1972

Year Book
OF
PEDIATRICS

GELLIS

THE YEAR BOOK *of* PEDIATRICS 1972

EDITED BY

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INTRODUCTION

Our careful readers may have noted how rarely we begin the YEAR BOOK OF PEDIATRICS with an introductory page. Omission of the page stems not from laziness but from a feeling that we are sufficiently wordy throughout the volume. However, a special note is indicated for the 1972 volume to mark the appearance of a new chapter, that on Adolescent Medicine. This chapter makes its debut as a result of unremitting pressure by Dr. Roswell Gallagher and Dr. Joseph Rauh, to whom we express our thanks. May their specialty and this chapter thrive.

We wish to acknowledge once again our gratitude to those kind souls who endure our questions and proddings and patiently produce guest editorial comments.

SYDNEY S. GELLIS

THE PREMATURE AND THE NEWBORN

Relation of Poverty and Race to Antenatal Infection. Perinatal infant mortality rates in poor families in the United States are far higher than those in the nonpoor. Most of the excessive mortality appears related to lower birth rates in the poor, a reflection of either premature delivery or antenatal growth retardation. Recently, undernutrition was found responsible for a degree of prenatal growth retardation in infants born to a group of poor urban mothers in the United States. Such undernutrition was not severe enough to explain all the excessive perinatal mortality in such poor families. The present study shows that antenatal infection may be a factor in the excessive mortality. Richard L. Naeye and William A. Blanc[1] examined material from 1,044 consecutive autopsies on newborn and stillborn infants and related the findings to the parents' socioeconomic status. Gestational ages calculated from the last menstrual period ranged from 20 to 44 weeks.

Infants from the poorest families had the highest percentage of chorioamnionitis, pneumonia and splenitis. With each improvement in economic status, the rate of inflammation decreased. Rates of inflammation were highest in blacks and lowest in whites. Puerto Ricans had rates just above those in whites. Infection may have been responsible for initiating labor in some of the premature deliveries. Membranes reportedly ruptured more than 20 hours before delivery in only 10% of the cases, and in many with such early rupture there was no associated chorioamnionitis, pneumonia or splenitis.

Chorioamnionitis, splenitis and pneumonia often appeared to be due to bacterial infection. Pneumonia was rarely associated with a normal umbilical cord and membranes. Pathogenic bacteria cultured from the lungs and blood were significantly correlated with pneumonia and splenitis. There was no positive correlation among Apgar scores, anatomic abnormalities in the neonate usually ascribed to hypoxia and newborn pneumonia and splenitis. In the entire series, infants with pneumonia and splenitis had adrenals that were 15% heavier for gestational age than glands in neonates without infection. The enlargement was due to hypertrophy of individual cells in a single zone of the glands, the permanent or adult cortex.

The study demonstrated a relation between chorioamnionitis in new-

(1) New England J. Med. 283:555-560 Sept. 10, 1970.

borns and pneumonia and splenitis. Antenatal aspiration of infected amniotic fluid appears to be the most common mechanism responsible for the pneumonia and splenitis. In all racial groups, as family income increased, the frequency of such infections decreased.

► [Data from this study lend additional support to the theory that fetal adrenal secretions may play an important part in initiating labor. G. C. Liggins (J. Endocrinol. 42:323, 1968) presented evidence that this may be true in lambs. In the present study, infected infants had adrenal glands that were significantly larger than the glands of infants without infection. The enlargement was shown to be caused by hypertrophy of cells in the permanent or adult cortex.

If the authors' findings are true, namely, that chorioamnionitis is the cause of congenital pneumonia and is more frequent in the poor, the reasons for the higher incidence in the poor are not clear. Anderson and co-workers (New England J. Med. 273:308, 1965) suggest that the higher perinatal death rate in the poor may be due to different standards of prenatal care in public and private hospitals. Naeye and Blanc speculate on the possibility that genetic factors may have a bearing on antenatal infections, pointing out that black newborn infants have lower serum IgM levels than white ones, referring to the work of Hardy *et al.* (J. Pediat. 75:1211, 1969) who showed that half the perinatal deaths have been reported to occur among infants with the lowest levels of IgM. The present study shows that different economic and racial groups receiving care in the same prenatal clinic had quite different rates of antenatal infection.

