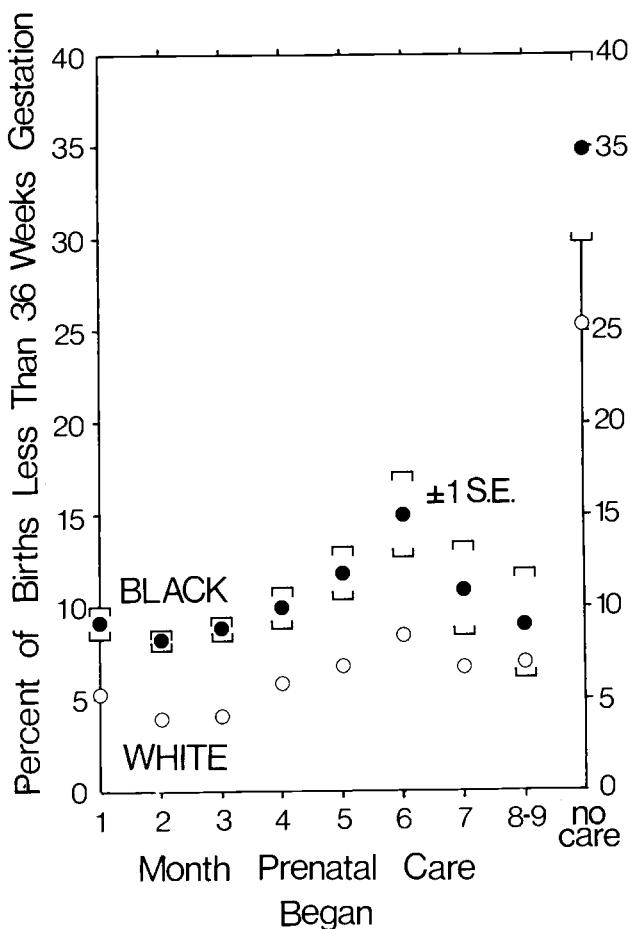


Fig. 1.3 Relation between month of initiation of prenatal care and percentage of births less than thirty-six weeks' gestation among white and black women. Live births and fetal deaths (Massachusetts, 1975-76).

for the lower proportion of preterm deliveries among late initiators of care, but also for the higher proportion of preterm deliveries among mothers who initiated care in the first month.

The data almost exclusively cover pregnancies of at least 20 weeks' duration. Hence, the cohort actually observed is likely to be more homogeneous than the original fetal cohort at the time of conception. The selective effect of fetal loss may therefore be less significant. Data from more complete fetal life table analyses (Bakketeig et al. 1978;

Mellin 1962; Taylor 1970), extending back to the weeks immediately after conception, reveal an initial period of relatively high hazard rates of pregnancy termination. As the ill-fated fetuses are progressively eliminated from the cohort, the overall hazard rate gradually falls. After approximately 20 weeks, the hazard rate then begins to rise. This increase in the hazard rate, however, does not imply that only robust infants remain in utero beyond 20 weeks. There is still likely to be substantial remaining variation in fetal characteristics up to and including the 40th week of pregnancy. Nevertheless, the censoring of early fetal deaths in our sample could bias estimates of the effect of prenatal care and other explanatory characteristics. If prenatal care prevented early fetal loss, for example, then it could extend an otherwise short pregnancy beyond the 20-week observational cutoff. On the other hand, the early medical attention could permit some women and their physicians to screen out and terminate an eventually ill-fated pregnancy before 20 weeks.

It is not at all clear how these complicated structural relationships can be identified with the available cross-section data. One possible strategy is to specify a model of the fetal selection process, and then to investigate how that model affects our inferences about the effects of prenatal care and other explanatory variables.

Let ε be a scalar index of fetal “defectiveness”, whose value is restricted to be positive. Infants with low values of ε are more robust than infants with high values of ε . Although fetal defectiveness cannot be directly observed, it is assumed to affect the hazard rate for pregnancy termination. I denote this dependence by $\lambda_T(t|\underline{X}, \varepsilon)$, retaining the specification that $\lambda_{TV} = (1 + \alpha)\lambda_T$. For a given cohort of pregnant women, ε initially has probability density $f(\varepsilon)$. Now let $G_{TV}(t|\nu, \underline{X}, \varepsilon)$ be the probability that a pregnancy of defectiveness ε , with observed characteristics \underline{X} and time of initiation of care ν , survives at least to gestational age t . Then, by Bayes Rule, the probability density of ε among those infants with characteristics (ν, \underline{X}) who remain in utero at least to age t is

$$(7) \quad f(\varepsilon|t, \nu, \underline{X}) = \frac{G_{TV}(t|\nu, \underline{X}, \varepsilon)f(\varepsilon)}{\int_0^{\infty} G_{TV}(t|\nu, \underline{X}, \zeta)f(\zeta) d\zeta}$$

A similar formula applies to mothers with no prenatal care, where G_{TV} is replaced by G_T , the corresponding probability of survival.

If λ_T is an increasing function of ε , then for a given (ν, \underline{X}) , both the mean and variance of $(\varepsilon|t, \nu, \underline{X})$ decline with increasing t . That is, as a result of fetal selection, those infants remaining in utero are on average less defective and more homogeneous as gestation advances.

In keeping with the proportional hazards specification, I now let $\lambda_T(t|\underline{X}, \varepsilon) = \lambda_T(t|\underline{X})\varepsilon$. Moreover, I let ε have a gamma density with mean 1 and variance $1/h$. That is,

$$(8) \quad f(\varepsilon) \propto \exp[-h\varepsilon] \varepsilon^{h-1}$$

Then for pregnancies with defectiveness ε and characteristics (v, \underline{X}) ,

$$(9) \quad G_{TV}(t|v, \underline{X}, \varepsilon) = \exp[-\Lambda_{TV}(t, v|\underline{X})\varepsilon]$$

where, in the case of mothers with no care, Λ_{TV} is replaced by Λ_T . From (7), (8), and (9), we obtain

$$(10) \quad f(\varepsilon|t, v, \underline{X}) \propto \exp[-(h + \Lambda_{TV}(t, v|\underline{X}))\varepsilon] \varepsilon^{h-1}$$

where, again, in the case of mothers with no care, Λ_{TV} is replaced by Λ_T . The conditional density of ε is therefore also gamma, but with mean $h/(h + \Lambda_{TV}) < 1$ and variance $h/(h + \Lambda_{TV})^2 < 1/h$. Since Λ_{TV} is an increasing function of t , the mean and variance of ε decline with gestational age. Moreover, if $\alpha < 0$, then Λ_{TV} is an increasing function of v . That is, late care accelerates the process of selecting the least defective infants.

