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THE PREMATURE AND THE NEWBORN

Twin Survival: Comparison of Mortality Rates of First and Second Twin. Most standard textbooks of obstetrics state, in principle, that apart from ascertaining a longitudinal lie of the second twin little else should be done to aid its delivery. This attitude of *laissez faire* has probably caused death of the second twin in many instances. To test this, James McD. Corston[1] (Dalhousie Univ.) studied 211 consecutive cases of twin pregnancy.

Of 422 babies delivered, 57 died (stillbirths and neonatal deaths). Almost twice as many second twins as first twins died—15 and 8, respectively. Further, in 17 instances both twins died prenatally or neonatally. In this group 13 were stillborn and 21 died within the first week. All these cases occurred in pregnancies terminated before the 8th month of gestation and 2 babies were delivered in the 5th month.

To determine why almost twice as many second as first twins died, the author examined the interval between delivery of the first and of the second twins and the method of delivery. He concludes that risk to the second twin can be diminished by avoiding delay in delivery between the first and second twin. The maximum interval should not exceed 15 minutes. Aided delivery of the second twin does not seem to affect its chance of survival. In fact, by assisting the birth of the second twin and thereby decreasing the interval, the baby may be protected from intrapartum death due to anoxia.

Postmature Baby. Beatrice E. Tucker and Harry B. W. Benaron[2] (Northwestern Univ.) studied 4,106 first-born infants, of which 832 were white and the rest Negro. Postmaturity is defined in terms of gestational age as calculated from the 1st day of the last menstrual period. A baby is postmature if the gestational age is 43 weeks or over. First-born babies were selected for study because the menstrual history of primiparous women is less erratic than that of multiparous ones.

(1) Obst. & Gynec. 10:181-183, August, 1957.
(2) Am. J. Obst. & Gynec. 73:1314-1320, June, 1957.

The incidence of postmaturity was 9% in the white and 6% in the Negro group. The perinatal (intrauterine and neonatal) mortality rate in the mature white group was 1.4%; in the postmature white group, 1.3%. In the mature Negro group it was 2.3% and in the postmature Negro group, 3.1%. Two small babies died of malnutrition and anoxia during labor and the placenta of each showed changes that might have been attributed to aging.

There were 5% fewer small babies (2,500 Gm.) in the postmature white group than in the mature, and 6% fewer small babies in the postmature Negro group than in the mature. In both the mature and postmature white groups the largest number of babies were in the 3,200-Gm. division. There was a 1% increase in 3,200-Gm. babies and a 10% increase in babies weighing 3,700 Gm. and over in the postmature over the mature white group. In the mature Negro group the largest number of babies were in the 2,700-Gm. division, whereas in the postmature Negro group the largest number were in the 3,200-Gm. division. There was a 13% increase in 3,200-Gm. babies and a 3% increase in babies weighing 3,700 Gm. and over in the postmature over the mature Negro group.

In a group of 1,507 large babies (4,500 Gm. and over) born of mothers of all races and parities the incidence of postmaturity was 15% as compared with 6% for average-sized babies. Of white premature babies classed by weight, 27% were considered mature when classed by gestational age. Of mature babies classed by weight, 28% were considered premature when classed by gestational age.

The authors conclude there is a high degree of error in determining fetal development by gestational age and any conclusion drawn from a study such as the present one must be evaluated accordingly. There is no statistically significant difference in the perinatal mortality rates of mature and postmature infants. Prolonged gestation is rarely accompanied by placental changes leading to malnutrition, anoxia and death. Postmature babies tend to be larger than mature babies. The postmature infant requires no different management than the mature infant.

► [Because of our increasing confusion over the conflicting opinions re-

garding the perinatal mortality rate associated with postmaturity, we asked Dr. Stewart H. Clifford to comment:

"Postmaturity per se is a very common condition; of all pregnancies, 25% extend to 41 completed weeks of gestation, 12% to 42 weeks and 5% to 43 completed weeks or over. As to etiology, to some extent postmaturity might be considered a natural and normal process; if one plots the incidence of pregnancies terminating at various gestational ages, on the one side is prematurity with an incidence of 6% and on the other postmaturity with an incidence of 5%, the resulting parabola resembles the curve of normal distribution. There is the hormonal theory of origin due to the failure of progesterone to fall. Others consider a constitutional and hereditary factor present. An endocrine disorder in the mother causing genital infantilism has been implicated. Probably the most important single cause is some condition that does not permit the normal descent of the head into the pelvis—malpositions, transpositions or occiput posteriors, cephalopelvic disproportion, tumors, etc.