We have never been particularly impressed with prenatal care as a factor in the health of the mother and her newborn infant, but we have felt that the mother who is sufficiently sophisticated to seek out or to be pushed into attending a prenatal clinic is more likely to be impressed with the importance of good diet and good hygiene during her pregnancy. If she comes from an economically higher level, she is even more likely to have better nutrition and good health practices. We vote for better housing, better education, better job training and opportunity for the poor, and fewer prenatal clinics.—Ed.]

Associations between Drugs Administered during Pregnancy and Congenital Abnormalities of the Fetus. Matilda M. Nelson and John O. Forfar[2] (Univ. of Edinburgh) studied the consumption of prescribed and self-administered drugs by pregnant women over a period of 2 years. During this time, 458 women gave birth to infants with congenital abnormalities, 175 with major and 283 with minor abnormalities. A control group consisted of 911 mothers of normal babies.

All mothers were interviewed before discharge from the hospital and questioned about drugs consumed during pregnancy. An attempt was made to recover the prescriptions issued to the mothers. The total numbers of prescribed drugs taken by the women in the study and control groups during the whole of pregnancy and in the 1st trimester were similar, as were the total numbers of self-administered drugs.

Analgesics were taken by a significantly higher proportion of mothers of infants with all and minor abnormalities during the whole of pregnancy and during the first 56 days. In particular, aspirin was taken by a significantly higher proportion of mothers of all abnormal infants and of infants with major abnormalities. The number of mothers taking antacids in the 1st trimester was significantly higher in the study group than in the control group.

Compared with controls, significantly fewer mothers of infants with major abnormalities consumed antiemetics during the 1st trimester. Significantly fewer mothers of infants with abnormalities took antihistamines in the 1st trimester compared with controls. No group or individual differences were evident for antibiotics, bronchodilators, hormones, diuretics and tranquilizers. Appetite suppressants, dex-

(2) Brit. M. J. 1:523-527, Mar. 6, 1971.

troamphetamine in particular, were taken by a higher proportion of mothers of infants with abnormalities during the whole of pregnancy and during the 1st trimester.

The most significant association between any drug and the occurrence of congenital abnormalities was seen with barbiturates. The proportion of mothers of abnormal infants taking barbiturates in the 1st trimester was significantly higher than in the control group. Dosages tended to be higher in mothers of abnormal infants. A higher proportion of mothers of abnormal infants took cough medicines.

Whereas in the first 56 days of pregnancy vitamins and iron were consumed by a significantly higher proportion of mothers of abnormal infants, the reverse was true when the whole of pregnancy was considered. The apparently protective effect of vitamins was specifically related to ascorbic acid and folic acid. Folic acid deficiency has a teratogenic effect in rats and a similar effect may be evident in human beings.

During pregnancy, self-medication with common household remedies such as aspirin and antacids should be avoided.

[Retrospective studies of drug intake and birth defects are subject to even greater errors than prospective studies. It is obvious that the only way we could monitor drug use exactly in pregnancy would be to assign one investigator to attend constantly each mother throughout her pregnancy. She could never be left alone: the American mother has a full range of medications in every pocketbook. It is all too easy for us to suspect every drug as a possible teratogen but much more difficult to obtain proof. We know nothing about drug combinations that might have a potentiating effect and be greater teratogens than the same drugs taken singly. Some drugs may be responsible for birth defects at such a low frequency rate that we shall never be able to incriminate them. Despite the lack of solid evidence against barbiturates, aspirin, antacids and all the other garbage modern society ingests, it continues to make sense for the pregnant woman to suffer the minor discomforts of pregnancy in silence and without medication. Women complain too much anyhow.

During the past year, other drugs have come under scrutiny as teratogens. Trimethadione was reported by J. German (Teratology 3:349, 1970) as the possible cause of 4 malformed children born to a mother who was taking the medication for epilepsy. When the drug was discontinued, she had 2 normal children. Study by the author of 14 other pregnancies during which trimethadione or paramethadione was taken revealed a high frequency of abnormality.

S. R. Meadow (Proc. Roy. Soc. Med. 63:12, 1970) has continued the investigation of possible association of cleft lip and cleft palate with antiepileptic drugs taken during pregnancy. The data suggest that the frequency of association is greater than would be expected, but the author properly insists that further investigation is needed.

