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Monitoring of the Fetus

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THE ABILITY to determine fetal maturity and well-being during or even before labor is an important requirement if fetal and neonatal wastage is to be prevented. Until recently, the physician caring for the pregnant woman had to rely on rather primitive and grossly inaccurate modalities of fetal surveillance, namely palpation of the abdomen, observation of the character of the amniotic fluid and intermittent auscultation of the fetal heart. Newer methods, introduced in the last 15 years, are based on advanced biophysical and biochemical technology and allow for greater accuracy in monitoring. Their understanding is mandatory for all physicians who participate in the care of the mother and her offspring. This review places special emphasis on commonly used techniques for intrapartum surveillance.

Antepartum Biochemical Evaluation

Maternal Urinary Estriol

Determination of 24-hour estriol excretion in the maternal urine, introduced in 1963, has become the most widely used test of fetal well-being before labor.[1] The hormone is synthetized by the placenta from androgen precursors originating chiefly in the fetal adrenal gland and the liver. Estriol passes from the fetus into the maternal circulation and is excreted by the kidneys. Thus, a breakdown any place along the transport and metabolic pathway can cause a reduction in estriol excretion, as can certain drugs, such as mandelamine and ampicillin.[2]

Maternal urinary estriol excretion increases with gestational age. However, substantial daily fluctuations can be observed (fig. 1), and abnormal values are difficult to define. Some clinicians feel that a reduction in estriol excretion near term must amount to at least 35–50 per cent of the mean value obtained by serial determinations over the preceding few days, in order to be significant.[3] An abnormally low or declining excretion of urinary estriol indicates a fetus in jeopardy.[1] Both have been found prior to fetal demise in pregnancies complicated by diabetes, hypertension or pre-eclampsia.

Human Placental Lactogen

The measurement of human placental lactogen (HPL) in maternal serum by radioimmunoassay may also be used as an index of fetal well-being.[4] Since the test can be performed rapidly it is useful for screening of a large number of patients. HPL, also known as human chorionic somatomammotropin, is a polypeptide produced by the placental trophoblast. Its physiologic role is unknown. HPL concentrations in the maternal plasma vary from 2 to 12 ng/ml. Values above 4 ng/ml are considered normal after the thirtieth week of gestation.[5]

Measurement of the Constituents of Amniotic Fluid

Bilirubin

Management of the Rh problem means detection and management of erythroblastosis fetalis, a hemolytic disease of fetal or neonatal life. Although the pathogenesis of this condition has been understood for the last 35 years,[6,7] overall perinatal mortality among affected infants remained as high as 25–30 per cent until the introduction of amniocentesis and amniotic fluid analysis, which, combined with aggressive treatment, have reduced it to approximately 10 per cent.[8,9] This newer approach followed the investigations of Bevis, who showed that the severity of disease correlated with the concentration of bilirubin (produced by the fetal hemolysis) in amniotic fluid.[10]

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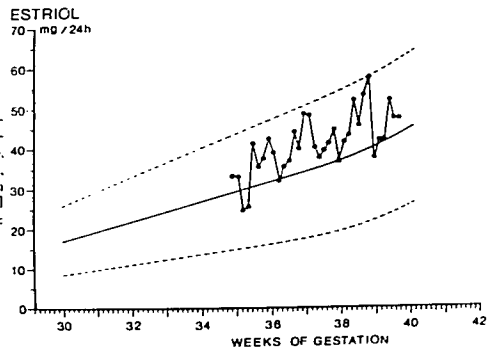

FIG. 1. Normal urinary estriol excretion pattern. Dotted lines represent the range of the mean ± 2 SD. Reproduced from Freeman RK, Kreitzer MS: Current concepts in antepartum and intrapartum fetal evaluation, Curr Probl Pediat 2(9): 1,1972, with permission.

In the management of an individual pregnancy the first imperative is to identify the fetus at risk by determining the degree of maternal isoimmunization. An antibody titer of 1:32 or higher implies the need for immediate amniocentesis.[11] The objectives of amniotic fluid analysis are to avoid intrauterine death in the severely affected fetus and to allow the mildly affected one to remain *in utero* until it is more mature.