From (9), the probability that a woman will still be pregnant at t , given characteristics \underline{X} and initiation of care at v , is

$$(11) \quad G_{TV}(t|v, \underline{X}) = \int_0^\infty G_{TV}(t|v, \underline{X}, \varepsilon) f(\varepsilon) d\varepsilon \\ = \left[\frac{h}{h + \Lambda_{TV}(t, v|\underline{X})} \right]^h$$

The probability that a woman will deliver at t , given initiation of care and v and characteristics \underline{X} , is therefore

$$(12) \quad \left[-\frac{\partial}{\partial t} G_{TV}(t|v, \underline{X}) \right] \cdot G_{TV}(t|v, \underline{X}) \\ = (1 + \alpha) \lambda_T(t|\underline{X}) \left[\frac{h}{h + \Lambda_{TV}(t, v|\underline{X})} \right]^{h+1}$$

Now suppose that we have independent observations $[t_i, \underline{X}_i; i = 1, \dots, N]$ on the durations of pregnancy and characteristics of women with no care, and independent observations $[t_j, v_j, \underline{X}_j; j = 1, \dots, M]$ on the durations of pregnancy, the times of initiation of care, and the characteristics of women with prenatal care. If there are no unobserved determinants of the hazard rate λ_V for initiation of care, then the joint likelihood of these observations is $L_V^I \times L_T^{II}$, where

$$(13) \quad L_T^{II} = \prod_{i=1}^N \lambda_T(t_i|\underline{X}_i) \left[\frac{h}{h + \Lambda_T(t_i|\underline{X}_i)} \right]^{h+1} (1 + \alpha)^M \\ \times \prod_{j=1}^M \lambda_T(t_j|\underline{X}_j) \left[\frac{h}{h + \Lambda_{TV}(t_j, v_j|\underline{X}_j)} \right]^{h+1}$$

Maximum likelihood estimates of the parameters $\{\alpha, \rho_T, \omega_T, \theta_{Tk}, 1/h\}$ in L_T^{II} are presented in Table 1.3. The estimate of the variance of ϵ is significantly different from zero. The maximum value of $\log L_T^{\text{II}}$ is substantially greater than the corresponding maximum value of $\log L_T^{\text{I}}$ in Table 1.2. Strictly speaking, Model I is the limiting case of Model II for $1/h \downarrow 0$. Therefore, its parameters are not properly in the interior of the parameter space of Model II. But it is sufficient to note that a null hypothesis of any arbitrarily small value of $1/h$ will be rejected in favor of Model II, and that L_T^{II} is right hand continuous at $1/h = 0$. Hence, Model II represents a substantially better fit than Model I.

The maximum likelihood estimate of α in Table 1.3 is -0.351 , as compared to -0.293 in Table 1.2. That is, our previous failure to account for fetal selection in Model I resulted in a biased estimate of the effect of prenatal care. The magnitude of this bias, however, is not too large. For example, the ratio of the mean gestational age of black mothers without prenatal care to the mean gestational age of black mothers with care throughout pregnancy is $(1 + \hat{\alpha})^{1/\hat{\omega}_T} = 0.977$ (standard error 0.008). (Under our proportional hazards specification, this ratio is independent of ϵ .) That is, for a 40-week pregnancy, the absence of care reduces the mean gestational age by about 0.94 weeks (standard error 0.32). For the parameter estimates in Model I, the corresponding reduction was 0.88 weeks (standard error 0.28).

The maximum likelihood estimate of ω_T in Table 1.3 is significantly greater than that in Table 1.2. When we ignore fetal selection, the hazard rate appears to rise more slowly because the high- ϵ (less robust) fetuses are being progressively eliminated from the cohort (see also Lancaster 1979). Similarly, the estimate of θ_{T5} in Table 1.3 exceeds the corresponding estimate in Table 1.2. That is, fetal selection operates more effectively on mothers with a prior history of fetal loss, and therefore failure to account for fetal selection leads to underestimates of the impact of this risk factor.

The model of equations (7) through (13) applies to the omitted regressor ϵ in the determination of the hazard rate λ_T . But a completely analogous argument could be applied to the determination of the hazard rate λ_V . If we assume that $\lambda_V(v|X, \delta) = \lambda_V(v|X)\delta$, where δ is the unobserved characteristic, and if δ similarly has a gamma density at the onset of pregnancy, and if δ is distributed independently of ϵ , then we can derive a likelihood L_V^{II} in a manner analogous to that for L_T^{II} .

Maximum likelihood estimates of the analogous parameters for L_V^{II} are presented in the right-hand column of Table 1.3. Again, the log likelihood substantially exceeds that in Table 1.2, and the estimate of the variance of the observed regressor has a small standard error. The estimate of ω_V is similarly increased. Moreover, many of the estimates of

Table 1.3 Maximum Likelihood Estimates of the Effect of Prenatal Care on the Rate of Termination of Pregnancy: Model II, Incorporating Unobserved Regressors (6,736 Black Women, Massachusetts, 1975-76)

		Parameter Estimates	
		L_T^{II}	L_V^{II}
Effect of prenatal care	α	-0.351 (0.084)	—
Weibull hazard parameters	ω	18.217 (0.198)	2.094 (0.039)
	ρ	0.026 (0.0002)	0.072 (0.006)
Parameters of explanatory variables			
Years of education	θ_1	-0.012 (0.006)	0.115 (0.037)
Years of age over 30	θ_2	-0.004 (0.008)	-0.019 (0.008)
Years of age under 20	θ_3	0.012 (0.014)	-0.104 (0.008)
Illegitimacy	θ_4	-0.044 (0.032)	-0.305 (0.025)
Prior perinatal loss	θ_5	0.134 (0.048)	-0.027 (0.046)
Primagravida	θ_6	-0.028 (0.036)	0.313 (0.053)
Variance of unobserved regressor	$1/h$	0.207 (0.018)	0.427 (0.034)
Log likelihood		-16652.9	-21214.7

Standard errors in parentheses.

θ_{Vk} in Table 1.3 differ significantly from those in Table 1.2. For example, since improved education accelerates the rate of initiation of care, it will tend to eliminate high- δ mothers from the cohort, and thus reduce the average hazard rate for initiation of care. Hence, the estimates of the effect of attained education in Table 1.2 will be biased downward. This is confirmed in Table 1.3.

The most important limitation of this analysis is the assumption that the unobserved regressors ϵ and δ are independently distributed. This restrictive assumption does not admit the possibility that fetal and maternal health characteristics are correlated with prenatal care demand fac-

tors and in particular, that mothers' perception of the health of their pregnancy affects the rate of initiation of care. For example, the statistically significant negative estimate in Table 1.2 of the effect of illegitimacy status on the hazard rate of pregnancy termination is pulled toward zero but remains negative in Table 1.3. Among black women delivering children out of wedlock, especially those of high parity, some mothers may have very low risk pregnancies. Those who anticipate an uneventful pregnancy may also have much lower rates of initiation of care. If we do not take account of fetal selection, illegitimacy status appears to deter preterm delivery. The introduction of two independent sources of variation in the hazards λ_T and λ_V apparently eliminates some of this bias. But it does not fully incorporate the possibility that the underlying health of the pregnancy affects the demand for care.

One possible solution to this difficulty is to allow for interdependence of the omitted regressors ϵ and δ . In the instant case, this suggestion would require a joint distribution whose marginal densities are gamma. Although there is a class of such bivariate gamma distributions (Johnson and Kotz 1972), they do not appear to admit a correlation coefficient that can assume both positive and negative values. More important, my preliminary experiments with such bivariate densities suggested that the correlation coefficient between ϵ and δ and the parameter α could not be simultaneously identified. For the present competing risk model, it appears difficult if not impossible to ascertain both the effect of prenatal care on the subsequent risk of preterm delivery and the possible feedback effect of the underlying health of the pregnancy on the demand for care. A similar statistical predicament has been noted for analogous normal models with discrete endogenous variables (Schmidt 1981).

Prenatal Care and the Rate of Intrauterine Growth

I now focus on the relation between prenatal care and birth weight. Since the duration of pregnancy indirectly affects weight at birth, I concentrate on the effect of prenatal care on birth weight conditional upon gestational age.

Figure 1.4 shows the relation between gestational age and mean birth weight according to the trimester of initiation of care, among mothers of all races in Massachusetts during 1975–76. Both live births and fetal deaths are included. These data correspond to the empirical intrauterine growth curves of the obstetrical literature (Gruenwald 1966, 1974; Lubchenco 1975; Williams 1975).