B. L. Murkin (Am. J. Obst. & Gynec. 109:930, 1971) demonstrated the transplacental passage of diphenylhydantoin from pregnant epileptic women receiving standard doses of the drug, showing that fetal blood samples obtained from the umbilical artery and vein at term and from the neonate on days 1 and 2 had essentially the same drug levels as were present in the mother's plasma. Of special interest is that 2 of the 3 infants studied had cleft lip and the third infant had both cleft lip and cleft palate.

Investigators continue to look closely at LSD as a potential teratogen. Eller and Morton (New England J. Med. 283:395, 1970) report data on an infant with multiple skeletal abnormalities, hydrocephalus and fusion of the frontal lobes of the brain who was born to a woman who took LSD at about the time of conception. Chromosome studies were normal and the infant's abnormalities can hardly be attributed to LSD.

O. K. Mjølnerød *et al.* (Lancet 1:673, 1971) reported the outcome of pregnancy in a woman with severe cystinuria treated with D-penicillamine daily during pregnancy. The newborn child had a generalized connective tissue defect including laxity of skin, hyperflexibility of joints and varicosities. The evidence seems overwhelming that the drug was responsible. The infant died of sepsis at age 7 weeks.

One of the most alarming reports of the past year relating drug therapy in pregnancy to abnormality and disease in offspring is that by A. L. Herbst *et al.* (New England J. Med. 284:878, 1971). Adenocarcinoma of the vagina, a rare tumor in young women, was found in 8 patients between 1966 and 1969 whose mothers had been treated with diethylstilbestrol

because of bleeding in the 1st trimester of pregnancy. If there has ever been a strong indictment of hormonal therapy in pregnancy, this is it. The evidence suggests that stilbestrol causes some abnormality of the developing fetal urogenital tract. Whether or not similar malignancies will be attributable to other estrogenic substances remains to be seen but should not be put to the test. The obstetrician must realize that any agent given to the pregnant woman will reach her fetus. The discovery that ill effects may not appear until the fetus is aged 20 is most distressing.—Ed.]

Birth Weight and Genital Mycoplasmas in Pregnancy. Peter Braun, Yhu-Hsiung Lee, Jerome O. Klein, S. Michael Marcy, Thomas A. Klein, David Charles, Paul Levy and Edward H. Kass[3] (Boston) prospectively studied a large series of unselected patients entering the prenatal clinic during 6 months to ascertain any relation between presence of genital mycoplasmas and outcome of pregnancy.

Among the 485 pregnant women who had cultures taken from both the cervix and urine, there were 464 single births, 6 pairs of twins, 10 stillbirths and 6 spontaneous abortions. The patients were classified into four groups according to presence or absence of T strains of mycoplasma and Mycoplasma hominis in a culture of either the urine or cervix.

The mean total birth weights and mean live birth weights in groups having T strains were significantly lower than those in groups not having T strains. The mean birth weight of 384 infants of mothers with a T strain was 202 Gm. less than that of 100 babies from mothers without T strains. The mean birth weight of infants of mothers whose cultures yielded M. hominis was 103 Gm. less than that of infants of mothers without M. hominis; the difference in mean birth weights was less significant than was noted with T strains. There was no significant relation between either T strains or M. hominis and risk factors for low birth weight such as previous history of prematurity, abortion or stillbirth, cigarette smoking or bacteriuria. Both T strains and M. hominis were significantly more prevalent among the 214 black patients than among the 270 white and other patients.

T strains were isolated from the noses and throats of 7 (28%) of 25 babies weighing less than 2,500 Gm. and 19 (5%) of 379 babies weighing more than 2,500 Gm. Isolation of T strains from vaginal cultures of 35 of 193 infants showed no relation to low birth weight. Mean birth weight and duration of gestation were significantly less in babies from whom a T strain was recovered from the nose or throat than in those without T strains, but no such difference was observed when the small group of babies (7) positive for M. hominis was compared with babies negative for this mycoplasma.

The data presented demonstrate a relation between genital T strain mycoplasmas in pregnancy and low birth weight of the infant.