"The latter point is very important; in my series of postmature fetal deaths at the Boston Lying-in occurring from 1942 through 1954, 25% died before onset of labor, 75% died during labor, and in 80% of these there was abnormality of fetal position or pelvic structure and a high incidence of uterine inertia and prolonged labor. Average length of labor was 40 hours.

"Of all our postmature fetal deaths, 75% of them occurred between 1942 and 1948, at which time there was a sharp change in the method of obstetric management, and instead of allowing nature to more or less take its course, a definite attempt to stop prolonged labor was instituted. After 8-10 hours of labor, every case had to be reappraised and the decision made then as to the need for interruption and the program for further action determined. I am sure that our current incidence of perinatal mortality is in the range reported by Tucker and Benaron—in our case one of the major problems of postmaturity was solved unexpectedly through an attack on the problem of prolonged labor.

"Postmaturity with placental dysfunction is the syndrome of my particular interest and a major reason for the confusion in the literature is that the distinction is not made between simple postmaturity and this condition. With an adequate placenta, postmaturity is well tolerated and may even be beneficial. It is in the fetus with postmaturity plus an inadequate placenta that malnutrition and anoxia may occur. An inadequate placenta may be encountered in any postmature infant no matter what the cause of postmaturity; when it coexists the situation is serious; fortunately, this combination occurs relatively rarely. An inadequate placenta, even more infrequently, may be encountered at term with a small infant showing the effects of malnutrition and anoxia at birth.

"The problem is how to detect the prolonged pregnancy with placental dysfunction. We have several leads; it is more apt to occur in the primipara and more frequently in the 'elderly primipara' or essential primipara. When it occurs, the amniotic fluid is greatly diminished and the uterus may shrink; the mother may lose weight. The amniotic fluid will be meconium stained if the dysfunction has proceeded to the anoxic phase.

"Tucker and Benaron have a remarkable series for modern times, for they state that all their cases are home deliveries. I would think this in itself would imply a certain selection of cases; it would not appear reasonable that a complicated case would be allowed home delivery and that prolonged labor or other complications during labor would not be trans-

ferred to hospitals. In any event, I know of no recent comparable series.

"The only criticism one could suggest as to their perinatal mortality figures in postmaturity would be because of the size of the series. In 4,106 cases, they saw 267 postmatures in 10 years or 27 a year. I reviewed the literature and collected reports on 105,000 births, of which 5,455 were 3 weeks or more postmature; the perinatal mortality rates were all between 40 and 120/1,000. Lindell studied 46,381 births in Stockholm and Uppsala, of which 1,219 were 3 weeks or more postmature; the perinatal mortality rate was 70/1,000. It is important to realize that these high mortalities can and have been reduced. As stated above, the better management of prolonged labor is an important approach. Since the occurrence of postmaturity may alert the obstetrician to the presence of an abnormal fetal position or pelvis and since a very small number of prolonged pregnancies may also have an abnormal placenta, I cannot agree with the authors that there is no difference in managing the mature and postmature pregnancy."

Clifford, S. H.: Postmaturity, in Levine, S. Z. (ed.): *Advances in Pediatrics* (Chicago: Year Book Publishers, Inc., 1957), vol. IX, pp. 13-58.

Lindell, A.: Prolonged pregnancy, Acta obst. et gynec. scandinav. 35: 136, 1956.—Ed.]

Dysmaturity is a term suggested by S. Sjöstedt, G. Engleson and G. Rooth[3] (Univ. of Lund) to denote the placental insufficiency syndrome, to prevent confusion between the terms prolonged pregnancy and postmaturity. Investigation was made of 1,171 newborn infants. They were classified on the basis of examination without reference to duration of pregnancy, delivery, amniotic fluid or placenta. Normal and premature infants without signs of dysmaturity were grouped in stage 0. Infants in stage 1 had cracked, dry, peeling skin and thin arms and legs. They were more awake and alert than usual, but some showed respiratory distress or a tendency to vomit. When there was doubt concerning early signs of dysmaturity, the infants were classed in stage 0-1. Infants exhibiting the signs attributed to stage 1 to a marked degree and who had thin trunks were classified in stage 2. In stage 3 the trunk and extremities were extremely thin and the infants appeared dystrophic. The skin peeled in large flakes. The nails and skin were yellowish.

