The Effect of Congenital Anomalies on Mortality Risk in White and Black Infants

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ABSTRACT

Objectives. This population-based study examined the effect of all major congenital anomalies on the mortality of White and Black infants by infant sex, birthweight, gestational age, and lethality of the anomaly. The study also determined the total contribution of anomalies to infant mortality.

Methods. California Birth Defects Monitoring Program data were merged with linked birth–death files for 278 646 singleton non-Hispanic White and Black infants born in 1983 through 1986. Malformed infants were compared with nonmalformed infants to determine the effect of anomalies on mortality.

Results. The presence of any congenital anomaly increased mortality 9.0-fold (95% CI = 7.3, 11.1) for Black infants and 17.8-fold (95% CI = 16.2, 19.6) for White infants. Even "nonlethal" anomalies increased mortality up to 8.9-fold. Overall, anomalies contributed to 33% of White infant deaths, to 19% of Black infant deaths, and to over 60% of deaths among Black and White neonates weighing over 1499 g.

Conclusions. The contribution of congenital anomalies to mortality of both low- (<2500 g) and normal-birthweight infants is substantially higher than previously estimated, representing a large public health problem for both Black and White infants. (*Am J Public Health.* 1999;89:887–892)

Congenital anomalies are the leading cause of infant mortality among US Whites and the third leading cause among US Blacks.[1] Most population-based studies of mortality from congenital anomalies among US White and Black infants have relied on death certificate data as the sole source of information on anomalies.[2–8] Although death certificate data are widely available and generally cover the entire US population, they are inadequate for studying the full effect of anomalies on mortality because only limited diagnostic information about anomalies is provided on death certificates and the reporting of specific anomalies may be inaccurate.[9,10]

Two studies have addressed these limitations by merging data from birth defects registries with matched birth–death files.[11,12] These studies compared mortality rates among all infants born with anomalies with rates among all live births. They found that malformed infants born during the 1980s had death rates approximately 6 times greater than that of the general population of live-born infants. However, these studies underestimated the effect of anomalies on mortality because they did not examine mortality rates among nonmalformed infants.

We used data from the California Birth Defects Monitoring Program (CBDMP)[13] to compare neonatal, postneonatal, and infant mortality rates for malformed vs nonmalformed White and Black infants. In addition, we examined the effect on infant mortality risks of anomalies that are generally considered nonlethal. We also used these data to determine the total contribution of anomalies to neonatal, postneonatal, and infant mortality. Last, since anomalies are strongly associated with low birthweight and intrauterine growth retardation,[14–17] we investigated the interrelations between birthweight, gestational age, anomalies, and mortality for White and Black infants.

Methods

Data Sources and Study Population

Two databases—CBDMP data and California linked birth–death cohort files—were merged for this study. Infants with congenital anomalies were identified by the CBDMP, a population-based registry that actively collects information on major structural and chromosomal anomalies from medical records of hospitals and genetic centers.[13] In determining whether an infant has an anomaly, the CBDMP considers the phenotypic description of the anomaly in the medical records, when the diagnosis was made (it must be between conception and the first birthday), and the specificity of the diagnostic approach used to confirm the diagnosis.[13] Certain conditions such as patent ductus arteriosus and lung hypoplasia were reportable only among infants at or beyond 38 weeks gestation or in combination with other reportable anomalies.

The California birth–death cohort files contain birth and infant death certificate information on all live-born California infants. The linked files were used to identify all singletons born alive between January 1, 1983, and December 31, 1986. An infant was

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TABLE 1—Mortality Rates Among Nonmalformed and Malformed Infants and Percentage of All Deaths That Occurred Among Malformed Infants, by Age at Death and Maternal Race: California, 1983–1986

	Whites (n = 240 413)			Blacks (n = 38 098)		
	Mortality Rate (per 1000)		% of Deaths,	Mortality Rate (per 1000)		% of Deaths,
Age at Death	Nonmalformed	Malformed	Malformed	Nonmalformed	Malformed	Malformed
Neonate (birth–27 d)	2.6	62.6	40.5	6.3	62.0	20.5
Postneonate (28 d–1 y)	2.5	27.9	23.7	5.3	42.4	17.2
Infant (birth–1 y)	5.1	90.5	33.2	11.6	104.4	19.0

eligible for the study if (1) the mother resided in one of the counties monitored by the CBDMP in 1983 through 1986 (representing 27% of California births), (2) the mother was identified on the birth certificate as either Black or non-Hispanic White, and (3) the delivery occurred in a nonmilitary facility. A total of 278 646 infants (240 525 White and 38 121 Black) were eligible for the study.