► [Dr. Ruth Kundsin and Dr. Mary Ampola commented:

"Braun and his collaborators are to be congratulated on this important and elegant study relating low infant birthweights to maternal T strain genital tract colonization. This article adds another dimension to the earlier work of other investigators who established the pathogenicity of T strain mycoplasmas. Shepard *et al.* associated T strain mycoplasmas with nongonococcic urethritis in the male in 1964,[1] calling it the sixth venereal disease. Kundsin and collaborators associated these organisms with reproductive failure ranging from infertility to spontaneous abortions and premature births in 1967[2] and 1968.[3] This research has been extended recently to the demonstration that T strain mycoplasmas can induce various

(3) New England J. Med. 284:167-171, Jan. 28, 1971.

types of chromosomal abnormalities in peripheral lymphocyte cultures (Kundsin *et al.*, 1971).[4]

"In the present study, the authors note no mycoplasma effect on the abortion rate. However, the average patient presented herself for prenatal care at 23 weeks of gestation, well beyond the period abortions usually occur (about 82% of abortions occur before 20 weeks[5]).

"Differences in the severity of the mycoplasma effects, ranging from primary infertility to the mild fetal growth retardation reported here, may be due to differences in strain virulence, severity of the infection, or host resistance.

"Several questions of technic, which may have influenced the outcome of this study, deserve comment. It has been the experience of most workers in the mycoplasma field that simultaneous inoculation of liquid and agar mediums for primary isolation is essential, especially for T strains. False positive and false negative results can occur if pH change in liquid medium is solely relied upon to determine the presence of these mycoplasmas. Bacteria or fungi can, on occasion, cause an alkaline shift in liquid urea mediums, and, conversely, T strains visualized on agar have not altered the pH of the urea mediums in a few instances.

"Because 70% of human mycoplasmas cannot be subcultured, dependence on subculture alone is not accepted practice. Further, if the dual primary isolation had been used, differentiation between classic and T strains would have been possible on the agar, using urea and manganous chloride, without resorting to mediums with antibiotics.

"The excitement in the scientific community upon the recognition of a new agent of human infection is real, important and understandable. Braun *et al.* in this study of 485 patients furnish additional evidence that mycoplasmas, and more specifically T strains, do indeed play a role in abnormal fetal development."

1. Shepard, M. C., *et al.*: Possible Role of T Strain Mycoplasmas In Nongonococcal Urethritis, J.A.M.A. 188:729, 1964.
2. Kundsin, R. B., *et al.*: Strain of Mycoplasma Associated With Human Reproductive Failure, Science 157:1573, 1967.
3. Kundsin, R. B., *et al.*: T Strain Mycoplasmas in Human Reproductive Failure, Bacteriological Proceedings, p. 78, 1968.
4. Kundsin, R. B., *et al.*: Chromosomal Aberrations Induced by T Strain Mycoplasmas, J. M. Genetics 8:181, 1971.
5. Shapiro, Sam, *et al.* (eds.): *Infant, Perinatal, Maternal and Childhood Mortality in the U.S.* (Cambridge, Mass.: Harvard Univ. Press, 1968), p. 70.—Ed.]

Infant Mortality is discussed by Bruce E. Balfe[4] (American Med. Association, Chicago). The infant mortality rate is not the best or even a good indicator of the health status of a nation or the efficacy of a nation's health delivery system. Infant mortality is, for the most part, a social rather than a medical problem. Such factors as poverty, malnutrition, poor housing, low education levels and racial or ethnic differences are more highly correlated with infant mortality than the number of physicians or hospitals. Even if infant mortality is used for discussion of a nation's social problems, it should be used in conjunction with other social indicators.

Infant mortality loses considerable significance when it is considered that 70% of the deaths in the United States are related to heart disease, cancer or stroke and only 2.2% of deaths are classified as infant mortality (1969 data).

If interest is in infant mortality as a social problem which can be improved, international comparisons are not particularly useful. Comparison of the United States with Sweden, for example, usually implies that the United States should adopt the Swedish health or social system. This ignores the differences between the countries in population size, area and heterogeneity of population.

The relevant information concerning infant mortality is whether the

(4) Missouri Med. 68:225-227, April, 1971.

American record is improving or deteriorating. The 1969 rate is less than half the 1940 rate.

If the health status of the population and the efficacy of the health delivery system is evaluated, a broad set of data, probably summarized into a weighted index, should be used. Such an index should not be used for fixing blame or delivering praise; rather, it should be used by those genuinely interested in improving social conditions in the United States.