The frequency of dysmaturity in this series was 16.7%. The advanced stages 2 and 3 were seen in only 1.8% of the infants (Table 1). Dysmaturity increased in frequency with prolonged pregnancy, but was also seen in infants born at term. Incidence of dysmaturity was greater for primiparas

(3) Arch. Dis. Childhood 33:123-130, April, 1958.

and male infants. There was no increasing frequency of dysmaturity with advancing age of the mother (Table 2).

Both normal and dysmature infants increase in weight during gestation, but from the 39th week until delivery the dysmature infants have a mean birth weight 200 Gm. below that of normal infants (Fig. 1). The infant's length increases irrespective of dysmaturity until and including the 43d week.

TABLE 1.—Stage and Percentage Distribution of Dysmaturity in 444 Newborn Infants

Stage of Dysmaturity					
0	0–1	1	2	3	1–3
58·6%	24·8%	14·9%	1·6%	0·2%	16·7%

TABLE 2.—Percentage Distribution of Maternal Age

Maternal Age	Stage of Dysmaturity			
	0	0–1	1	2+3
	%	%	%	%
<20	7·0	6·2	7·5	8·9
20–24	22·8	22·4	27·5	17·8
25–29	32·9	35·0	31·0	29·0
30–34	23·5	22·0	19·3	22·2
35–39	10·8	12·3	13·0	15·6
≥40	2·8	1·9	1·6	6·7

As dysmaturity becomes increasingly severe, oxygen saturation in cord blood decreases, whereas hemoglobin, plasma pentoses, protein-bound hexoses, bilirubin and nonprotein nitrogen increase.

The reduced weight, hypoxia, increased hemoglobin and possibly also the increased nonprotein nitrogen indicate a reduction in the diffusion capacity of the placenta. The increase in bilirubin may be a sign of the increased blood formation and a concomitant increased turnover of hemoglobin pigments. It may also result from hypoxic disturbances in liver function. The frequency of albumin in the urine of the newborn infant increases with the degree of dysmaturity, showing that such infants also have impaired renal function. Frequency of glycosuria also increases with the degree of dysmaturity.

This study shows the importance of distinguishing between the gestation time of the infant and the state of the infant, i.e., whether the infant is normal or dysmature. Prolongation of pregnancy per se does not have untoward effects on the infant. Labor should be induced if the infant

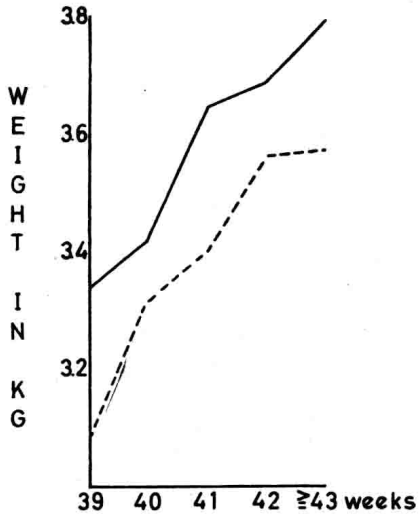

Fig. 1.—Weight curves of normal and dysmature infants. Solid line: normal infants, stages 0 and 0-1; broken line: dysmature infants, stages 1, 2 and 3. (Courtesy of Sjöstedt, S., *et al.;* Arch. Dis. Childhood 33:123-130, April, 1958.)

is dysmature, but this cannot be verified as a rule until after the child is born. If the Ballantyne-Runge sign is present (decrease in circumference of the abdomen late in pregnancy due to decreased amount of amniotic fluid), the infant is likely to be dysmature. Fetal ECG studies also indicate a possible method of diagnosing dysmaturity before birth.

► [Dr. Stewart H. Clifford was asked his opinion of diminishing circumference of the abdomen and fetal ECG as aids to the determination of dysmaturity. He was also asked if examination of the amniotic fluid by tapping would be helpful. He commented as follows:

"Unfortunately, there is no reliable way to know before the birth of the child that prolongation of pregnancy is accompanied by placental dysfunction. There are some suggestive findings, one of which was mentioned, i.e., the reduction in the circumference of the abdomen. To this could be added reduction in the distance between the symphysis and height of the fundus. Walker feels that sudden loss in weight on the part of the mother is also indicative of absorption of amniotic fluid secondary to placental dysfunction.