Figure 1.4 appears to confirm the commonplace finding that prenatal care improves birth weight, conditional upon gestational age (Chase et al. 1973; Gortmaker 1979; Kessner et al. 1973; Lewit 1977; Russell and Burke 1975; Shah and Abbey 1971; U.S. National Center for Health

Statistics 1978). In the range from 39 to 42 weeks' gestation, mothers who initiated care in the first trimester have infants with mean birth weights 200–300 grams greater than mothers who received no care. The relation between the timing of care and birth weight follows a dose-response pattern.

The results in Figure 1.4 could merely reflect the confounding influence of such factors as education, socioeconomic status, and race, all of which could affect both the timing of care and birth weight. To eliminate this possibility, we must specify a model of the effect of care on birth weight, conditional upon these potentially confounding variables as well as upon gestational age. As in the previous sections, it is more appropriate to confine the analysis to a single race, rather than to employ an indicator variable for race in a study of the entire sample. Beyond that, however, the choice of an appropriate model is not clear.

One complicating factor is that the data of Figure 1.4 represent weight at birth among a cross-section of infants of different gestational ages, and not the intrauterine growth curve of any one infant during the course of pregnancy. If there is a systematic relation between the duration of gestation and the rate of intrauterine growth across infants, then the slopes of the empirical curves in Figure 1.4 are biased measures of the rate of intrauterine growth. Since the determinants of these variations in the risk of pregnancy termination or the rate of intrauterine growth may be difficult to observe, we must again confront the problem of fetal selection. This means that prenatal care and other explanatory factors will affect not only the intrauterine growth rate of a given infant, but also the distribution of these unobserved factors across infants. Unless we are prepared to make strong parametric assumptions, the net effect of these complicated interactions is not obvious.

In order to compare the effects of prenatal care on intrauterine growth rates with the previously discussed effects on the duration of pregnancy, I shall specify a relatively simple model. Let the rate of growth of fetal weight be a function of gestational age and other explanatory factors, including the extent of prenatal care. This function is assumed to take the form

$$(14) \quad \frac{dw}{dt} = \Phi(t, \underline{X}) \Psi(v) + v$$

where dw/dt is the growth rate of weight, t is gestational age, \underline{X} is a vector of explanatory variables, $\Psi(v)$ measures the proportional effect of prenatal care, and v is a stochastic error term. I further approximate $\Phi(t, \underline{X})$ by the polynomial

$$(15) \quad \Phi(t, \underline{X}) = \beta_1 + 2\beta_2 t + 3\beta_3 t^2 + \sum_{k=1}^K \eta_k X_k$$

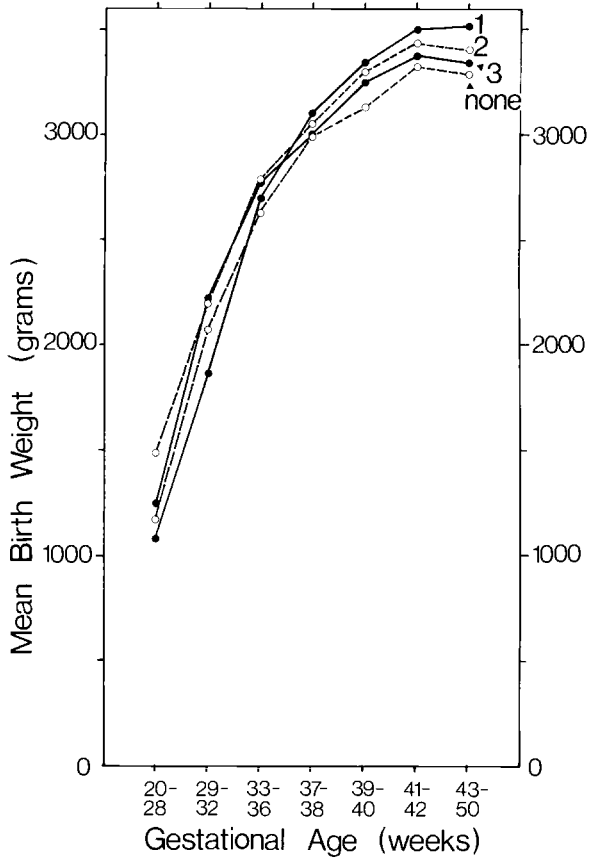


Fig. 1.4 Relation between gestational age and mean birthweight according to trimester of initiation of prenatal care. Live births and fetal deaths (Massachusetts, all races, 1975-76). Note: the gestational age intervals are of unequal duration.

In accord with the presentation of the data in Figure 1.4, I let

$$(16) \quad \Psi(v) = \prod_{i=1}^3 (1 + \gamma_i Y_i)$$

where $Y_i = 1$ if initiation of care occurs in trimester i , and zero otherwise. From (14), (15), and (16), and the initial condition $w(0) = 0$,

$$(17) \quad w = (\beta_1 t + \beta_2 t^2 + \beta_3 t^3 + \sum_{k=1}^K \eta_k X_k t) \prod_{i=1}^3 (1 + \gamma_i Y_i) + vt$$

In this parameterization, the parameters η_k measure the absolute contribution of each explanatory variable X_k to the rate of intrauterine

growth (in grams per week), while the parameters γ_i measure the proportional effect of prenatal care. Moreover, the variance of the stochastic error νt increases with gestational age. A simple regression model of absolute birth weight with homoskedastic errors would therefore attach too much statistical weight to the high gestational age infants.

Table 1.4 presents maximum likelihood estimates of the parameters of (17) under the assumption that the error component νt is normally distributed with mean zero and variance $\sigma^2 t^2$. The estimated effects of maternal age, legitimacy status, prior perinatal loss, and parity are statistically significant. Prenatal care appears to increase the rate of intrauterine growth by about 2% in comparison to no care. But the estimated effect is statistically insignificant (at the 5% level). Moreover, there is no

Table 1.4 Effect of Trimester of Initiation of Care on Rate of Intrauterine Growth (6,736 Black Women, Massachusetts, 1975–76)

	Parameter Estimates	
Gestational age (weeks)	β_1	-183.946 (7.775)
Gestational age squared (weeks ²)	β_2	13.612 (0.479)
Gestational age cubed (weeks ³)	β_3	-0.175 (0.007)
Years of education	η_1	0.042 (0.080)
Years of age over 30	η_2	0.533 (0.093)
Years of age under 20	η_3	-0.197 (0.147)
Illegitimacy	η_4	-2.293 (0.377)
Prior perinatal loss	η_5	-1.265 (0.463)
Primigravida	η_6	-1.208 (0.405)
Care initiated 1st trimester	γ_1	0.020 (0.020)
Care initiated 2nd trimester	γ_2	0.024 (0.020)
Care initiated 3rd trimester	γ_3	0.027 (0.024)
Variance of error term ν	σ^2	187.622 (2.180)

Standard errors in parentheses.

clear dose-response relation between the onset of care and the rate of intrauterine growth.

Although the specification (17) is hardly general, a weak effect of prenatal care on birth weight was reproduced when I specified an additive effect for care rather than the multiplicative form (14), when I employed alternative measures of the quantity of care, and when I allowed for different multiplicative interactions between prenatal care and other explanatory variables.

Consider a multiparous, married black mother in her 20s, with twelve years of education, with no prior history of perinatal loss. If she received no prenatal care and delivered at 38 weeks, then from Table 1.4 her infant's birth weight is expected to be 3,063 grams. If we hold constant the duration of pregnancy, then prenatal care initiated in the first trimester adds an expected 61 grams to birth weight. However, if we calculate the total effect of initiation of care in the first trimester, inclusive of its effect on gestational age (about 1 week), then prenatal care adds an expected 169 grams to birth weight. With respect to the determination of birth weight, the contributing effect of prenatal care on the rate of intrauterine growth is considerably less than the contributing effect of prenatal care on gestational age.