The linked birth–death files were used to obtain information on infant (from birth to 1 year of age), neonatal (from birth through 27 days of age), and postneonatal (from 28 days to 1 year of age) mortality; birthweight (grams); completed weeks of gestation; sex; and maternal race. Completed weeks of gestation were calculated on the basis of the mother's last menstrual period. If the month and year of the last menstrual period were known but the day was not, a value of 15 days was imputed. Gestational ages of less than 20 or more than 46 completed weeks were assigned missing values. A total of 13 991 (5.0%) infants (11 406 White and 2585 Black) had missing gestational ages, and 120 infants (99 White and 21 Black) had missing birthweights.

All analyses were performed separately for White and Black infants, because overall infant mortality rates, and the contribution of anomalies to infant mortality, differ substantially for these 2 groups.[1] Although we considered it possible that the minute portion of a population's gene pool that informs racial classification might increase susceptibility to certain lethal congenital anomalies, we considered race primarily a social, cultural, and political construct that results in varying types and severity of exposures and access to care for Blacks and Whites in US society.

We divided birthweight into 3 groups for analyses: very low birthweight (<1500 g), moderately low birthweight (1500–2499 g), and normal birthweight (≥2500 g). Gestational age was also divided into 3 groups: term gestations (≥37 completed weeks), preterm gestations of 28 through 36 weeks, and preterm gestations of fewer than 28 completed weeks. Intrauterine growth retardation was assessed by using Yerushalmy's 5-group classification that divides moderately-low-birthweight and normal-birthweight infants into term and preterm births.[18]

Analysis of Mortality Risks: Malformation Status and Lethality Class

The effect of anomalies on mortality was assessed in 2 ways. First, infants who were determined by the CBDMP to have at least 1 congenital anomaly were considered malformed and were compared with all other infants who were considered nonmalformed. Infants (112 White and 23 Black) whose only diagnosis was intussusception of the intestine were excluded from these analyses because it could not be determined whether the obstruction was due to an unidentified underlying congenital anomaly or to some other source.

Second, malformed infants were assigned to 1 of 4 lethality groups and were compared with nonmalformed infants, who served as the reference for each group. Lethality class reflected the probability of infant death and was determined by a pediatric geneticist who reviewed (blind to vital status) a listing of all anomaly diagnoses and classified each diagnosis as having a very low, low, high, or very high likelihood of resulting in infant death (a complete listing is available from the first author). Infants with more than 1 anomaly were assigned to the lethality class of their most lethal anomaly. Only 4 infants could not be classified owing to insufficient diagnostic information.

Statistical Methods

Mortality was the outcome under study. Neonatal, postneonatal, and infant mortality rates were calculated by dividing the number of deaths in the appropriate time period by the total number of live births. All rates are expressed per 1000 live births. Rate ratios (RRs) and 95% confidence intervals (CIs) were computed with Epistat.[19]

Results

The overall infant mortality rate was 7.4 per 1000 live births for Whites and 13.9 per 1000 for Blacks. A total of 967 (2.5%) Black and 6551 (2.7%) White infants had at least 1 major congenital anomaly identified by the CBDMP. Approximately 9% (90.5 per 1000) of all malformed White infants and 10% of all malformed Black infants died in the infant period (Table 1). Among Whites, the infant mortality rate for malformed infants was 17.8 times greater than for nonmalformed infants (95% CI = 16.2, 19.6); among Blacks, the rate ratio comparing malformed to nonmalformed infants was 9.0 (95% CI = 7.3, 11.1) (Table 1). Rate ratios comparing mortality of White malformed to nonmalformed infants were higher in the neonatal (RR = 24.3; 95% CI = 21.5, 27.5) than in the postneonatal (RR = 11.1; 95% CI = 9.4, 13.1) period, whereas the rate ratios for Black infants did not differ substantially by age at death.

