Medical Hypotheses

Racial Differences in Pregnancy Duration and its Implications for Perinatal Care

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Abstract — The average length of gestation is about 5 days shorter in black populations than in white populations. Although some of this difference is accounted for by higher preterm delivery rates in blacks, the most common gestational week of delivery at term is the 39th in black populations, the 40th in white. Black gestational age specific neonatal mortality is lower than that of whites until the 37th week of gestation, but higher thereafter.

These observations suggest the hypothesis that complications of postmaturity occur sooner in black fetuses. If this hypothesis is confirmed, antepartum surveillance for signs of fetal compromise should be initiated earlier in gestation in black parturients, perhaps by setting the estimated date of confinement at 275 days after the LMP, rather than the conventional 280 days.

Racial difference in pregnancy duration

The average duration of pregnancy has been thought to be 280 days since Reid's paper on the subject in 1850 (1). However, inferences about the average and optimum duration of human pregnancy have been based almost entirely on observations made in white populations (1 – 4). Pregnancy duration in different racial and ethnic groups has not been extensively studied, but published reports from a variety of populations, providing mean gestational age data by race (Table), suggest a shorter mean pregnancy duration in black populations, usually by 3 – 7 days (3 – 12).

Gestational age distributions in two US populations

Figure 1 plots the distribution of live births by gestational week of birth in two very disparate United States populations, one northern, urban and industrial (New York City, 1976 – 1978), the other southern, rural and agricultural (South Carolina,

Table 1 Studies describing gestational age in both black and white populations. Mean gestational age in days

Author	Year	Blacks	Whites	White-Black difference	Population
Meyer (3)	1915	276.9	279.8	2.9	Baltimore
Hotelling (4)	1932	276.3	281.4	5.1	Stanford
Anderson (5)	1943	275.3	280.7	5.4	Cincinnati
Taback (6)	1951	269.8	279.4	9.6	Baltimore
Erhardt (7)	1964	271.6	278.6	7.0	NY City
Westphal (8)	1964	272.2	280.1	7.9	US Multi-Center Study
Henderson (9)	1967	263.6	272.1	8.5	Baltimore
Tuck (10)	1983	275.1	278.6	3.5	London
Papiernik (11)	1986	270.8	275.8	5.0	Clamart,
		270.0	275.6	5.6	France
(SES Groups)		273.0	275.9	2.9	
		270.3	273.7	3.4	
Alexander (12)	1987	273.7	279.3	5.6	N & S Carolina
(Lo/Hi Risk)		268.1	276.5	8.4	
These data					
1976 - 78		271.0	275.4	4.4	NYC
1975 - 84		269.5	277.2	7.7	S Carolina

[1] The small number of Asian and Native American births in these two populations have been included with white births, hence, the term non-black.

Gestational Age Distribution by Race
Live Births: NYC 1976-78 & SC 1975-84
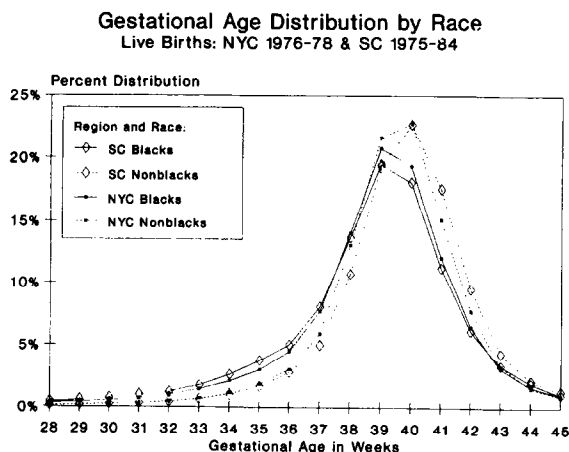
Fig. 1

1975 – 1984). In these two large and complete vital record data sets (about 750,000 live births), the pattern of the gestational age distribution between the two black sub-populations is very similar, and distinctly different from the pattern in the corresponding non-black sub-populations[1].