The finding that prenatal care has a relatively weak effect on intrauterine growth rates among black infants is not so surprising. Although retarded fetal growth (in particular, placental insufficiency) can be detected during pregnancy, there is little in the way of treatment (Shearman et al. 1974). Although maternal cigarette smoking substantially retards intrauterine growth (Hasselmeyer et al. 1980), there is little evidence that the advice of medical practitioners has affected this practice. Only approximately 30% of current female smokers of all races quit smoking during pregnancy. Among women of all races who were last pregnant during the period 1965-75, only 35% of cigarette smokers reported receiving any physician advice about smoking (J. Harris, unpublished). Nor can I find any evidence that prenatal care has induced mothers to forego alcohol abuse. Despite all the recent advances in understanding nutrition and maternal weight gain (Niswander, Gordon et al. 1972; Habicht et al. 1974), a recent controlled trial of nutritional supplementation among black women in New York City yielded negative results (Rush et al. 1980). This study permits the striking interpretation that caloric supplementation for pregnant mothers merely ends up distributed to remaining family members (Jacobson 1980).

Prenatal Care and Infant Mortality: A Repeat Examination

Birth weight has been repeatedly found to be a critical determinant of perinatal survival (Cunningham et al. 1976; Niswander, Gordon et al. 1972; Shah and Abbey 1971; Shapiro, Schlesinger and Nesbitt 1968; U.S.

National Center for Health Statistics 1965, 1972). At any given birth weight, neonates of preterm gestational age are at greater risk than full term infants (Susser, Marolla, and Fleiss 1972). The consensus of the literature, however, is that prenatal care exerts an influence on mortality solely through its effect on birth weight. The Institute of Medicine study, for example, noted that in a linear regression with infant death as a dependent variable, the addition of a medical care "adequacy" index plus six other independent variables had no explanatory power beyond that of birth weight alone (Kessner et al. 1973, p. 63). In Gortmaker's (1979) multiple contingency table analysis, prenatal care had no consistent effect on neonatal mortality among white mothers when birth weight was included as a predetermined variable. Among black mothers, prenatal care of "intermediate" adequacy (as opposed to "adequate" or "inadequate" care) was found to have a significant effect. Shah and Abbey (1972) similarly found birth weight to be the critical intervening variable in the determination of neonatal and post-neonatal survival. Neonatal mortality, adjusted for birth weight, they found, was lower among women who initiated care in the third trimester.

The problem with all these conclusions on the effect of prenatal care is that they do not square with a critical fact about the recent, renewed decline in infant mortality in the United States.

From 1965 to 1970, the U.S. infant mortality rate declined from 24.7 to 20.0 deaths per 1,000 live births, an absolute decrease in the mortality rate equal to that for the entire period from 1950 to 1965. By 1978, the U.S. infant mortality rate had reached an estimated 13.6 per 1,000 (U.S. National Center for Health Statistics 1977, 1979, 1980a). In contrast to the pattern of mortality decline during the first half of this century, most of the recent absolute decline in infant mortality represented an improvement in neonatal survival. At least beyond 20 weeks' gestation, a substantial decline in fetal death rates was also observed. These improvements in infant survival applied to all races.

Figure 1.5 depicts the relation between birth weight and neonatal mortality, determined from matched birth and death records for the United States in early 1950 and 1960 (U.S. National Center for Health Statistics 1972, Table D), and for Massachusetts during 1969 to 1978 (Massachusetts Department of Public Health, unpublished). From 1950 to 1960, the largest proportional decline in mortality occurred among infants weighing over 2,500 grams. This category comprised only about one-quarter of all neonatal deaths in 1960. During 1969 to 1978, by contrast, there was a substantial decrease in mortality for infants weighing between 1,000 and 2,500 grams.

The contributions of these changes in birth weight-specific mortality to the total absolute decline in neonatal mortality in Massachusetts is calculated in Figure 1.6. The height of each open bar represents the observed

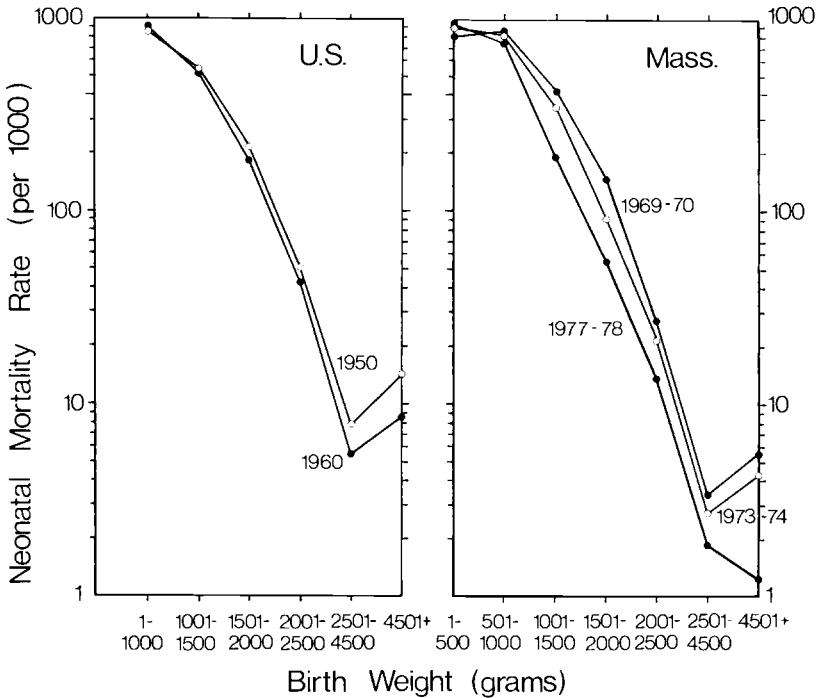


Fig. 1.5 Relation between birthweight and neonatal mortality rate per 1000 live births. United States, 1950-60; Massachusetts, 1969-78.

neonatal mortality rate among all races in Massachusetts for each year from 1969 to 1978. The height of the combined open and cross-hatched areas for the years 1970 to 1978 represents the birth weight-adjusted neonatal mortality rate. I calculated this rate by applying the birth weight-specific mortality rates for each year to the distribution of birth weights prevailing in 1969. Over 90% of the absolute decline in neonatal mortality in Massachusetts, Figure 1.6 shows, represents an improvement in birth weight-specific mortality.

There is considerable indirect evidence that the trends indicated by Figure 1.5 are representative of the entire U.S. experience (Pakter and Nelson 1974, p. 859; Kleinman et al. 1978; Chase 1977). The percentage of low birth weight and very low birth weight infants in the United States has declined somewhat during the past fifteen years. But this change is a fraction of the amount required to explain the decline in mortality if birth weight-specific mortality had remained unchanged (Lee et al. 1980).