Amniocentesis is performed under aseptic conditions, with a 3½-inch, 20- or 21-gauge needle. In order to avoid penetration of the placenta, its location may be first ascertained by sonography, or, more simply, puncture may be performed via the suprapubic area, thus missing the placenta (fig. 2).[11] A bimanual examination can localize the fetus, and elevation of the presenting part will prevent fetal trauma.

Five to 10 ml of amniotic fluid are aspirated, centrifuged, and passed through Whatman filter paper to remove epithelial cells and vernix caseosa. The bilirubin concentration is then determined using spectrophotometry, which scans the absorbance of the amniotic fluid at wavelengths between 300 and 600 mμ. Figure 3 reproduces serial spectrophotometric scans, demonstrating a progressive increase in bilirubin concentration. The presence of bilirubin is indicated by increased optical density between 375 and 525 mμ, with a peak at 450 mμ. The height of this "bilirubin hump" is expressed as ΔOD at 450 mμ. A straight (broken) line is drawn from 357 to 525 mμ, and a vertical (solid) line is drawn from the peak at 450 mμ. ΔOD is determined by subtracting the value of optical density at the intersection of the two lines from that at the top of the vertical line.

The following management, based on ΔOD values, has been proposed by Freda,[11] and is commonly used in our institution:

ΔOD 0–0.20 (1 + abnormal curve): the test should be repeated at ten-day intervals.

ΔOD 0.20–0.34 (2+) indicates that the fetus is Rh-positive and affected to some extent by hemolysis. Delivery before 37 weeks is not indicated unless there is a history of previous severe hemolytic disease. With a history of a previous Rh stillbirth, fetal transfusion should be performed.

ΔOD 0.35–0.70 (3+) indicates that the fetus is in distress due to circulatory impairment. If pregnancy has reached the thirty-second week, delivery should be undertaken without delay. Before the thirty-second week intrauterine transfusion should be considered.

ΔOD above 0.70 (4+) indicates that fetal death is imminent. Delivery should be undertaken immediately, but the prognosis is poor.

In normal gestation, spectrophotometric analysis of the amniotic fluid may be used to determine fetal maturity.[12] Bilirubin is found in amniotic fluid in normal pregnancies as early as the twelfth week, and reaches its peak at 20 weeks.[13,14] The concentration of this pigment decreases gradually after the twenty-

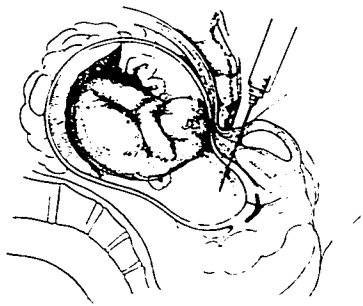

FIG. 2. Amniocentesis. Reproduced from Freda VJ: Hemolytic disease, Clin Obstet Gynecol 17:90, 1973, with permission.

sixth week until, at 36 weeks, when prematurity should not be a factor in the newborn's survival, there is normally no spectrophotometric evidence of the pigment.

With the introduction of amniocentesis, several additional methods of fetal surveillance were made possible, through the study of other amniotic fluid constituents.

LECITHIN–SPHINGOMYELIN RATIO

One of the critical events accompanying transition to extrauterine life is the initiation of respiration. After the first breath, the ability of expanded newborn lungs to maintain normal functional residual capacity depends on the presence of pulmonary surface-active material (surfactant) in the alveolar lining layer.[15] Surfactant, through its detergent-like action at