For Whites, infants with at least 1 major anomaly comprised 33.2% (95% CI = 31.1, 35.5) of all infant deaths and 40.5% (95% CI = 37.4, 43.6) of all neonatal deaths (Table 1). For Blacks, malformed infants constituted 19.0% (95% CI = 15.8, 22.7) of all infant deaths, with similar percentages occurring in the neonatal and postneonatal periods.

Lethality Class

The prevalence of malformed infants varied by lethality class. For both Whites and Blacks, infants assigned to the very-low-lethality class were the most prevalent (16 per 1000), whereas the prevalences of infants assigned to the low-, high-, or very-high-lethality class varied between 3 per 1000 and 4 per 1000.

As expected, mortality among Black and White malformed infants increased as lethality increased (Table 2). High-lethality and very-high-lethality anomalies were associated with large mortality rate ratios in both the neonatal and postneonatal periods. Contrary to expectations, Black and White malformed infants assigned to the very-low-

TABLE 2—Mortality Rate Ratios (RRs) Comparing Malformed and Nonmalformed Infants, by Age at Death, Lethality Class, and Maternal Race: California, 1983–1986

	Neonatal Period		Postneonatal Period		Infant Period
Lethality Class	No. of Deaths	RR (95% CI)	No. of Deaths	RR (95% CI)	RR (95% CI)
White					
Nonmalformed[a]	603	1.0	588	1.0	1.0
Very low	38	4.0 (2.9, 5.5)	32	3.4 (2.4, 4.9)	3.7 (2.9, 4.7)
Low	12	4.5 (2.6, 8.0)	12	4.6 (2.6, 8.2)	4.6 (3.1, 6.8)
High	96	40.2 (32.7, 49.4)	65	27.9 (21.8, 35.8)	34.1 (29.3, 39.7)
Very high	263	118.2 (103.9, 134.4)	74	34.1 (27.0, 43.0)	76.7 (69.3, 84.8)
Black					
Nonmalformed[a]	233	1.0	197	1.0	1.0
Very low	9	2.4 (1.2, 4.6)	11	3.4 (1.9, 6.3)	2.9 (1.8, 4.5)
Low	0	NA[b]	2	3.0 (0.8, 11.9)	1.4 (0.3, 5.4)
High	20	22.0 (14.3, 33.7)	13	16.9 (9.9, 28.9)	19.7 (14.4, 26.9)
Very high	31	52.6 (38.3, 72.0)	15	30.1 (18.5, 48.8)	42.3 (33.7, 53.0)

Note. CI = confidence interval.
[a]Reference group.
[b]Not available because there were no deaths among Black infants assigned to the low-lethality class.

lethality or low-lethality class had mortality rates 1.4 to 4.6 times higher than those of nonmalformed infants (Table 2) and accounted for 20% of all deaths among malformed infants.

Analysis by Infant Sex

Among Whites, infant mortality rate ratios for malformed compared with nonmalformed infants were substantially higher for girls (RR = 23.9; 95% CI = 20.6, 27.6) than for boys (RR = 14.1; 95% CI = 12.5, 16.1). While this interaction between infant sex and anomalies occurred in both the neonatal and postneonatal periods, it was more marked in the latter.

The interactions between infant sex and anomalies among Whites were primarily due to 2 factors: (1) among infants with very-high-lethality anomalies, the absolute postneonatal mortality rates were higher for girls (108.6 per 1000) than for boys (66.4 per 1000), and (2) postneonatal mortality rates were lower for nonmalformed girls (19.9 per 1000) than for nonmalformed boys (30.2 per 1000).

Among Blacks, postneonatal mortality rate ratios for malformed compared with nonmalformed infants were likewise higher among girls (RR = 11.4; 95% CI = 7.2, 18.2) than among boys (RR = 5.9; 95% CI = 3.7, 9.4); sparse data limited further exploration.