A small fraction of the observed improvement in birth weight-specific mortality may represent favorable shifts in maternal age and parity (Mor-

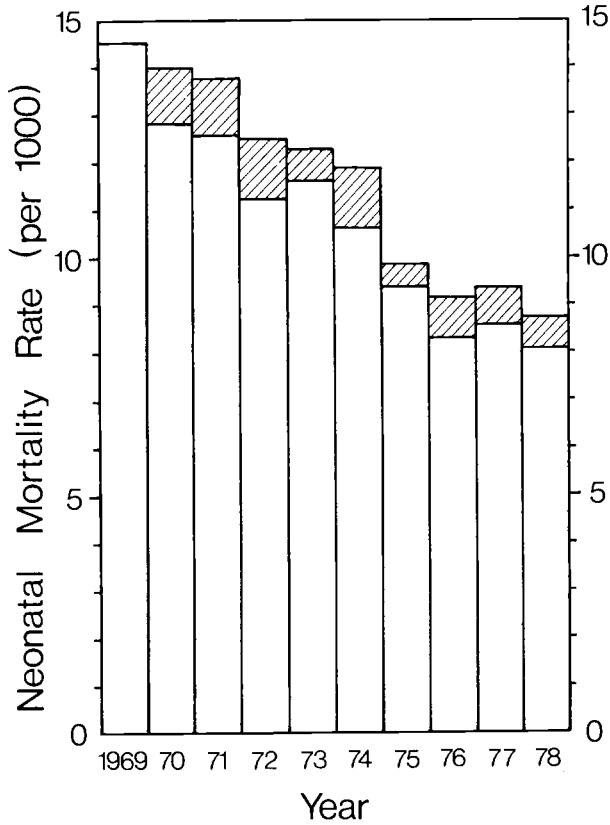


Fig. 1.6 Observed and birthweight-adjusted neonatal mortality rate per 1000 live births (Massachusetts, all races, 1969–78). Neonatal mortality rates correspond to open bars. Birthweight-adjusted neonatal mortality rates correspond to summation of open and cross-hatched bars. See text.

ris et al. 1975). Although the measurement convention for gestational age was made more precise only after 1968, there is little evidence that the joint distribution of birth weight and gestational age has changed significantly.

There are two explanations for this trend. First, we now have better medical care for the perinatal period, including labor, delivery, and early neonatal life. This improved care includes advances in neonatal intensive care, transport of high-risk mothers to regional centers, treatment of Rh-incompatibility and neonatal jaundice, and improved understanding of neonatal respiratory distress syndrome (Borkowf et al. 1979; Kitchen and Campbell 1971; T.R. Harris 1978; Stern 1976; Usher 1977).

Second, infants of a given birth weight have become healthier. This explanation frequently invokes family planning, contraception, elective abortion, genetic screening, and the elusive fact that babies are now more wanted (U.S. National Center for Health Statistics 1980*b*; Jacobowitz and Grossman 1980).

It is hardly clear what role, if any, prenatal care has played in this scenario. If the continued growth in the demand for prenatal care has had a significant impact on infant survival, then we should expect to observe a relation between prenatal care and birth weight-specific mortality in cross-section data. Moreover, if prenatal care in fact prevents early termination of pregnancy or enhances intrauterine growth rates, it is unclear why concomitant changes in the proportion of premature infants were not observed.

Table 1.5 shows the relation between birth weight and neonatal mortality according to the month of initiation of prenatal care for all races. Except for the Unknown Care category, the neonatal mortality rates for mature infants (over 2,500 grams) are indistinguishable. In the low birth weight category, those mothers who initiated care in the first month are at somewhat greater risk. The neonatal mortality rate then increases as care is delayed to the sixth month. But among those initiating care in the third trimester, the mortality rate for low birth weight infants is substantially lower.

We now see the pitfalls of a priori classifications of the amount of care based upon clinical standards (Kessner et al. 1973; Gortmaker 1979; U.S. National Center for Health Statistics 1978). Aggregation of mothers with no care and third trimester care into a single "inadequate" care category would produce a contradictory relationship between adequacy of care and birth weight-specific mortality.

The results in Table 1.5 again confront us with the problem of fetal selection. I have already suggested that the fetal population varies substantially in the rate of pregnancy termination. The sources of this variation, I further suggested, are largely unobserved. Similarly, infants of the same birth weight may vary considerably in their survival characteristics, with the sources of this heterogeneity also largely unobserved. The results in Table 1.5 suggest that those latent characteristics determining the pregnancy termination rate are correlated with those latent characteristics that determine birth weight-specific mortality.

This explanation is certainly plausible. Congenital anomalies, infection, maternal smoking, or placental insufficiency may shorten gestation and affect survival characteristics. The task of devising a structural model to test this hypothesis, however, is plagued by problems of identification.

Let μ be a latent characteristic that affects the probability of survival. An infant survives, I assume, if $\mu \leq \bar{\mu}$, where $\bar{\mu}$ may depend on various explanatory variables \underline{X} , including birth weight, gestational age, and the amount or timing of prenatal care. For a given cohort of pregnant

Table 1.5 Neonatal Mortality in Relation to Birth Weight and Month of Initiation of Prenatal Care (All Races, Massachusetts, 1975-76)

Month of Initiation of Care	Birth Weight ≤ 2500 gm	Birth Weight > 2500 gm
1st	115.4 (8.7)	1.8 (0.3)
2nd	99.0 (4.9)	1.9 (0.2)
3rd	90.8 (6.1)	2.0 (0.3)
4th	88.6 (10.4)	1.3 (0.4)
5th	94.0 (14.3)	1.2 (0.5)
6th	113.3 (22.2)	2.0 (1.0)
7th	30.5 (15.0)	2.2 (1.3)
8th or 9th	16.1 (16.0)	3.0 (2.1)
No Care	173.3 (30.9)	1.9 (1.9)
Unknown	143.3 (19.8)	5.3 (1.4)

All rates per 1,000 live births. Standard errors in parentheses.

women, if μ were distributed independently of ε , then the probability of death, conditional upon (t, v, \underline{X}) is

$$(18) \quad \int_{\bar{\mu}}^{\infty} \bar{\mu}(t, v, \underline{X}) f(\mu) d\mu$$

where $f(\mu)$ is the marginal density of μ . When μ has a logistic distribution, for example, equation (18) is the logistic model (Lewit 1977). When μ is normally distributed, (18) is a probit equation.

Suppose, however, that ε and μ were not independent across pregnant women. Then the distribution of μ , like ε , will change during the course of pregnancy. If ε and μ are positively correlated, then as gestation progresses, the proportion of low- ε types, and therefore the proportion of low- μ types, will increase. If $f(\mu|\varepsilon)$ is the conditional density of μ given ε , then the probability of death, conditional upon (t, v, \underline{X}) is now

$$(19) \quad \int_0^{\infty} \int_{\bar{\mu}}^{\infty} \bar{\mu}(t, v, \underline{X}) f(\mu|\varepsilon) f(\varepsilon|t, v, \underline{X}) d\mu d\varepsilon$$

where $f(\varepsilon | t, v, \underline{X})$ is defined in (7). Hence, if μ and ε are correlated, the single equation probit or logistic model (18) will lead to biased estimates of the effect of prenatal care and other explanatory variables. The structural parameters of $\bar{\mu}(t, v, \underline{X})$ cannot be estimated separately from those determining the hazard rate for pregnancy termination and therefore the density $f(\varepsilon | t, v, \underline{X})$.

The main problem in applying (19) to our data is that we must impose some restriction on the density $f(\mu | \varepsilon)$ in order to identify these structural parameters. That is, we must decide in advance how the selective process of eliminating high- ε infants affects the distribution of μ . Unfortunately, our inferences about the structural parameters are likely to be very sensitive to the type of restriction imposed.

The results of one such restriction are illustrated in Table 1.6. Both columns represent estimates of the parameters of $\bar{\mu}(t, v, \underline{X})$, which is assumed to be a linear function of gestational age, the duration of care, and other explanatory variables, including birth weight. Both neonatal and fetal deaths are included.

The left hand column of Table 1.6 (denoted Model III) corresponds to the case where μ and ε are assumed to be independently distributed (18). Specifically, I assume μ has gamma distribution with mean 1 and variance $1/h$. If we have independent observations $\{t_p, v_p, \underline{X}_p : p = 1, \dots, P\}$ on surviving infants and $\{t_q, v_q, \underline{X}_q : q = 1, \dots, Q\}$ on perinatal deaths, then the likelihood of these observations, conditional upon the time of initiation of care and the explanatory variables, is $L_T^{\text{II}} \times L^{\text{III}}$, where

$$(20) \quad L^{\text{III}} = \prod_{p=1}^P J(\bar{\mu}(t_p, v_p, \underline{X}_p)h; h) \\ \times \prod_{q=1}^Q [1 - J(\bar{\mu}(t_q, v_q, \underline{X}_q)h; h)]$$

where $J(x; y) = [\Gamma(y)]^{-1} \int_0^x e^{-z} z^{y-1} dz$ is the complete gamma function.