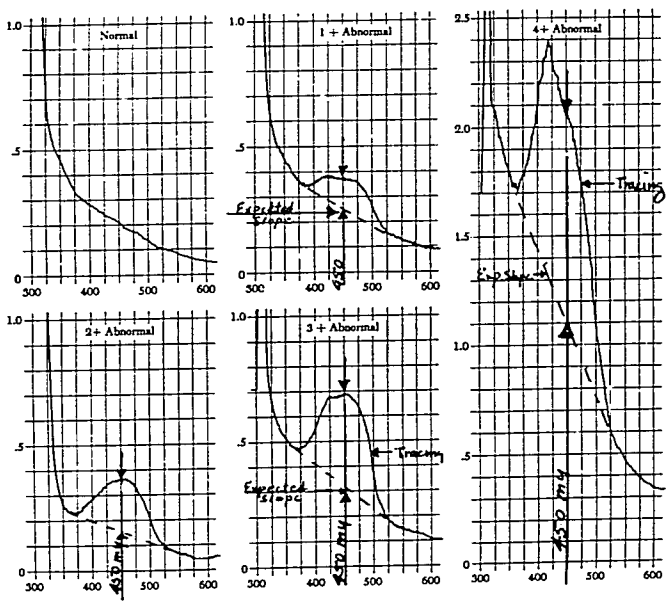

FIG. 3. Serial tracings on amniotic fluid specimens demonstrating the progressive increase in bilirubin concentration. Horizontal coordinates represent various wavelengths and vertical coordinates, absorbance in units of optical density. Reproduced from Freda VJ: Hemolytic disease, Clin Obstet Gynecol 16:91, 1973, with permission.

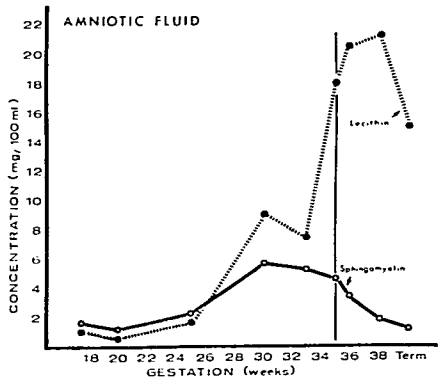

FIG. 4. Mean concentrations in amniotic fluid of sphingomyelin and lecithin during gestation. The acute increase in lecithin at 35 weeks marks pulmonary maturity. Reproduced from Gluck L, Kulovich MV, Borer RC, et al: Diagnosis of the respiratory distress syndrome by amniocentesis, Am J Obstet Gynecol 109:441, 1971, with permission.

the air–liquid interface, decreases the pressure needed to distend the lung and to prevent alveolar collapse during expiration. Deficiency or absence of surfactant, common among premature infants, is associated with the development of respiratory distress syndrome (RDS), characterized by respiratory difficulties and progressive atelectasis that may result in the formation of hyaline membranes and death.[16] Thus, functional maturation and alveolar stability of the fetal lung involve synthesis of surfactant (by Type II alveolar lining cells) and its secretion into the alveolar lumen.[17,18]

Pulmonary surfactant is a lipoprotein, containing 74% phospholipids, among which saturated lecithin comprises 56%. Dipalmitoyl lecithin is the predominant moiety.[19] Two major pathways of lecithin biosynthesis have been identified: 1) the choline-incorporation pathway, or phosphocholine transferase system,[20,21] and 2) the methylation pathway or methyltransferase system.[21,22] In the primate lung, lecithin is synthesized primarily via the choline-incorporation pathway. In the fetus, at approximately 90 per cent of term, an abrupt increase in the activity of this pathway is followed by increases in lung and amniotic fluid lecithin concentrations.[23]

Gluck and associates were the first to suggest that determinations of phospholipids in the amniotic fluid might provide means for antepartum assessment of maturity of the fetal lung.[24] They found that during gestation, concentrations of sphingomyelin and lecithin were nearly equal prior to 35 weeks, following which the lecithin concentration rose to four times that of sphingomyelin. In subsequent weeks the lecithin concentration continued to increase while sphingomyelin declined (fig. 4). Calculation of the ratio of lecithin to sphingomyelin (L/S ratio) obviated problems related to variable volumes of fluid in the amniotic sac. Furthermore, the value of the L/S ratio was found to be predictive of lung maturity.[24,25] When the L/S ratio exceeds 2.0, the newborn is unlikely to develop RDS; at a ratio between 1.5 and 1.99, distress is usually mild; a ratio less than 1.49 signals moderate to severe RDS (table 1).