"We have explored the possibility of abdominal tap to discover the nature of the amniotic fluid in several cases. In one, a dry tap was obtained, and a repeat tap at cesarean section was also dry and as soon as the uterus was opened, the reason was apparent because there was almost complete lack of amniotic fluid except in a small pocket at the fundus. Dr. Walker advocates tapping of the hind water in these questionable cases. He uses a long curved catheter which he inserts up to the fundus and then obtains a sample of the amniotic fluid from this region. If it is normal, he does nothing; if it is meconium stained, he advocates immediate cesarean section. By this technic, he believes he has markedly reduced fetal mortality. This method has not been accepted in this country, and I do not know of a single clinic that uses it.

"The most valuable help in this connection is from the statistical material which allows you to disregard the multiparas who are postmature, as the incidence of mortality and morbidity in their children is no different from that in the normal population. On the other hand, the occurrence of postmaturity in an elderly primipara is extremely significant. In my experience, most postmature women are postmature because of some malposition of the fetal head or some factor that prevents the fetal descending into the pelvis in its normal manner. About 80% of our postmature women had some mechanical factor explaining why the pregnancy was prolonged. The most common finding is occiput-posterior position or a transverse position, cephalopelvic disproportion, or obstructions to the entrance of the head into the pelvis. In my opinion, the most important factor about postmaturity is that when it occurs the obstetrician should reappraise the entire situation and decide whether or not there are any abnormal factors responsible for this postmaturity. If postmaturity is also accompanied by a failing placenta, then you get the various stages of placental dysfunction syndrome that I have previously described."

Dr. Sjöstedt was asked if examination of the amniotic fluid would help in establishing a diagnosis of dysmaturity. He replied as follows:

"Often the amount of amniotic fluid is very small when the infant is dysmature and for that reason it may be difficult or even impossible to puncture the amniotic sac with success. If I succeed in obtaining amniotic fluid and find meconium staining, I take this as an indication to induce labor or sometimes to do a cesarean section."—Ed.]

Neonatal Anemia Due to Fetal Hemorrhage into Maternal Circulation. H. H. Gunson[4] (Univ. of Toronto) reports a case. When the infant was aged 4 hours, the concentration of hemoglobin in the capillary blood was 9.5 Gm./100 ml. The blood groups differed from the mother's in both ABO and Rh systems and fetal erythrocytes were demonstrated in the maternal circulation by differential agglutination. Ashby counts on the mother' blood showed fetal cells persisted in the maternal circulation for about 80 days, after which the mother developed Rh antibodies. Slightly increased numbers of reticulocytes and nucleated erythrocytes were present in the child's peripheral blood, proving that active erythropoiesis was occurring.

(4) Pediatrics 20:3-6, July, 1957.

Occult fetal hemorrhage in utero, as distinct from a massive fetal hemorrhage immediately before birth leading to a newborn infant in shock, may be clinically suspected in pale infants with a low concentration of hemoglobin during the 1st day of life. The anemia is not progressive and the infants do not become jaundiced, which differentiates the condition from hemolytic disease of the newborn.

Anemia in newborn infants subsequent to hemorrhage from the placenta may occur often. The blood groups of mother and infant must have certain differences before the differential agglutination test can be used to detect fetal cells in the mother's blood. But given this difference and with careful technic, a relatively small proportion of fetal erythrocytes can be demonstrated. In some cases failure to make an absolute diagnosis may be due to hemorrhage remaining localized near the placental site and not entering the general circulation of the mother. Another possibility is that fetal cells may be destroyed as they enter the maternal circulation which would be expected with group O mothers who have group A or B babies, as the mother's serum normally contains anti-A and anti-B agglutinins.

In the present case, antibody formation in the mother to both the D and E antigens of the child was demonstrated. This supports the theory that fetal hemorrhage into the mother's circulation may be one mechanism of Rh isoimmunization as a result of pregnancy. In most cases in which the mother forms antibodies, the quantity of fetal blood entering the maternal circulation is probably small and causes no significant anemia in the child.

Shock in Newborn Caused by Transplacental Hemorrhage from Fetus to Mother is reported by Jack G. Shiller[5] (Columbia Univ.). Diagnosis was based on the following findings. At birth the infant was pale, hypotonic and lethargic. The concentration of hemoglobin determined by heel prick immediately after birth was 15.1 Gm.; 3 hours later it was 13.1 Gm. and bilirubin was 2.5 mg. Hemolytic disease of the newborn had to be considered because there was incompatibility of Rh and ABO factors. Hemolytic disease was considered unlikely because of a negative Coombs test, absence

(5) Pediatrics 20:7-12, July, 1957

of spherocytosis and the course of the disease. No hemolytic process of any sort was demonstrated. In the immediate postnatal period the distinction between shock from splanchnic vasodilatation and shock from loss of blood is difficult. The concept of blood loss in this case was supported by the amount of blood necessary to stabilize the concentration of hemoglobin at 16 Gm. After transfusion of an amount of blood equivalent to one-third the predicted blood volume of the infant, the concentration of hemoglobin rose only to 16 Gm.