Since L^{III} does not involve any of the parameters of L_T^{II} , Table 1.6 displays only the parameters of L^{III} . The log likelihood at the bottom of this column is the maximized value of $\log L^{\text{III}}$.

The right-hand column of Table 1.6 (denoted Model IV) corresponds to a special case of interdependence between ε and μ (19). Specifically, I assume that ε and μ have a degenerate one-dimensional distribution; that is, they have an identical gamma density with mean 1 and variance $1/h$. Again consider the likelihood of the observations $\{t_p, v_p, \underline{X}_p : p = 1, \dots, P\}$ on surviving infants and $\{t_q, v_q, \underline{X}_q : q = 1, \dots, Q\}$ on perinatal deaths. In each subset, some mothers will report prenatal care, others will not. The likelihood of these observations, conditional upon the time of initiation of care and the explanatory variables, is $L_T^{\text{II}} \times L^{\text{IV}}$, where

Table 1.6 Maximum Likelihood Estimates of the Effect of Prenatal Care on the Probability of Perinatal Survival (6,736 Black Women, Massachusetts, 1975–76)

	Model III Parameter Estimates	Model IV Parameter Estimates
Constant term	-0.254 (0.403)	0.379 (0.229)
Gestational age (weeks)	0.095 (0.013)	0.001 (0.007)
Duration of prenatal care (weeks)	-0.010 (0.009)	0.002 (0.002)
Birth weight (kilograms)	1.212 (0.295)	0.646 (0.032)
Annual volume of deliveries (thousands)	0.066 (0.032)	0.045 (0.018)
Years of education	-0.014 (0.023)	-0.005 (0.012)
Years of age over 30	-0.034 (0.027)	-0.013 (0.016)
Years of age under 20	-0.033 (0.034)	-0.024 (0.023)
Illegitimacy	-0.035 (0.097)	0.062 (0.064)
Prior perinatal loss	-0.224 (0.133)	-0.156 (0.077)
Primagravida	0.048 (0.151)	-0.029 (0.083)
Variance of omitted regressor μ	0.413 (0.143)	0.176 (0.015)
Log likelihood	-314.09	-17013.75

Standard errors in parentheses. Estimates of the parameters $\{\alpha, \rho_T, \omega_T, \theta_{TK}\}$ for Model IV are not displayed.

$$(21) \quad L^{IV} = \prod_{p=1}^P J(\bar{\mu}(t_p, v_p, \underline{X}_p)[h + \Lambda_{TV}(t_p, v_p | \underline{X}_p)]; h) \times \prod_{q=1}^Q [1 - J(\bar{\mu}(t_q, v_q, \underline{X}_q)[h + \Lambda_{TV}(t_q, v_q | \underline{X}_q)]; h]$$

where Λ_{TV} is replaced by Λ_T in cases where no care was obtained. The partial likelihood L^{IV} involves not only the parameters of $\bar{\mu}$, but also $\{\alpha, \rho_T, \omega_T, \theta_{TK}\}$, which appear in L_T^II . Unlike Model III, the parameters of L_T^II and L^{IV} must be estimated jointly. Since the estimates of $\{\alpha, \rho_T, \omega_T, \theta_{TK}\}$ were very close to those in Table 1.3, they are not shown

in Table 1.6. The log likelihood at the bottom of the right-hand column is the maximized value of $\log(L_T^{\text{II}} \times L_T^{\text{IV}})$.

For Model III, with μ independent of ε and therefore no fetal selection, birth weight and gestational age significantly affect the probability of survival. The duration of care, on the other hand, has an estimated negative effect. For Model IV, which incorporates fetal selection, the effect of prenatal care is weakly positive, whereas the influence of gestational age appears to be reduced. The latter parameter, however, captures only the direct effect of gestational age on $\bar{\mu}$, that is, the effect of increased duration of pregnancy on the survival rate of a given infant. There is also an indirect effect on Λ_{TV} , that is, the effect of increased duration of pregnancy on the distribution of latent characteristics.

From the parameter estimates in Table 1.6, I can calculate the elasticity of the perinatal mortality rate with respect to each continuous explanatory variable. For a married, multiparous black mother in her 20s, with twelve years of attained education and no prior perinatal loss, who delivers a 3,100 gram infant at 38 weeks in a hospital with 3,100 deliveries annually, I obtain the following elasticities:

	<i>Model III</i>		<i>Model IV</i>
Birth weight	- 7.79		- 8.72
Gestational age	- 0.75	{ direct	- 0.25
		indirect	- 0.32
Duration of care	0.52		- 0.23
Annual Volume of deliveries	- 0.42		- 0.61

In both models, birth weight has the dominant effect. Comparison of the direct and indirect elasticities for gestational age suggests that a substantial fraction of the observed effect of duration of pregnancy on mortality represents fetal selection over time. Although prenatal care has a favorable direct effect on mortality in Model IV, its elasticity is small. (The indirect effect of retarding fetal selection was negligible in this example.) Therefore, the main effect of prenatal care on perinatal mortality will still be its influence on birth weight. In this example, the complete absence of care would result in a 20% proportional increase the perinatal mortality rate, conditional upon birth weight and gestational age. Using the estimates derived in the previous section, I calculate that the absence of care, through its effect on the intrauterine growth rate and therefore birth weight, would result in a 17% proportional increase in the perinatal mortality rate. Similarly, the absence of care, through its effect on gestational age and therefore, on birth weight, would result in a 32% proportional increase in the perinatal mortality rate.

Finally, it is noteworthy that the annual volume of deliveries in the hospital of birth has a significant effect on survival. This finding supports

the hypothesis that perinatal medical care, as opposed to prenatal care, has an important influence on birth weight-specific mortality.

Summary and Conclusions

Four main issues, I have demonstrated, underlie the controversy about prenatal care and pregnancy outcome.

First, the relationship between the timing of prenatal visits and the duration of pregnancy has been poorly characterized. Mothers with little or no prenatal care, it has been repeatedly observed, have a higher proportion of preterm babies. This fact suggests that prenatal care prevents premature labor. But early termination of pregnancy from any cause necessarily interrupts the course of prenatal care. In order to sort out these two confounding explanations, I devised a continuous time stochastic model in which the initiation of care and premature delivery were competing risks. Applying this model to a sample of black women's pregnancies, I found that prenatal care indeed prevented preterm delivery. The magnitude of this effect was equivalent to an approximate 1-week increase in the average duration of gestation.

Second, the risks of early termination of pregnancy vary considerably among unborn infants. These variations in risk set up a powerful selection mechanism in which less healthy fetuses are progressively eliminated from a cohort of pregnant women as gestation proceeds. As a result of this selection, those mothers who initiate care late in pregnancy necessarily have infants with characteristics quite different from those who initiate care earlier during gestation. Moreover, the underlying health characteristics subject to selection may be subtle and difficult to measure.

To investigate the potential errors of inference produced by this selection phenomenon, I included a mathematical model of fetal selection in my analysis of prenatal care and length of gestation. I showed that failure to account for fetal selection can indeed result in biased inferences about the effects of prenatal care and other prenatal risk factors. In particular, if prenatal care retards the early termination of pregnancy, then it also retards the fetal selection mechanism. Unless we incorporate the effect of prenatal care on the distribution of fetal characteristics over time, its influence on the duration of pregnancy will be understated.