► [We have expressed repeatedly our annoyance with those who attribute the high infant mortality rate in the United States to lack of medical care. This does not mean that we are opposed to good prenatal care, good care during labor and delivery, and close supervision of the newborn infant. Such care must be available to all. However, there is little likelihood that medical care will affect the mortality rate, which is tied closely to educational and social problems. We do not agree with the author that infant mortality rate loses considerable significance when it is considered that 70% of the deaths in the United States are related to heart disease, cancer or stroke and only 2.2% of deaths are classified as infant mortality. We can't alter our basic infant mortality rate by dumping it into the over-all figure. All we can conclude is that we don't do well either by infants or adults and must work hard to improve the lot of both. Such improvement will not come about by prenatal visits or annual physical examinations but by the improvement in all aspects of daily living, factors that are not controlled by the medical profession. If we spent less time being apologetic about our infant mortality rate and more of our energies in stressing the relationship of the mortality rate to social problems, the infants might benefit greatly.

E. G. Ludwig and J. C. Collette (Some Misuses of Health Statistics, J.A.M.A. 216:493, 1971) point out the difficulty of drawing conclusions from health statistics. Because many countries that have lower infant mortality rates than ours have home deliveries and midwives, it has been argued that this system would help reduce our rate. "Yet in the only section of the United States where midwifery is at all common, the low-income rural South, the infant mortality is generally the highest in the nation."

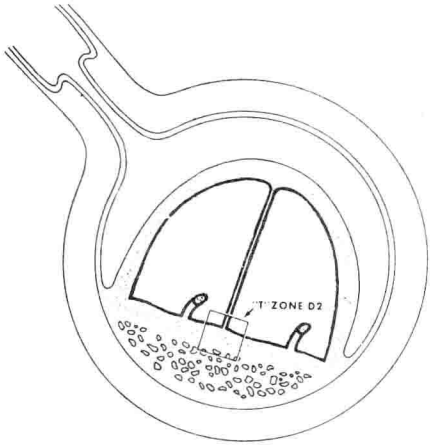
C. L. Erhardt *et al.* (Arch. Environ. Health 20:743, 1970) summarize the problem well: "It is frequently stated that community health should not be weighed in terms of cost, that efforts should be strained to maintain health and prevent illness, and that interest in community health will redound to better citizenry and more effective functioning of citizenry. It is also said that funds should be made available at all times for experiment with methods of administering and rendering health services and be concerned primarily with the achievement of better health for all in the community. In return, implementation of approved programs would subsequently be reflected in more mature and healthier babies and with lowered fetal and infant death rates. However, it is still a matter of conjecture whether merely pouring in huge, loosely controlled sums of money to mount programs of health, without clear thought and planning as to what the programs are designed to achieve, will bring about intended results."—Ed.]

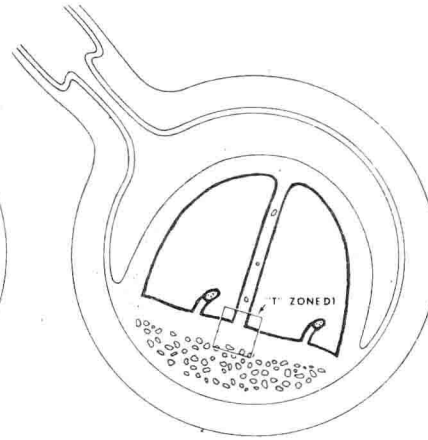
Twin Birth: Identical or Fraternal Twins? Twins of opposite sex are fraternal; no other anatomic finding proves conclusively that twins are fraternal. M. Shannon Allen, Jr., and U. G. Turner, III[5] (Univ. of Virginia) discuss the usefulness of the examination of the placenta and membranes in twin births in determining zygosity. If a single chorionic sac is present, the twins are identical (Fig. 1). Although identical twins may have either one or two chorionic sacs, only those with one chorionic sac (about 80% of identical twins) can be positively identified as identical by examining the placenta and membranes. Twins of the same sex with two chorionic sacs (Fig. 2) may be either identical or fraternal.

Rarely, a single placental mass is found with only one amnionic sac (Fig. 3). When no complete or partial dividing membrane is present between the fetuses, only one amnion and one chorion exist and the twins are identical.

If blood or air bubbles from the vessels of one fetal placental circulation can be pushed into the vasculature of the other fetal placental circulation, this supplies good gross evidence that only one chorion

(5) Obst. & Gynec. 37:538-542, April, 1971.