Certain maternal disorders may accelerate fetal lung maturation (L/S ratios of or above 2 prior to the thirty-third week); others may retard it (mature L/S ratio after the thirty-seventh week).[24,25] The former include severe toxemia, renal and cardiovascular hypertension, sickle-C disease, degenerative diabetes mellitus (classes D, E, F), amnionitis, chronic abruptio placentae, and heroin addiction.[26] Delayed maturation is associated with mild diabetes mellitus (classes A, B, C), hydrops fetalis, and chronic non-hypertensive glomerulonephritis.

The method of Gluck *et al.* utilizes thin-layer chromatography to separate lecithin from sphingomyelin.[24,25] Relative amounts of the

two compounds are determined with a double-beam reflectance densitometer.

A simpler and more rapid test, popularly known as the "shake test," was devised by Clements and associates.[27] It is based on the ability of pulmonary surfactant to generate stable bubbles in the presence of ethanol. Ethanol excludes other substances that can also form a stable foam (proteins, bile salts, salts of free fatty acids) from the surface of the amniotic fluid, but permits lecithin to compete for the surface film. In order to perform the "shake test," small volumes of amniotic fluid, 0.9 per cent saline solution, and 95 per cent ethanol are pipetted into three test tubes in such proportions as to achieve the following ratios of amniotic fluid to ethanol: 1:1, 1:1.3, and 1:2. The tubes are stoppered, shaken vigorously for 15 seconds, and placed in vertical racks. They are examined 15 minutes later. A tube is labelled positive when a complete ring of bubbles can be seen at the meniscus. A negative test at 1:1 dilution is associated with a high risk of RDS if the newborn is delivered within 24 hours. A positive test at 1:2 dilution indicates minimal risk of RDS. By this method, surfactant becomes detectable in amniotic fluid at about 33 weeks, but the time of appearance is variable from 25 weeks to term.

Creatinine

The measurement of creatinine in the amniotic fluid is regarded as the second most reliable method of determining fetal maturity. An increasing concentration of this substance during gestation probably reflects growth of fetal muscle mass and development of renal function. A concentration greater than 1.8–2.0 mg/100 ml indicates that the fetus is more than 36 weeks old and weighs more than 2,500 g.[28,29,30]

Alpha-Fetoprotein

Recently, measurements of alpha-fetoprotein (AFP) in maternal serum and amniotic fluid have been introduced for the identification of several fetal disorders.[31,32] Initially thought to be of fetal origin, AFP has been found to be present in men and in nonpregnant women. In early gestation, elevated plasma and amniotic fluid levels of AFP strongly suggest the presence of a neural-tube lesion (anencephaly, hydrocephaly, spina bifida); in late pregnancy they may indicate esophageal atresia, fetal distress, or death.

Fetal Cell Culture

Fetal cells grown in tissue culture have been used in the early diagnosis of chromosomal disorders and hereditary metabolic diseases.[33] At approximately 14–17 weeks' gestation, 10–20 ml of fluid are obtained by amniocentesis and the cells are separated and grown in tissue culture for 3–4 weeks. At the end of this period cells are harvested and subjected to chromosomal and biochemical analyses. Using this technique, one can diagnose such disorders as trisomy-21 or Down's syndrome and predict sex-linked anomalies such as hemophilia and Duchenne's muscular dystrophy. The list of hereditary metabolic diseases identifiable *in utero* is growing almost daily.

TABLE 1. Correlation of Respiratory Status of the Newborn Infant as Predicted by Lecithin–Sphingomyelin Ratio and the Time after Amniocentesis When Delivery Occurred

L/S Ratio	Time of Delivery after Amniocentesis			
	Within 24 Hours	24–48 Hours	48–72 Hours	72 Hours–4 Weeks
0–0.99 (severe RDS)	8/8*	9/9	7/7	4/6
1.0–1.49 (moderate to severe RDS)	10/10	7/7	6/7	4/8
1.5–1.99 (mild to moderate RDS)	12/12	7/9	4/9	0/8
2.0 or more (mature lung; no RDS)	21/21	15/15	10/10	4/4
TOTAL	51/51	38/40	27/33	12/26

* Figures represent number of correct predictions to left of diagonal and total number of patients to right of diagonal.