Unfortunately, this solution to the problem of fetal selection requires overly restrictive assumptions about the determinants of the demand for prenatal care. There is the possibility that mothers could ascertain those fetal characteristics that are not revealed to the investigator. These latent characteristics could in turn affect the demand for medical care. When these possibilities were introduced in the analysis, it became impossible to make inferences about both the effect of prenatal care on the risk of preterm delivery and the influence of these risks on the demand for care.

Third, the frequently observed correlation between the quantity of

prenatal care and birth weight lacks a convincing biological or behavioral explanation. Prenatal surveillance, to be sure, might indirectly improve birth weight by preventing early termination of pregnancy. But a mechanism for a direct effect of prenatal care on the rate of intrauterine growth is more elusive. I therefore specified a model in which the timing of care affected the rate of intrauterine growth. Applying this model to the cross-section data, I found that the influence of care on birth weight was only weakly positive and statistically insignificant. Through its effect on intrauterine growth rates, prenatal care would increase birth weight in a typical pregnancy by about 60 grams. By contrast, through its indirect effect on the duration of gestation, prenatal care would increase birth weight in a typical pregnancy by about 110 grams.

These findings are consistent with current understanding of the determinants of birth weight. In contrast to premature labor, which can be treated if not detected in advance (Chard 1974), there is no available treatment for placental insufficiency or other forms of intrauterine growth retardation (Spearman et al. 1975). Physician advice does not clearly alter maternal smoking, alcohol use, or nutrition. We cannot with certainty make any stronger inferences about the effect of prenatal care on birth weight when the underlying mechanisms of the effect remain in doubt.

Fourth, past analyses of prenatal care have not squarely confronted a critical point about the recent decline in U.S. neonatal and infant mortality; that is, the decline in mortality primarily reflects a striking improvement in the survival rates of low-birth-weight infants. By contrast, there has been comparatively little change in the proportion of low-birth-weight infants or the fraction of preterm deliveries. If the recent growth in prenatal care had a significant impact on infant survival, then we should expect to observe a relation between prenatal care and birth weight-specific mortality in cross-section data. Moreover, if prenatal care in fact affects birth weight, it is unclear why concomitant changes in the rate of prematurity were not observed.

I examined the relation between birth weight-specific mortality and the timing of prenatal care, and confirmed the frequently cited, contradictory observation that mothers who initiate care late during pregnancy have infants with considerably lower birth weight-specific mortality. This finding is consistent with the effects of fetal selection. If the fetal population varies in its risks of early termination of pregnancy, then among those mothers remaining pregnant into the third trimester, there will be a smaller proportion of high-risk infants. If the risk of premature delivery is correlated with fetal survival characteristics, then the fetal selection mechanism will also affect the distribution of these survival characteristics.

I formulated a specific model of the relation between fetal selection and fetal survival characteristics. Applying this model to the subsample of

black women's pregnancies, I found that prenatal care had a weakly positive effect on birth weight-specific perinatal mortality. Unfortunately, this conclusion is contingent upon my specifying a particular mechanism of sorting unobserved characteristics. In the case where these latent regressors affect the demand for prenatal care or other fetal health characteristics, even stronger restrictions are required to identify the statistical parameters. The effect of prenatal care on fetal health, the distribution of fetal health characteristics, and the feedback effect of these characteristics on the demand for prenatal care cannot jointly be identified from cross-section data of vital records.

My analysis of the prenatal care controversy has side-stepped a number of additional difficulties. No attempt was made here to evaluate the quality, as opposed to the quantity, of prenatal care. Although I distinguished formally between prenatal care and perinatal care, the potential interaction of these factors has not been considered. Thus it is possible that prenatal care serves primarily to facilitate certain treatments in the perinatal period. This possibility is consistent with the finding that black women with prenatal care have lower risks of preterm delivery. Moreover, many of the results of this paper were derived from a sample of black women's pregnancies. Since medical intervention may vary in its influence on the health of different races, the quantitative estimates cannot be applied generally at this time. Finally, my analysis made only passing reference to the problem of nonrandomly missing observations. It ought to be recognized that those vital records with omitted entries for prenatal care and other data may be the most critical ones.

Would a detailed longitudinal study of the natural histories of many pregnancies overcome all these problems? So long as the expectant mothers choose when and if to seek prenatal care, such a study cannot overcome the problem of fetal selection. Nor can it eliminate the competition between early care and early fetal loss. Perhaps nothing short of a controlled, randomized trial will do. Even in that case, we cannot merely wait until an experimental subject recognizes her pregnancy and then assign her to a particular prenatal regimen. Instead, we would need to assign large cohorts of women to alternative experimental treatments prior to the onset of pregnancy. Moreover, independent ascertainment of the onset of pregnancy would be required. Such an experiment is surely difficult to perform.

Perhaps the most feasible approach is to design clinical studies that are more narrowly focused on certain types of prenatal intervention. They may not resolve the value of the millions of routine visits women make to their obstetricians. But we could at least learn something about prenatal diet, weight gain, vitamin supplementation, exercise, ultrasound studies, and other aspects of medical care.

References

- American College of Obstetricians and Gynecologists. 1974. *Standards for obstetric-gynecologic services*. Chicago: American College of Obstetricians and Gynecologists.
- Bakketeig, Leiv S.; Seigel, Daniel G.; and Sternthal, Phyllis M. 1978. A fetal-infant life table based on single births in Norway, 1967-1973. *American Journal of Epidemiology* 107:216-25.
- Borkowf, H.I.; Grausz, J.P.; and Delfs, E. 1979. The effect of a perinatal center on perinatal mortality. *Obstetrics and Gynecology* 53:633.
- Chard, T. 1974. The fetus at risk. *Lancet* 2:880-82.
- Chase, Helen C., ed.; Erhardt, Carl L.; and Nelson, Frieda G. 1973. A study of risks, medical care, and infant mortality. *American Journal of Public Health* 63(Supp.):1-56.
- . 1974. Perinatal mortality: overview and current trends. *Clinics in Perinatology* 1:3-17.
- . 1977. Time trends in low birth weight in the United States. In Dwayne M. Reed and Fiona J. Stanley, ed., *The epidemiology of prematurity*. Baltimore and Munich: Urban & Schwarzenberg.
- Cunningham, George C.; Hawes, Warren E.; Madore, Carol; Norris, Frank D.; and Williams, Ronald L. 1976. Intrauterine growth and neonatal risk in California. Santa Barbara: University of California, mimeo.
- David, H.A., and Moeschberger, M.L. 1978. *The theory of competing risks*. Griffin's Statistical Monograph Series, No. 39. London: Charles Griffin & Company, Ltd.
- Drillien, C.M. 1957. The social and economic factors affecting the incidence of premature births. Part I, Premature births without complications of pregnancy. *Journal of Obstetrics and Gynecology of the British Empire* 64:161-84.
- Eastman, N.J. 1947. Prematurity from the viewpoint of the obstetrician. *American Practice* 1:343-52.
- Gortmaker, Steven L. 1979. The effects of prenatal care upon the health of the newborn. *American Journal of Public Health* 69:653-60.
- Gruenwald, Peter. 1966. Growth of the human fetus. Part I, Normal growth and its violation. *American Journal of Obstetrics and Gynecology* 94:1112.
- . 1974. Pathology of the deprived fetus and its supply line. In *Size at birth*, Ciba Foundation Symposium 27 (new series). Amsterdam: North-Holland.
- Habicht, Jean-Pierre; Lechtig, Aaron; Yarbrough, Charles; and Klein, Robert E. 1974. Maternal nutrition, birth weight, and infant mortality.