Three hours post partum the concentration of fetal hemoglobin in the mother's peripheral blood was 2.4%, indicating antepartum transfer of blood from fetus to mother. At 2 weeks post partum the concentration was 0.28%, thus providing further evidence for prior significant elevation. The concentration of bilirubin in maternal serum was 2.8 mg./100 ml. 3 hours after delivery, and 24 hours later was 0.7 mg. The concentration of bilirubin in the serum of 9 normal mothers 3 hours after delivery ranged from 0.2 to 1.1 mg. In the present case the mother's anti-A titer rose over the 3 postpartum months, then fell.

The most convincing evidence of transplacental hemorrhage from fetus to mother was found in microscopic sections of the placenta which showed nucleated fetal erythrocytes in the maternal vascular spaces. It is suggested that this finding be checked in similar cases and in a suitable number of controls. Transplacental hemorrhage from fetus to mother may be suspected if the mother shows jaundice just before delivery or shows a systemic reaction suggestive of transfusion of incompatible blood. The treatment of choice is simple transfusion of the infant with recommended quantities of whole blood, repeated if necessary, until the clinical appearance improves and satisfactory concentrations of hemoglobin are maintained.

► [It is quite clear from the increasing number of reported cases of neonatal anemia due to hemorrhage into the mother's circulation that we have failed to diagnose this condition properly in the past. It is obvious that the diagnosis must be considered in any pale, anemic newborn and that prompt transfusion may save these infants. The authors point out that there may be other clues to aid in the diagnosis, such as chills in the mother and/or jaundice before delivery or shortly after birth of the child, indicating that fetal blood has entered the mother's circulation and

produced a transfusion reaction. Further, obstetricians must be alert to the possibility in mothers with massive blood loss per vaginam; the blood should be tested for fetal hemoglobin to determine whether or not the infant, too, is losing blood.—Ed.]

Measurement of Fetal Hemoglobin in Newborn Infants: Correlation with Gestational Age and Intrauterine Hypoxia. Charles D. Cook, Hugh R. Brodie and David W. Allen[6] (Harvard Med. School) determined the percentage of fetal hemoglobin in 152 infants within 4 hours of birth. All but 2 of 29 infants of less than 36 weeks' gestation had more

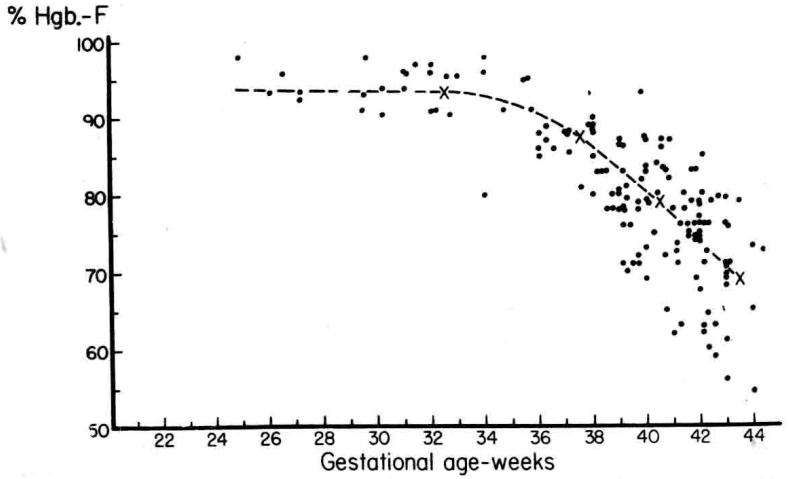

Fig. 2.—Percentages of fetal hemoglobin present at birth in relation to gestational age in 152 infants of 25-44 weeks' gestation. Broken line represents mean percentages. (Courtesy of Cook, C. D., *et al.*: Pediatrics 20:272-278, August, 1957.)

than 90% fetal hemoglobin, and all but 3 of 123 infants of 36 weeks' or more gestation had less than 90% fetal hemoglobin (Fig. 2). In infants of more than 34 weeks' gestation, there was an inverse relation between gestational age and percentage of fetal hemoglobin, the average fetal hemoglobin being less by about 2.5-4% for each further week of gestation. This is similar to the postnatal weekly decrease reported by other authors. Thus, it appears that at least in the normal infant the shift from fetal to adult hemoglobin is related to the age of the infant from the time of conception

(6) Pediatrics 20:272-278, August, 1957.

and is not affected by the change from intra- to extrauterine existence. Further, these data provide physiologic evidence in support of the clinical impression that the postmature infant differs from the normal, full-term infant.