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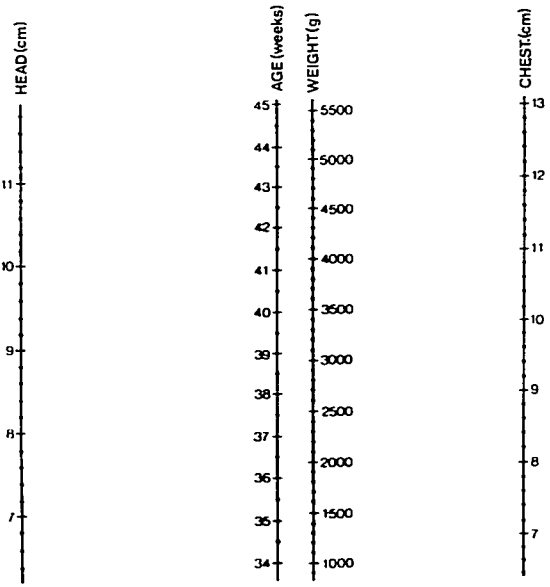

FIG. 5. Nomogram for estimating fetal weight and gestational age. Reproduced from Thompson HE: Ultrasonic diagnostic procedures in obstetrics and gynecology. J Clin Ultrasound 1:160, 1973, with permission.

the most notable example being Tay-Sachs disease. Depending on the diagnosis, the pregnancy can be terminated, as in the case of trisomy-21, or dietary adjustments can be carried out as in galactosemia and methylmalonic acidemia.

Ultrasonography

It is often desirable to determine the approximate gestational age at a time when amniocentesis for phospholipid analysis is not advisable or is impossible to perform. The growth rate of the fetal biparietal diameter is reasonably linear until the twenty-eighth to thirtieth weeks,[34,35,36] and, although variations of growth with underlying disease may occur,[37,38] a good correlation exists between biparietal diameter, fetal weight, and gestational age.[39,40] After the twenty-eighth to thirtieth weeks, measurement of the anterior–posterior diameter of the fetal thorax can be added to increase the accuracy of the estimate (fig. 5).[35] Ultrasonic scanning techniques allow for an accuracy of measurement of ±1.8 weeks or ±290 g.[41] Quite often it is of value to perform serial measurements at weekly or biweekly intervals in order to ensure accuracy and to determine fetal growth patterns.

Ultrasonography can help in the management of the high-risk pregnancy in other ways. Fetal viability can be ascertained in cases of threatened abortion.[42] Localization of the placenta for amniocentesis and for appropriate interpretation of third-trimester bleeding is accurate in approximately 97 per cent of cases.[43] Multiple gestation, fetal position, fetal anomalies, and hydatidiform mole can also be diagnosed. Finally, ultrasound has been used to determine fetal chest-wall movements.[44,45] These breathing movements occur at a frequency of 30–70 per minute, are normally present for about 65 per cent of the time, and are considered a sensitive index of fetal well-being, as they are promptly reduced or abolished by fetal hypoxia, hypoglycemia, and by depressant drugs.[46,47]

Amnioscopy

Amnioscopy was first described in 1962. Its application has reportedly caused a significant decline in fetal loss.[48,49] It involves the introduction of a conical endoscope, with an attached light source, into the cervix in order to observe the color of the amniotic fluid through intact membranes. The presence of meconium, long considered an indicator of possible fetal jeopardy, will cause the amniotic fluid to appear brown or green.

Amnioscopy probably has greatest value in pre-eclampsia and postmaturity. The procedure is performed every other day. As long as the fluid is clear, the fetus is felt to be in satisfactory condition.