Analysis by Birthweight and Gestational Age

Among both Blacks and Whites, neonatal mortality rate ratios for malformed infants compared with nonmalformed infants increased substantially as birthweight increased (Table 3). For example, among very-low-birthweight Whites, mortality for malformed neonates was 1.5 times higher than for nonmalformed neonates (95% CI = 1.3, 1.8), whereas among normal-birthweight Whites the neonatal mortality rate ratio was 53.1 (95% CI = 42.8, 66.0). In addition, among normal-birthweight and moderately-low-birthweight infants, the effect of anomalies on neonatal mortality was almost identical for Blacks and Whites (Table 3). In the postneonatal period, the mortality rate ratios for White infants did not increase as dramatically with increasing birthweight, and among Black infants there was no notable increase (Table 3).

The percentage of all deaths that occurred among malformed infants also varied considerably by birthweight and age at death (Table 3). The contribution of anomalies to neonatal mortality was much higher among infants with birthweights over 1499 g than among very-low-birthweight infants. Anomalies were present in approximately 60% of all neonatal deaths occurring among both Black and White infants with

TABLE 3—Mortality Rate Ratios (RRs) (Malformed vs Nonmalformed) and Percentage of All Deaths That Occurred Among Malformed Infants, by Age at Death, Birthweight, and Maternal Race: California, 1983–1986

	Neonatal Period		Postneonatal Period		Infant Period	
Birthweight, g	RR (95% CI)	% of Deaths, Malformed	RR (95% CI)	% of Deaths, Malformed	RR (95% CI)	% of Deaths, Malformed
White						
<1500	1.5 (1.3, 1.8)	20.4	2.9 (1.8, 4.7)	33.3	1.7 (1.4, 1.9)	22.1
1500–2499	25.2 (18.4, 34.5)	67.9	6.7 (4.4, 10.2)	35.9	15.5 (12.3, 19.6)	56.5
≥2500	53.1 (42.8, 66.0)	57.1	10.6 (8.7, 12.8)	21.0	20.3 (17.8, 23.2)	33.8
Black						
<1500	0.6 (0.3, 1.1)	4.7	4.5 (2.3, 8.6)	28.2	1.1 (0.7, 1.6)	8.7
1500–2499	26.1 (12.5, 54.6)	62.1	7.2 (3.9, 13.3)	31.1	12.2 (7.9, 18.8)	43.2
≥2500	56.0 (34.0, 92.2)	54.1	5.7 (3.4, 9.5)	10.7	14.4 (10.5, 19.6)	23.2

Note. CI = confidence interval.

TABLE 4—Neonatal and Postneonatal Mortality Rate Ratios (RRs) (Malformed vs Nonmalformed), by Birthweight, Lethality Class, and Maternal Race: California, 1983–1986

	Neonatal Mortality			Postneonatal Mortality		
Lethality Class	<1500 g RR (95% CI)	1500–2499 g RR (95% CI)	≥2500 g RR (95% CI)	<1500 g RR (95% CI)	1500–2499 g RR (95% CI)	≥2500 g RR (95% CI)
White						
Nonmalformed[a]	1.0	1.0	1.0	1.0	1.0	1.0
Very low	0.9 (0.6, 1.3)	1.7 (0.5, 5.5)	6.6 (3.8, 11.4)	2.5 (1.2, 5.3)	3.7 (1.7, 8.0)	2.5 (1.6, 4.0)
Low	0.5 (0.2, 1.3)	4.3 (1.4, 13.6)	8.9 (3.6, 21.6)	6.0 (2.8, 12.8)	NA[b]	2.6 (1.1, 6.3)
High	1.1 (0.8, 1.7)	29.0 (18.8, 44.8)	107.0 (78.0, 146.9)	2.4 (1.0, 5.7)	8.2 (4.0, 16.8)	33.7 (25.6, 44.5)
Very high	3.1 (2.7, 3.6)	69.2 (50.8, 94.2)	309.7 (246.0, 389.9)	2.7 (1.2, 6.2)	13.1 (7.8, 22.0)	39.1 (29.6, 51.6)
Black						
Nonmalformed[a]	1.0	1.0	1.0	1.0	1.0	1.0
Very low	0.5 (0.2, 1.3)	8.4 (2.4, 29.7)	5.1 (1.2, 21.3)	2.5 (0.8, 7.8)	4.0 (1.4, 11.1)	2.1 (0.8, 5.7)
Low	NA[b]	NA[b]	NA[b]	NA[b]	NA[b]	NA[b]
High	0.6 (0.2, 1.8)	63.8 (25.3, 160.4)	134.0 (68.7, 261.4)	4.0 (1.3, 12.2)	7.5 (1.9, 30.0)	20.4 (10.3, 40.4)
Very high	1.0 (0.3, 3.5)	92.3 (41.4, 205.8)	414.6 (248.6, 691.6)	13.4 (6.1, 29.4)	25.5 (12.2, 53.1)	17.3 (6.6, 45.3)