In each of these two populations, black births occur more commonly than non-black births at each gestational week prior to the 39th week. In the black sub-populations, approximately seven percent fewer births occur in the 40th week than in the 39th (7.2% fewer in South Carolina, 6.7% fewer in New York City). By contrast, between the 39th and 40th gestational week, births *increase* in the non-black sub-population by 17.6 percent in South Carolina and by 5.1 percent in New York City. After the 40th week, births decline in both populations, but more precipitously in the black sub-populations. The common belief that the modal gestational week of birth is the 40th is true only in non-blacks; for black births, the modal week of delivery is the 39th week of gestation in both South Carolina and New York City.

Gestational age-specific mortality

At gestational ages classified as preterm (<37 weeks), black neonatal mortality has frequently been found to be lower than that of whites (8, 9, 12, 13, 14). An examination of gestational age specific neonatal mortality in South Carolina and New York City reveals findings consistent with these reports (Fig. 2). In the preterm range, black neonatal mortality is lower than than that of non-blacks. These relationships change distinctly at 37 weeks. From that point on, black neonatal mortality is higher in each gestational week.

To illustrate further the differences in the relationship of mortality to gestational age in black and non-black infants, we plotted the neonatal
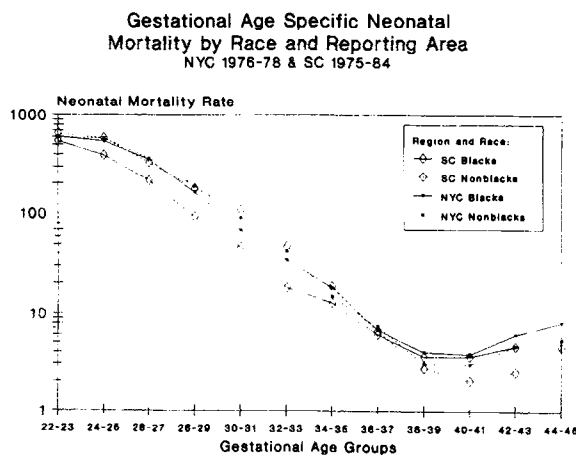
Gestational Age Specific Neonatal
Mortality by Race and Reporting Area
NYC 1976-78 & SC 1975-84

Neonatal Mortality Rate

Region and Race:
-⊕- SC Blacks
◇ SC Nonblacks
— NYC Blacks
• NYC Nonblacks

Gestational Age Groups

Fig. 2

mortality in relation to 'standardized' (Z-score) gestational age (Fig. 3). In this figure, data for the black and non-black sub-populations in both regions have been combined. The 'standardized' gestational age is a measure of the deviation of the gestational age of each infant from the mean gestational age of the racial sub-population to which it belongs. This deviation is expressed in terms of the number of standard deviations from the mean gestational age. Thus, in Figure 3, zero represents the mean gestational age in each sub-population, and the position labelled -1 represents gestational ages one standard deviation shorter than the mean.
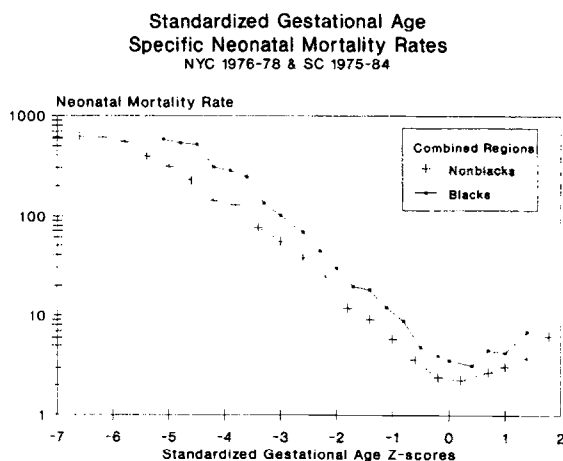
Standardized Gestational Age
Specific Neonatal Mortality Rates
NYC 1976-78 & SC 1975-84

Neonatal Mortality Rate

Combined Regions
+ Nonblacks
— Blacks

Standardized Gestational Age Z-scores

Fig. 3

When the two sub-populations are compared, it can be seen that for each degree of deviation from

the gestational age mean, mortality is higher in black infants, indicating a generally higher force of mortality operating among black infants at all standardized gestational ages. But for each racial group, the lowest risk of neonatal mortality occurs slightly beyond the mean gestational age. Inasmuch as that mean is earlier in the black sub-populations, mortality rises at an earlier gestational age in black infants (as seen in Fig. 2).

Intrinsic or extrinsic differences?