Intrapartum Determination of Fetal Well-being

Prior to labor, the normal fetus is neither hypoxic nor acidotic. Animal experiments have shown that transplacental gradients for *p*H and P_{CO_2} are approximately 0.05 *p*H units and 5 torr, respectively.[50] Furthermore, normal acid–base values were found in femoral arterial blood of human fetuses during intrauterine exchange transfusions.[51] During pregnancy there is a reduction in maternal arterial P_{CO_2} to approximately 32 torr,[52] so that the normal fetal arterial P_{CO_2} is not greater than that in the nonpregnant adult although it exceeds that in the mother.

During labor, uterine contractions decrease the blood flow through the intervillous space of the placenta, or may stop it completely.[53,54] On the fetal side, compression of the umbilical cord occurs frequently during the final stages of vaginal deliveries. Analyses of cord blood at birth have revealed that slight hypoxia and acidosis are indeed common even after normal labor and delivery.[55]

Severe fetal asphyxia occasionally develops as a result of fetal and maternal complications such as a tight nuchal or body cord, a prolapsed cord, premature separation of the placenta, or uterine hyperactivity.[56] Passive hyperventilation of the mother,[57] aortocaval compression,[58,59] and maternal hypotension due to hemorrhage[60] or sympathetic blockade during regional anesthesia[61,62] may also lead to a reduction in the intervillous flow of sufficient magnitude to cause fetal asphyxia.

During asphyxia, changes in blood gases and hydrogen ion concentration are rapid. Investigations performed on newborn dogs and monkeys have shown that the oxygen content of arterial blood falls to near zero in 2½ minutes, while the *p*H declines at a rate of nearly 0.1 *p*H unit per minute,[63,64] initially due to accumulation of carbon dioxide and, subsequently, due to the end products of anaerobic glycolysis.

A significant correlation between the acid-base state of the infant at birth and its clinical condition has been demonstrated.[55] Measurements of oxygen concentrations were not rewarding, which is not surprising in view of the aforementioned complete exhaustion of arterial oxygen after a brief period of asphyxia. Animal experiments have subsequently indicated that the hydrogen ion concentration of the blood and tissues is the single most important factor that determines survival during asphyxia. In fetal rhesus monkeys asphyxiated immediately after delivery by hysterotomy, correction of arterial *p*H maintained the heart rate and blood pressure, and either prevented or minimized morphologically recognizable brain damage.[65] Rarely, fetal acidosis is unrelated to fetal hypoxia or asphyxia but results from maternal acidosis due to increased muscular activity, dehydration, or starvation. This form of fetal acidosis is not ominous *per se*, but it reduces fetal tolerance to subsequent episodes of asphyxia.

Biochemical Monitoring

Assessment of the acid–base state of the fetus during labor first became possible in the early 1960's with the development by Saling of a fetal capillary blood-sampling technique.[66] Blood is usually obtained from the scalp but may equally well be collected from the buttocks in a breeech presentation. The patient is placed in the lithotomy position, and a conical endoscope is introduced into the vagina. With a light source attached, the endoscope is advanced through the cervix and applied against the skin of the fetus. The skin is dried with cotton swabs and covered with a thin layer of silicone jel in order to minimize the adhesive forces of the surface and facilitate globule formation by the blood sample. A puncture incision is made with a

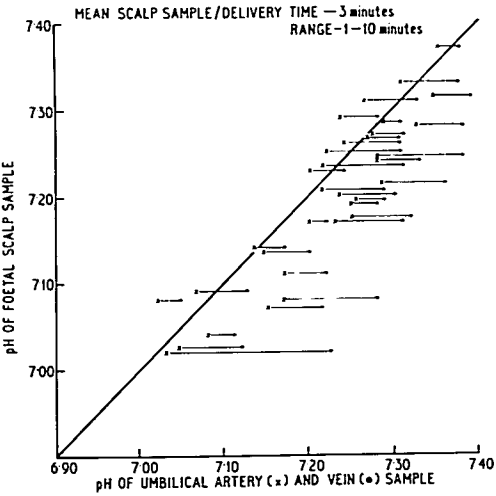

FIG. 6. Comparison of values for pH in fetal scalp blood taken just before delivery, and pH in the umbilical artery and vein blood at birth. Mean scalp sample-to-delivery time was 3 minutes (range 1 to 10 minutes). Reproduced from Bowe ET, Beard RW, Finster M, et al: Reliability of fetal blood sampling. Am J Obstet Gynecol 107: 281, 1970, with permission.