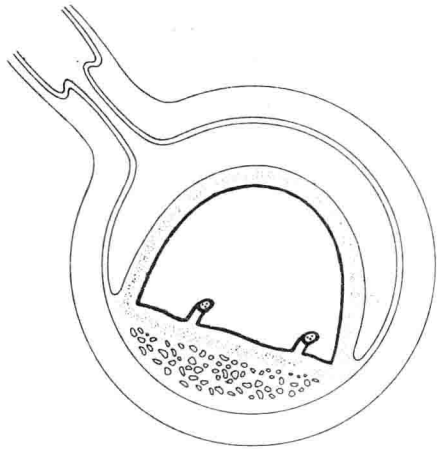


Fig. 1 (above left).—Monochorionic twin pregnancy. No chorionic tissue extends into dividing septum between the two fetal cavities.

Fig. 2 (above).—Dichorionic twin pregnancy. Chorionic tissue extends between amnions forming septum between the two fetal cavities.

Fig. 3 (left).—Rare monoamnionic-monochorionic twin pregnancy. No septum.

(Courtesy of Allen, M. S., Jr., and Turner, U. G., III: Obst. & Gynec. 37:538-542, April, 1971.)

exists. If twin placentas are completely separate, they are almost always dichorionic; if they are more or less fused (one placental mass), they may be dichorionic or monochorionic. The completely fused placenta is monochorionic; the incompletely fused placenta is dichorionic.

Close gross inspection of the septum between the two fetal cavities determines whether the septum consists of amnion-amnion or amnion-chorion-chorion-amnion. If the septum is relatively translucent with no chorionic tissue between the amnions and if the amnions can be readily pulled away from the placental surface leaving a smooth flat surface, it is extremely likely that only one chorion exists.

The gross observation of the placenta can be verified by microscopic examination of a rolled segment of the septum between the two fetal cavities; the septum will be seen to consist of two amnions with no chorion between them if the septum is achorionic. Microscopic examination of the "T" section of the placenta, where the relationship of the

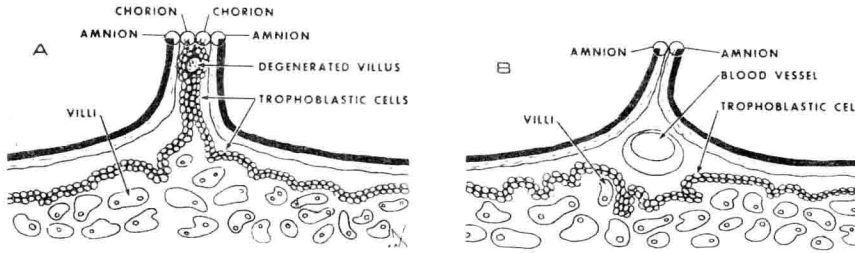


Fig. 4.—Comparison of anatomic arrangement of "T" zone of *A*, a dichorionic placenta, and *B*, a monochorionic placenta. (Courtesy of Allen, M. S., Jr., and Turner, U. G., III: Obst. & Gynec. 37:538–542, April, 1971.)

membranes as they leave the placenta to form a septum between the two fetal cavities is readily identified (Fig. 4), also verifies the gross observation.

► [This article shows how to distinguish fraternal from identical twins. If the pediatrician is not interested, he can at least shove the article under the nose of his obstetric colleague to emphasize the need to submit the placenta for identification. This chance will never occur once the placenta is thrown away.

We asked Dr. Kurt Benirschke to tell us about the accuracy of the determination:

"The identification of the composition of the dividing membrane, that membrane which separates the two cavities in which the fetuses reside in utero, is easily accomplished. The amnion consists of a layer of epithelial cells beneath which there is a layer of connective tissue cells that never contains any blood vessels. In the case of a diamnionic membrane, one would see two layers of epithelial cells and two thin layers of directly adjacent connective tissue elements. In the case of a diamnionic-dichorionic twin placenta, two additional membranes would be present, namely, the two chorions. These are again connective tissue layers and they would be separated from the amnion by a thin slit, a potential space that has been occluded by the pressure from within the amnionic cavity. Aside from these two additional layers, it is easily possible to identify a few remnants of degenerated villi that have undergone compression atrophy and perhaps a few trophoblastic and decidual remnants as well in between the two chorionic leaves. The identification of two vs. four layers in well-prepared T sections as advocated by the author or in membrane roll from the dividing membranes is absolutely positive and can be accepted as definitive proof of the type of placentation of a given set of twins. It should become a record entry of every twin delivered and is best accomplished in the pathology laboratory."—Ed.]