One interpretation of these observations is that black populations have an inherently shorter duration of gestation than do white populations and mature faster in utero. Mortality then is lower in black preterm infants of the same early gestational age because the same gestational age is less deviant in black populations. By the same token, mortality is higher at later gestational ages in blacks because these ages are correspondingly more deviant than they are in white populations. Indeed, the gestational-age specific mortality rates in Figures 2 and 3 appear to be the same curves in each race, but shifted one week earlier in the black population. Support for this hypothesis derives from studies suggesting earlier maturation in black fetuses and infants in several biological parameters (15, 16). A very similar interpretation has been made of birth weight distributions and birth weight specific mortality in black and white populations (14, 17).

It is important to note that an inherent racial difference in gestational duration is not the only possible explanation for these observed racial variations. Several lines of evidence point to biological stressors capable of accelerating maturation. Infants whose mothers experienced hypertension or pre-eclampsia have been reported to have lower rates of hyaline membrane disease, and germinal matrix hemorrhage (18 – 20). Premature rupture of membranes has also been found to accelerate the maturation of the fetal lung (18, 21). Endogenous adrenal secretion of surfactant may be a mechanism for this effect, since administration of such agents matures the fetal lung (22).

Black infants demonstrate earlier lung maturation and lower rates of hyaline membrane disease than whites (23 – 25). Moreover, higher levels of pre-eclampsia, premature rupture of the membranes and hypertension are also observed among black populations (10, 26). Thus the lower mortality for gestational age among preterm black infants may be a response to exposure to more

stressful environments in utero than those encountered by white fetuses. The observation of higher mortality for black infants at each *standardized* gestational age interval further supports this hypothesis.

The shorter gestational interval in black infants may result, in this model, either directly from the accelerated maturation of the black fetus in response to stress, or because the excessive rates of pregnancy complications promote early expulsion of the fetus from a less than optimum environment. Since we have found that the entire gestational age distribution is different in black infants, the former hypothesis seems more compelling.

Maturation, aging and senescence in the feto-placental unit

Optimal healthy birth takes place during a relatively short window of time that lasts about six weeks. Delivery on either side of this time span can result in serious illness in the newborn. While the consequences of preterm birth constitute by far the most serious hazard to perinatal health, delivery much later than the mean expected time of delivery is also associated with excess morbidity and mortality (27 – 35). Aging begins at conception, and post-maturity may perhaps be viewed as the first clinical manifestation of aging in humans. The physiologic derangements in the post-mature fetus and infant appear to emerge after the 38th week of gestation in white populations (36). In Ballantyne's memorable phrase 'the placenta is senile before the infant is born' (27).

Early studies emphasized the impact of post-maturity on perinatal mortality, especially antepartum stillbirths and neonatal deaths due to aspiration pneumonia (27 – 29). Recent authors have found less impact of post-maturity on perinatal mortality, although a very recent report has described a significant excess in antepartum stillbirths (30). The major emphasis in recent papers has been on the higher rates of intrapartum and neonatal morbidity found in post-mature infants (31 – 35). These include meconium passage, fetal distress, and shoulder dystocia, which are paralleled by higher rates of both forceps delivery and Caesarean section. In the neonate, depressed Apgar scores and meconium aspiration syndrome are increased in frequency, as are rates of endotracheal intubation and admission to special care.

Impairment of fetal gas exchange in the aging placenta may account for the higher risk of unexplained antepartum stillbirth in pregnancies that persist substantially beyond term, as well as the higher frequency of labors complicated by fetal distress. The high frequency of meconium passage in utero may result either from the increased peristaltic activity of a more mature fetus or from fetal distress. In either case, the risk of meconium pneumonitis will be raised when intrauterine distress is accompanied by fetal gasping.

The dysmature infant, with long fingernails, cracked and peeling skin, hyper-alert appearance, meconium staining of skin and cord, and, in severe cases, loss of subcutaneous fat and wasted appearance was first described in detail by Clifford in 1954 (28). Severely dysmature infants are still seen on occasion (33).