Of 152 infants, 28 were considered to have had significant prenatal hypoxia, as evidenced by striking meconium staining of the skin, nails and umbilical cord at birth. The gestational age was, by calculation, 42 or more weeks in 21 cases, and these infants had the complete picture of postmaturity and placental insufficiency. These postmature, hypoxic infants showed no evidence of an increased percentage of fetal hemoglobin in response to lack of oxygen.

Infants with intrauterine hypoxia showed a significant increase in total concentration of hemoglobin (adult and fetal) above that of normal infants of the same gestational age, the hypoxic infant averaging 19.2 Gm. compared to 17 Gm. for the normal. There was no apparent increase in reticulocytosis in response to intrauterine hypoxia, however. These findings suggest that the increase in total concentrations of hemoglobin in infants with prenatal hypoxia may not be the result of an increase in the total amount of hemoglobin in the body, but rather the result of hemoconcentration. Such an explanation seems consistent with the clinical impression of dehydration in these infants.

► [Apparently, intrauterine anoxia if associated with postmaturity is not sufficiently prolonged to affect the percentage of fetal hemoglobin. Bromberg *et al.* (J. Obst. & Gynaec. Brit. Emp. 63:875, 1956) found that fetal hemoglobin concentration is not increased in infants having anoxia at birth due to complications arising during labor. However, in infants of mothers with chronically lowered oxygen blood content due to heart disease with persistent dyspnea and cyanosis, the fetal hemoglobin at birth varied from 94.5 to 100%, suggesting that the production of adult hemoglobin by the fetus is delayed only in those conditions in which oxygen supply to the fetus is chronically reduced for a considerable part of the pregnancy. If their observations are correct, anoxia associated with placental insufficiency from postmaturity is too brief to affect the fetal adult hemoglobin ratio."

Dr. Cook was asked if he had theoretical explanation for the failure of the hypoxic fetus to *increase* its hemoglobin. He replied:

"We only present evidence that the total amount of hemoglobin apparently does not increase in response to hypoxia. Blood volume measurements must be done to verify this. Unfortunately, I have no good theoretical explanation of this phenomenon. In experimental newborn animals, however, hemoconcentration occurs as a result of anoxia during short-term experiments. The long-term studies still need to be done."—Ed.]

Chemical Analyses of Blood from Umbilical Cord of Newborn: Relation to Fetal Maturity and Perinatal Distress is discussed by Loren G. MacKinney, Irving D. Goldberg, Frances E. Ehrlich and Katherine C. Freymann[7] (Children's Hosp., Buffalo) on the basis of findings in 607 cases. The oxygen saturation of the blood in the umbilical vein is significantly higher when the initial respiration occurs before clamping. Presumably, an increment of oxygen is thus added, making the oxygen saturation at the moment of sampling no longer the oxygenation status attained by means of the placenta alone. To evaluate data on oxygen saturation in the blood of the umbilical cord, consideration must be given to this time relationship.

In general, in infants with poor clinical status at birth, the oxygen saturation sampled before respiration is significantly lower than in normal infants. In premature infants, oxygen saturation sampled before respiration has lower values than in mature or postmature infants. This difference is not explained solely by the less favorable neonatal status of some premature infants. The oxygen saturation of postmature infants sampled before respiration is not lower than that of mature infants. Infants delivered by cesarean section have a lower oxygen saturation than do those delivered by other means.

Walker has reported data on oxygen saturation of blood in the umbilical vein in infants of 30 to 43 weeks' gestation. On the basis of data from 8 infants delivered by cesarean section before labor, he concludes that the oxygen saturation at 30 weeks' gestation is about 70%, at 40 weeks 60% and at 43 weeks 30%. These findings are at variance with present findings which show that in the "before respiration" group premature infants have a significantly lower oxygen saturation than either mature or postmature infants. The authors are unable to account for these contradictory results unless cesarean section or absence of labor can be shown to reverse oxygen-saturation patterns related to gestational age.

Although it is generally thought that abnormal coloring of the vernix and amniotic fluid is associated with fetal dis-

(7) Pediatrics 21:555-564, April, 1958.