guarded blade and the drop of blood which appears is collected into heparinized glass capillary tubes. Determinations of pH, P_{CO_2}, P_{O_2} and base excess are carried out immediately with the use of appropriate microelectrodes.[67] A half-full capillary tube (0.12 ml) is necessary for complete acid–base determination. For determination of pH only 0.04 ml is required. After collection of the sample the incision site is compressed with a dry cotton swab for a few minutes to ensure hemostasis.

The reliability of capillary blood in the assessment of the acid–base state of the fetus has been established in several ways. In animal studies using pregnant ewes or monkeys, blood samples were obtained simultaneously from the scalp, the carotid artery, and the jugular vein of the fetus.[68,69] These studies showed that the pH of the scalp sample lies between arterial pH and venous pH. In the fetal monkeys, the mean value for the scalp blood pH was 0.017 higher than that of the venous blood and 0.028 lower than that of the arterial blood. In man, a high correlation was demonstrated between pH of fetal capillary blood immediately before delivery and pH of umbilical vein and umbilical artery blood obtained at the time of delivery (fig. 6), indicating that even in the second stage of labor blood flow through the fetal scalp is not sufficiently impaired to alter pH significantly.[70] However, the formation of a caput succedaneum as a result of severe head compression may be associated with an abnormally low pH of blood in scalp capillaries, due to local stasis.[71]

Studies based on serial fetal blood sampling confirmed the decline in fetal blood pH and increase in P_{CO_2} occurring even during normal labor and delivery.[72,73,74] A fetal capillary blood pH of 7.20 was defined as the lowest limit of normal,[72] while values between 7.20 and 7.24 were considered "preacidotic."[74] The validity of this classification was upheld by a good correlation between the pH of fetal samples collected shortly before delivery and subsequent Apgar scores of the babies at 2 minutes of life (fig. 7).[73] When pH was 7.25 or higher, 92 per cent of infants had Apgar scores of 7 or more. When pH was less than 7.16, 80 per cent of babies had Apgar scores of 6 or less. Figure 8 indicates, however, that a single measurement of fetal blood pH may be misleading.[70] Segment A contains values obtained from those infants who had relatively normal acid–base states during labor

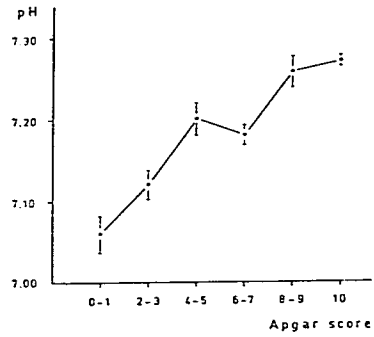

FIG. 7. Mean *p*H values for paired Apgar scores. Vertical bars represent ± SE Reproduced from Beard RW, Morris ED, Clayton SG: *p*H of foetal capillary blood as an indicator of the condition of the foetus, J Obstet Gynaecol Br Commonw 74:815, 1967, with permission.

but were depressed at birth. Low Apgar scores observed in this group may have been due to a number of factors, including sedative drugs, infection, prematurity, and congenital anomalies. Segment D contains values obtained from those infants who were acidotic but vigorous at birth. Maternal acidosis was the major factor contributing to the values in this group.

To distinguish acidosis originating in the mother from asphyxial acidosis in the fetus one must compare the base deficit in the mother with that in the fetus.[70,75] When the mother develops metabolic acidosis but placental function remains unimpaired, transfer of respiratory gases and of hydrogen ions proceeds normally and the difference between fetal and maternal base deficit (ΔBD) is small (mean 2.3 mEq/l). During fetal asphyxia associated with impairment of placental circulation there is accumulation of hydrogen ions in the fetal blood, resulting in ΔBD in excess of 4.6 mEq/l.