Several protocols have been proposed for the management of pregnancies in which spontaneous labor has not occurred by some fixed period after the expected date of confinement (30, 32, 35, 37, 38). All of these protocols are characterized by increased surveillance of fetal well-being followed by intervention to terminate pregnancy, either in response to a test casting doubt on fetal well-being or to an extended elapsed interval since the expected date of confinement. The appropriate time to initiate these interventions, as well as the surveillance that precedes them, is directly dependent on the expected duration of pregnancy, knowledge of the rate of aging of the human placenta, and, in particular, the time of onset of its senescence. The appearance of higher neonatal mortality in black infants than in whites at about 37 – 38 weeks and the continuation of this increased mortality risk suggest that post-maturity and its related conditions occur sooner in black populations.

Clinical implications

The data analyzed in this paper describe both an earlier modal gestational age in black infants and an earlier rise in mortality thereafter. The clinical implication that follows from these observations is that complications of post-maturity occur sooner in black infants. Unfortunately, the clinical consequences of post-maturity are not well described on death certificates. Most of the morbidity of postmaturity does not result in neonatal death, and where it does, the listed principal cause (e.g. pneumonia) may not specifically reflect pathology attributable to postmaturity. Thus, further investigation of the question of whether post-maturity complications occur sooner in black populations

will require analysis of clinical data sets in which these complications are recorded.

Regardless of the underlying cause, the clinical implications are the same. Based on serial reporting over this century, the average length of gestation appears to be approximately 5 days shorter in black populations than in white populations. The commonest gestational week of delivery in our data is the 39th in black populations and the 40th in whites. Correspondingly, the optimal gestational age in terms of neonatal survival occurs earlier in black infants. These observations suggest that aging occurs earlier in the black feto-placental unit and that black fetuses experience an earlier onset of complications of post-maturity. Antepartum surveillance for signs of fetal compromise should therefore be initiated earlier in gestation in black parturients, perhaps by setting the estimated date of confinement at 275 days after the LMP, rather than the conventional 280 days. In populations with higher levels of socio-economic and medical risk, earlier monitoring and awareness of potential abnormal conditions is both prudent and warranted.

References

1. Reid J. On the duration of pregnancy in the human female. Lancet 1850; i: 438 – 440, 596 – 599; ii: 77 – 81.
2. Karn MN. Length of human gestation with special reference to prematurity. Ann Eugenics 1947; 14: 44 – 59.
3. Meyer AW. Fields, graphs and other data on fetal growth. Contributions to Embryology, No 4, Pub No 222, Carnegie Institute of Washington, 1915.
4. Hotelling H, Hotelling F. A new analysis of duration of pregnancy data. Am J Obstet Gynec 1932; 23: 643 – 657.
5. Anderson NA, Brown EW, Lyon RA. Causes of prematurity III. Influence of race and sex on duration of gestation and weight at birth. Am J Dis Child 1951; 65: 523 – 534.
6. Taback M. Birth weight and length of gestational with relation to prematurity. JAMA 1951; 146(10): 897 – 901.
7. Erhardt C, Joshi G, Nelson F, et al. Influence of weight and gestation on prenatal and neonatal mortality. Amer J Pub Health 1964; 54: 1841 – 55.
8. Westphal MC, Joshi GB. The interralationship of birth weight, length of gestation and neonatal mortality. Clin Obstet Gynecol 1964; 7: 670 – 686.
9. Henderson M, Kay J. Differences in duration of pregnancy. Arch Environ Hlth 1967; 14: 904 – 11.
10. Tuck SM, Cardozo LD, Studd JWW, Gibb DM. Obstetric characteristics in different racial groups. Brit J Obstet Gyn 1983; 90: 892 – 7.
11. Papiernik E, Cohen H, Richard A, et al. Ethnic difference in duration of pregnancy. Ann Hum Biol 1986; 13: 265.
12. Alexander GR, Cornely D. Racial disparities in pregnancy outcomes: The role of prenatal care utilization and maternal risk. Am J Prev Med 1987; 3(5): 254 – 261.
13. Sappenfield WM, Buehler JW, Binkin NJ, Hogue CJR, Strauss LT, Smith JC. Differences in neonatal and post-neonatal mortality by race, birth weight and gestational