Note. CI = confidence interval.
[a]Reference group.
[b]Not available since there was no more than 1 death among malformed infants in this stratum.

birthweights over 1499 g (Table 3). In contrast, the contribution of anomalies to postneonatal mortality was greatest among very-low-birthweight and moderately-low-birthweight Black and White infants, contributing to approximately 30% of these deaths (Table 3).

Neonatal mortality rate ratios for malformed vs nonmalformed Black and White infants increased greatly with each increase in gestational age (from <28 weeks to 28–36 weeks to ≥37 weeks), and the magnitudes of the increases were similar to those observed with increasing birthweight. For example, among White infants born at 28 through 36 weeks of gestation, mortality was 21.3 times higher for malformed neonates than for nonmalformed neonates (95% CI = 16.5, 27.5), whereas among Whites born at term, mortality was 61.2 times higher for malformed neonates than for nonmalformed neonates (95% CI = 49.6, 75.4). In the postneonatal period, the magnitude of the increases was not as marked, particularly among Blacks.

Except among very-low-birthweight neonates, lower-lethality anomalies continued to have a substantial effect on the neonatal and postneonatal mortality of Black and White infants after stratification by birthweight (Table 4). Further, in the neonatal period, the effect of anomalies on mortality increased with increasing birthweight or gestation for most lethality classes, although the magnitude of the increases was largest for infants with high- or very-high-lethality anomalies, especially among Blacks (Table 4). These latter findings were attributable to a far steeper decline in mortality among nonmalformed neonates with increasing birthweight (e.g., 264.5 per 1000 among very-low-birthweight Whites vs 0.63 per 1000 among normal-birthweight Whites) than among malformed neonates (e.g., among Whites with very-high-lethality anomalies, 815.4 per 1000 for very low birthweight vs 194.5 per 1000 for normal birthweight).

Analysis by Intrauterine Growth Retardation

We lacked sufficient data to examine the relation between anomalies, intrauterine growth retardation, and mortality among Black infants. However, among Whites we assessed mortality rate ratios for neonates with very-high-lethality anomalies vs nonmalformed neonates by using Yerushalmy's classification scheme.[18] The effect of very-high-lethality anomalies on the mortality of moderately-low-birthweight growth-retarded (≥37 weeks) White neonates (RR = 138.3; 95% CI = 77.2, 247.8) was roughly 3 times greater than for moderately-low-birthweight preterm neonates (RR = 46.2; 95% CI = 30.6, 69.9). A similar increase was observed among normal-birthweight neonates. There were no substantial differences in absolute mortality risks for malformed growth-retarded neonates compared with malformed preterm neonates. The observed interactions between growth retardation and very-high-lethality anomalies were due to lower absolute mortality risks among nonmalformed, growth-retarded, moderately-low-birthweight, and normal-birthweight neonates compared with nonmalformed preterm neonates.

The contribution of all anomalies to neonatal mortality among Whites was also higher among growth-retarded moderately-low-birthweight neonates; anomalies contributed to 79.4% (95% CI = 67.0, 88.1) of all neonatal deaths among these infants, compared with 59.6% (95% CI = 48.6, 69.7) among preterm moderately-low-birthweight infants.

Discussion

Clinicians have long recognized that infants born with serious congenital anomalies die more frequently than infants born without anomalies. Nonetheless, our study is the first to assess the magnitude of this increased risk in White and Black infants by using population-based data. We found that, in comparison to nonmalformed infants, the presence of any congenital anomaly diagnosed in the first year after birth increased mortality 9.0-fold for Black infants and 17.8-fold for White infants.