- In *Size at birth*, Ciba Foundation Symposium 27 (new series). Amsterdam: North-Holland.
- Harris, T.R.; Isaman, J.; and Giles, H.R. 1978. Improved neonatal survival through maternal transport. *Obstetrics and Gynecology* 52:294.
- Hasselmeyer, Eileen G. et al. 1980. Pregnancy and infant health. In *The health consequences of smoking for women, a report of the surgeon general*. Washington,; U.S. Department of Health and Human Services.
- Hellman, L.M. 1953. The prevention of premature births. In *Prematurity, congenital malformation and birth injury*. New York: Association for the Aid of Crippled Children.
- Jacobson, Howard N. 1980. A randomized controlled trial of prenatal nutritional supplementation (editorial). *Pediatrics* 65:835-36.
- Jacobowitz, S., and Grossman M. 1980. Determinants of variations in infant mortality rates among counties of the United States: the roles of social policies and programs. Presented at World Congress on Health Economics, Leiden University, The Netherlands, 8-11 September 1980.
- Johnson, Norman L., and Kotz, Samuel. 1972. *Distributions in statistics: continuous multivariate distributions*. New York: John Wiley.
- Kane, S.H. 1964. Significance of prenatal care. *Obstetrics and Gynecology* 24:66-72.
- Kessner, David M.; Singer, James; Kalk, Carolyn E.; and Schlesinger, Edward R. 1973. *Infant death: an analysis by material risk and health care*. Washington: National Academy of Sciences, Institute of Medicine.
- Kitchen, W.H., and Campbell, D.G. 1971. Controlled trial of intensive care for very low birth weight infants. *Pediatrics* 48:711-14.
- Kleinman, Joel; Kovar, Mary G.; Feldman, Jacob J.; and Young, Cecelia A. 1978. A comparison of 1960 and 1972-74 early neonatal mortality in selected states. *American Journal of Epidemiology* 108:454-69.
- Lancaster, Tony. 1979. Econometric methods for the duration of unemployment. *Econometrica* 47:939-59.
- Lee, Kwang-Sun; Paneth, Nigel; Gartner, Lawrence M.; Pearlman, Mark A.; and Bross, Leslie. 1980. Neonatal mortality: an analysis of the recent improvement in the United States. *American Journal of Public Health* 70:15-21.
- Lewit, Eugene M. 1977. Experience with pregnancy, the demand for prenatal care, and the production of surviving infants. Ph.D. diss., City University of New York.
- Lubchenco, Lula O. 1975. Assessment of weight and gestational age. In Gordon B. Avery, ed., *Neonatology*. Philadelphia: J.B. Lippincott.

- Mellin, Gilbert, W. 1972. Fetal life tables: a means of establishing perinatal rates of risk. *Journal of the American Medical Association* 180:91-94.
- Morris, N.M.; Udry, J.R.; and Chase, C.L. 1975. Shifting age-parity distribution of births and the decrease in infant mortality. *American Journal of Public Health* 65:359-62.
- Niswander, Kenneth R., Gordon, Myron et al. 1972. *The women and their pregnancies*. Collaborative Perinatal Study, DHEW Publ. No. (NIH) 73-379. Washington: National Institute of Neurological Diseases and Stroke.
- Oppenheimer, E. 1961. Population and changes and perinatal mortality. *American Journal of Public Health* 51:208-16.
- Pakter, Jean, and Nelson, Frieda. 1974. Factors in the unprecedented decline in infant mortality in New York City. *Bulletin of the New York Academy of Medicine* 50:839-68.
- ; Rosner, H.J.; Jacobziner, H.; and Greenstein, R. 1961. Out-of-wedlock births in New York City. Part II, Medical aspects. *American Journal of Public Health* 51:846-65.
- Rush, David; Stein, Zena; and Susser, Mervyn. 1980. A randomized controlled trial of prenatal nutritional supplementation in New York City. *Pediatrics* 65:683-97.
- Russell, Louise B., and Burke, Carol S. 1975. Determinants of infant and child mortality: an econometric analysis of survey data from San Juan, Argentina. Report prepared for the Agency for International Development. Washington: National Planning Association.
- Shah, Farida K., and Abbey, Helen. 1971. Effects of some factors on neonatal and postneonatal mortality: analysis by a binary variable regression method. *Milbank Memorial Fund Quarterly* 49:33-57.
- Shapiro, Sam; Schlesinger, Edward R.; and Nesbitt, Robert E.L., Jr. 1968. *Infant, perinatal, maternal, and childhood mortality in the United States*. Cambridge: Harvard University Press.
- Shearman, Rodney P.; Shutt, Donald A.; and Smith, Ian D. 1974. The assessment and control of human fetal growth. In *Size at birth*, Ciba Foundation Symposium 27 (new series). Amsterdam: North-Holland.
- Schmidt, Peter, 1981. Constraints on the parameters in simultaneous tobit and probit models. In Daniel McFadden and Charles Manski, eds., *Structural analysis of discrete data with econometric applications*. Cambridge: M.I.T. Press (forthcoming).
- Shwartz, Samuel. 1962. Prenatal care, prematurity, and neonatal mortality. *American Journal of Obstetrics and Gynecology* 83:591-98.
- , and Vinyard, J. H. 1965. Prenatal care and prematurity. *Public Health Reports* 80:237-48.
- Slesinger, Doris P., and Travis, Harry P. 1975. A study of infant mortal-

- ity in Wisconsin, 1969, from linked birth and death records: an application of log-linear models. Working Paper 75-15, Center for Demography and Ecology, University of Madison, Wisconsin.
- Stern, Leo. 1976. The high-risk neonate. In William N. Spellacy, ed., *Management of the high-risk pregnancy*. Baltimore: University Park Press.
- Susser, Mervyn; Marolla, Francis A.; and Fleiss, Joseph. 1972. Birth weight, fetal age, and perinatal mortality. *American Journal of Epidemiology* 96:197-204.
- Taylor, William F. 1970. The probability of fetal death. In F. Clarke Fraser and Victor A. McKusick, eds., *Congenital malformations*. Proceedings of the Third International Conference, The Hague, 1969. Amsterdam: Excerpta Medica Foundation.
- Terris, Milton, and Glasser, Marvin. 1974. A life table analysis of the relation of prenatal care to prematurity. *American Journal of Public Health* 64:869-75.
- , and Gold, E. 1969. An epidemiologic study of prematurity. *American Journal of Obstetrics and Gynecology* 103:371-79.
- U.S. National Center for Health Statistics. 1965. Weight at birth and survival of the newborn, United States, early 1950. *Vital and Health Statistics*, series 21, no. 3.
- U.S. National Center for Health Statistics. 1972. A study of infant mortality from linked records: comparison of neonatal mortality from two cohort studies. *Vital and Health Statistics*, series 20, no. 13.
- . 1977. *Vital Statistics of the United States*. Volume II, Mortality, Part A. Washington: U.S. Government Printing Office.
- . 1978. Prenatal care: United States, 1969-1975. *Vital and Health Statistics*, series 21, no. 33.
- . 1979. Provisional statistics, annual summary for the United States, 1978. *Monthly Vital Statistics Report* 27:1-28.
- . 1980a. *Health, United States, 1979*. DHEW Publication No. (PHS) 80-1232. Washington. U.S. Government Printing Office.
- . 1980b. Wanted and unwanted births reported by mothers 15-44 years of age: United States, 1976. *Advance Data From Vital and Health Statistics*, no. 56.
- Usher, R. 1977. Changing mortality rates with perinatal care and regionalization. *Seminars in Perinatology* 1:309.
- Wells, Henry B.; Greenberg, Bernard G.; and Donnelly, James F. 1958. North Carolina fetal and neonatal death study. Part I, Study design and some preliminary results. *American Journal of Public Health* 48:1583-95.
- Williams, Ronald L. 1975. Intrauterine growth curves: intra- and international comparisons with different ethnic groups in California. *Preventive Medicine* 4:163-72.