Mechanical Ventilation of Newborn Infants.—*III. Historical comments and development of a scoring system for selection of infants.*—William J. R. Daily, H. Belton P. Meyer, Philip Sunshine and Penelope C. Smith[6] (Stanford Univ.) developed a scoring system based on arterial pH, Po_2, Pco_2 and apnea to define respiratory failure of uniform severity. Review was made of the data on 204 infants seen with neonatal respiratory failure in a 5-year period. Respiratory distress was diagnosed if an infant had expiratory grunting, nasal alar flaring, chest retractions and reticulogranularity and an air bronchogram on a chest x-ray.

All infants received penicillin and kanamycin. Sodium bicarbonate was infused through an umbilical catheter when the arterial pH was below 7.3. Oxygen was given with signs of distress, generalized cyanosis or Pao_2 below 60 mm. Hg. The lungs were manually ventilated when the $Paco_2$ was over 65 mm. Hg or respiratory arrest occurred. When this treatment failed and the score was 3 or higher on pure oxygen inhalation, intermittent positive-pressure ventilation was given. A score of 3 was assigned with Pao_2 below 40 mm. Hg, pH below 7.0, $Paco_2$ over 80

(6) Anesthesiology 34:119-126, February, 1971.

mm. Hg or apnea, with lower scores for lesser degrees of abnormality. Normal values for Pao_2, $Paco_2$ and pH were, respectively, over 70 mm. Hg, less than 60 mm. Hg and 7.31-7.5.

A score of 3 or higher was given to 185 infants, all but 12 of whom had a score of 3 on at least one criterion. Respiratory distress syndrome was diagnosed in 118 infants, including 101 with a score of 3 on at least one criterion. Two or more episodes of apnea with bradycardia or cardiac arrest constituted an indication for mechanical ventilation in 131 infants, 70 of whom had respiratory distress syndrome. Respiratory distress was diagnosed in 36 of 59 infants with Pao_2 below 40 mm. Hg and in 19 of 38 with $Paco_2$ over 80 mm. Hg. Two of 13 infants with pH below 7.0 had respiratory distress.

There is no evidence that one method of mechanical ventilation of newborn infants is better than any other. Acidosis and respiratory arrest in the extremely hypoxic infant does not further reduce the chance of survival after mechanical ventilation.

IV. Technic of controlled intermittent positive-pressure ventilation.—There is no evidence that the type of mechanical ventilation used for newborn infants with respiratory failure affects survival, but changes in the pattern of intermittent positive-pressure ventilation predictably influence oxygenation and ventilation in those with respiratory distress syndrome. Smith and Daily[7] studied the data on 204 mechanically ventilated newborn infants with respiratory distress who failed to respond to standard treatment. Manual hyperventilation with pure oxygen for 3-5 minutes preceded nasotracheal intubation. Foregger plastic tubes are now used. McGill forceps are not needed with slight neck flexion or external pressure on the larynx.

The largest endotracheal tube possible was inserted, and chest x-rays were obtained after fixation of the tube. Tubes were changed at 24 hours, and then every 48-72 hours. A Bennett PR-2 unit with a low dead space infant circle was used. Curarization has been abandoned. An 80-100% oxygen concentration was used initially; the level was reduced until a Pao_2 of 60-100 mm. Hg was obtained without metabolic acidosis. Every effort was made to reduce the inspired oxygen level to below 70% as soon as possible. When a higher concentration was needed, the ventilator was powered with oxygen and air was added proximal to the humidifier and bacterial filter.

All equipment was washed in Cidex and sterilized in ethylene oxide before use. Pulmonary physiotherapy, including vibration, percussion and suctioning, was done every 2-4 hours. Suctioning was done using a catheter 1 cm. longer than the endotracheal tube. Patients were treated in the supine and oblique positions. The endotracheal tube and airways were suctioned hourly.

Infants were weaned from mechanical ventilation by reducing the inspired oxygen level and then altering the ventilatory pattern. Extubation was attempted when spontaneous breathing had been tolerated for 8 hours or longer. One to 17 days were required for weaning. Weaning was

(7) Anesthesiology 34:127-131, February, 1971.