age. Pub Hlth Rep 1987; 102(2): 182 – 192.
14. Alexander GR, Tompkins ME, Altekruse JM, Hornung CA. Racial differences in the relation of birth weight and gestational age to neonatal mortality. Pub Hlth Rep 1985; 100(5): 539 – 47.
15. Geber M, Dean RFA. The state of development of newborn African children. Lancet 1957; 272(i): 1216 – 9.
16. Parkin JM. The assessment of gestational age in Ugandan and British newborn babies. Devel Med Child Neurol 1971; 13: 784 – 88.
17. Wilcox AJ, Russell IT. Birth weight and perinatal mortality: III. Towards a new method of analysis. Intl J Epid 1986; 15(2): 188 – 96.
18. Lee KS, Eidelman AL, Tseng PL, et al. Respiratory distress syndrome of the newborn and complications of pregnancy. Pediatrics 1976; 58: 675 – 80.
19. Yoon JJ, Kohl S, Harper RG The relationship between maternal hypertensive disease of pregnancy and the incidence of idiopathic respiratory distress syndrome. Pediatrics 1980; 65: 735 – 39.
20. Leviton A, Kuban KC, Pagano M, et al. Maternal toxemia and neonatal germinal matrix hemorrhage in intubated infants less than 1751 g. Obstet Gynecol 1988; 72: 571 – 76.
21. Thibeault DW, Emmanulides GC. Prolonged rupture of fetal membranes and decreased frequency of respiratory distress syndrome and patent ductus arteriosus in preterm infants. Am J Obstet Gynecol 1977; 129: 43 – 46.
22. Liggins GC, Howie RN. A controlled trial of antepartum glucocorticoid treatment for prevention of the respiratory distress syndrome in premature infants. Pediatrics 1972; 50: 515 – 25.
23. Olowe SA, Akinkugbe A. Amniotic fluid lecithin/sphingomyelin ratio: Comparison between an African and a North American community. Pediatrics, 1978; 62(1): 38 – 41.
24. Fujikura T, Froehlich LR. The influence of race and other factors on pulmonary hyaline membranes. Am J Obstet Gynec 1966; 95(4): 572 – 8.
25. Farrell PM, Wood RE. Epidemiology of hyaline membrane disease in the United States: Analysis of National Mortality Statistics. Pediatrics 1976; 58(2): 167 – 76.
26. Flood B, Naeye RL. Factors that predispose to premature rupture of the fetal membranes. JOGN Nursing 1984; 119 – 22.
27. Ballantyne, JW. The problem of the post-mature infant. J Obstet Gynaecol Br Emp 1902; 2: 521 – 554.
28. Clifford SH. Post-maturity with placental dysfunction. J Pediatr 1954; 44: 1 – 18.
29. McClure-Browne JC. Post-maturity. Am J Obstet Gynecol 1963; 85: 573 – 582.
30. Eden RD, Seifert LS, Winegar A, Spellacy WN. Perinatal characteristics of uncomplicated postdate pregnancies. Obstet Gynecol 1987; 69(3,Part 1): 296 – 9.
31. Zwerdling MA. Factors pertaining to prolonged pregnancy and its outcome. Pediatrics 1967; 40(2): 202 – 12.
32. Vorherr H. Placental insufficiency in relation to postterm pregnancy and fetal postmaturity. Am J Obstet Gynec 1975; 123(1): 67 – 103.
33. Sachs BP, Friedman EA. Results of an epidemiological study of postdate pregnancy. J Reprod Med 1986; 31(3): 162 – 66.
34. Boyd ME, Usher RH, McLean FH, Kramer MS. Obstetric consequences of postmaturity. Am J Obstet Gynec 1988; 158(2): 334 – 8.
35. Shime J, Gare DJ, Andrews J, Bertrand M, Salgado J,

Whillans G. Prolonged pregnancy: Surveillance of the fetus and the neonate and the course of labor and delivery. Am J Obstet Gynec 1984; 148(5): 547 – 51.
36. Bouw GM, Stolte LAM, Baak JPA, et al. Quantitative morphology of the placenta. II. The growth of the placenta and the problem of post-maturity. Europ J Obstet Gynecol Reprod Biol 1978; 8: 31 – 42.
37. Schneider JM, Olson DM, Curet CB. Screening for fetal and neonatal risk in the postdate pregnancy. Am J Obstet Gynec 1978; 131(5): 473 – 8.
38. Lagrew DC, Freeman RK. Management of postdate pregnancy. Am J Obstet Gynec 1986; 154(1): 8 – 13.