Our estimates are much larger than the 6-fold increased risks noted in the 2 previous registry-based studies that compared mortality among malformed infants to mortality among all infants.[11,12] The comparable rate ratio based on our data is 11.1, still almost double those earlier estimates. Our observed mortality rate among malformed infants (90 per 1000) is considerably higher than that of the New York State study[11] (68.4 per 1000) but similar to that of the Atlanta study[12] (83 per 1000). While the New York registry relies on passive surveillance, the CBDMP and Atlanta registries have very similar ascertainment methods and inclusion criteria.[20] Differences in the effects of anomalies on mortality risks by region may also be due to regional differences in the mortality rates of nonmalformed infants.

Our findings show that anomalies contributed to 33% of all deaths among White infants and 19% of all deaths among Black infants. These findings are 33% to 46% higher than those of previous reports based on underlying-cause-of-death data[1,2] and 14% to 25% higher than those of a study that also used multiple-cause-of-death data.[2] In the postneonatal period, underlying-cause-of-death data appear to especially underestimate (by over 50%) the contribution of anomalies to Black mortality.[2,21]

We considered whether our decision to include deaths among infants with only very-low-lethality or low-lethality anomalies produced overestimates of the true contribution of anomalies to infant mortality. Our data indicated that these lower-lethality anomalies should not be discounted; we found them to be strong predictors of infant mortality, resulting in up to 8.9-fold increased mortality rate ratios, even after controlling for birthweight (Tables 2 and 4).

We classified anomalies by their potential lethality, as opposed to a classification scheme based on organ systems or on purported etiology (e.g., chromosomal abnormalities or congenital infections), because our intent was to reduce heterogeneity with respect to the likelihood of infant death. Anomalies involving the same organ system or having a similar alleged etiology often were assigned to very different lethality classes. While some anomalies might be judged by others to be of a higher or lower lethality than the ones we assigned, we think it is unlikely that anomalies classified as very low lethality would be judged to be of high or very high lethality.

The effects of high-lethality and very-high-lethality anomalies on neonatal mortality were shown to increase more than 100-fold as birthweight increased (Table 4). We considered whether underascertainment of anomalies among very-low-birthweight infants could have contributed to these findings. Since many anomalies may not be diagnosed at birth,[22] and since very-low-birthweight infants often die within the first few hours after birth,[23] underascertainment may have been greater among very-low-birthweight neonates who died. We found, however, that for both Black and White infants, the live-birth prevalence of congenital anomalies increased substantially as birthweight decreased (data not shown); this is similar to the findings of previous studies.[14,15,17] An alternative explanation for the observed differences in mortality rate ratios by birthweight is that there is such a high mortality rate among very-low-birthweight nonmalformed neonates (approximately 25%) that the added presence of even highly lethal anomalies can have little added mortality effect.

Contrary to expectations, we found that anomalies were stronger predictors of postneonatal mortality among infant girls than among infant boys. These larger mortality rate ratios among girls were a function of higher absolute postneonatal mortality risks among malformed girls than among malformed boys, and of lower postneonatal mortality risks among nonmalformed girls than among nonmalformed boys. One possible explanation for this finding is that the general survival advantage for girls may merely postpone mortality among malformed girls to the postneonatal period.

Historically, congenital anomalies have been considered endogenous risk factors that, as such, would exert their greatest influence during the neonatal period.[6] Our data indicate that for moderately-low-birthweight and normal-birthweight Black and White infants, this assumption holds true; however, we found that among very-low-birthweight infants—and especially Black very-low-birthweight infants—anomalies were a stronger predictor of postneonatal mortality (Table 3).

Few studies have been dedicated to understanding the relationship between anomalies and infant mortality. Our study shows that the contribution of congenital anomalies to the mortality of moderately-low-birthweight and normal-birthweight infants is remarkably high for Blacks as well as Whites. Further exploration of the effect of anomalies on infant mortality will require even larger mortality studies that use prevalence data from birth-defects surveillance systems. In addition, our data indicate that if we are to achieve continued reductions in infant mortality in the United States, there is a critical need for more etiologic and prevention research on congenital anomalies in Black and White infants. □

Contributors

L. H. Malcoe, G. M. Shaw, and E. J. Lammer conceived the study. Malcoe, Shaw, Lammer, and A. A. Herman all contributed to analyzing and interpreting the data and writing the paper.

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