Capillary blood obtained from the presenting part of the fetus can be used for determinations other than acid–base balance. For example, measurements of bilirubin levels and hematocrit during labor can be used as an indicator of the urgency of the need for exchange transfusion in the immediate postpartum period. Furthermore, donor blood can be crossmatched against fetal blood obtained by this technique.

Biophysical Fetal Monitoring

Biophysical monitoring provides the physician with a continuous source of data relating to the fetus.[76,77] A fetal monitor is a two-channel recorder of instantaneous fetal heart rate (FHR), along with associated uterine activity, both sets of data being necessary for the proper evaluation of labor. Depending on the method of obtaining fetal data, biophysical fetal monitoring can be characterized as direct or indirect.

Direct Fetal Monitoring

This type of surveillance is the most reliable because the fetal signal for the computation of instantaneous heart rate (fetal "R" wave) is obtained from an electrode attached to the fetal scalp (fig. 9). Intrauterine pressure is recorded continuously by a strain gauge, attached to a small catheter inserted trans-

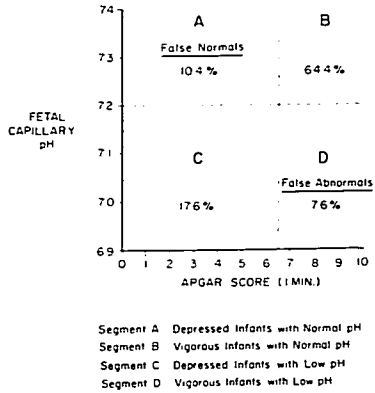

FIG. 8. Fetal *p*H as an index of infants' condition at birth in 355 patients during labor. Reproduced from Bowe ET, Beard RW, Finster M, et al: Reliability of fetal blood sampling, Am J Obstet Gynecol 107: 285, 1970, with permission.

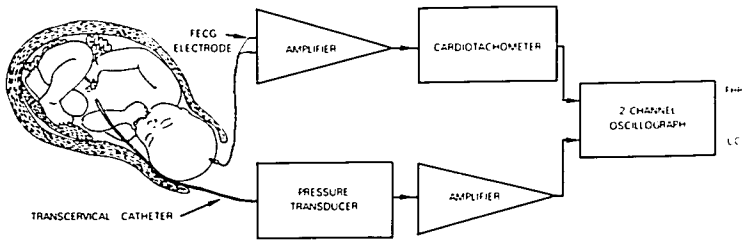

FIG. 9. Diagram of a direct fetal monitoring system. Reproduced from Freeman RK: Intrapartum fetal evaluation, Clin Obstet Gynecol 17:84, 1974, with permission.

cervically into the amniotic cavity. This system provides accurate and quantitative information on frequency, duration, and intensity of contractions. However, it requires that the membranes be ruptured, cervical dilatation be at least 1.5 cm, and that the presenting part be dipping into the true pelvis. This type of direct monitoring is obviously limited to the true intrapartum period.

INDIRECT FETAL MONITORING

The indirect form of fetal surveillance relies on data obtained from transducers applied to the maternal abdomen overlying the gravid uterus. The following three systems are used to obtain FHR recordings: electro-, phono-, and ultrasound cardiography (fig. 10). Problems of separating fetal and maternal electrocardiographic complexes recorded from the maternal abdominal wall limit the applicability of this approach.[78] The use of a microphone is also limited because of a poor noise-to-signal ratio, particularly during uterine contractions. Thus, the most consistent method of obtaining fetal data from the maternal abdomen is by means of ultrasound cardiography.[79] The ultrasound signal indicating the fetal heart activity is composed of four approximately equal-sized signals derived from opening and closing the mitral and aortic valves. Any one of these four signals may be randomly selected by the machine. Random selection introduces certain inaccuracies into the continuous computation of the FHR.[78]

Recording of uterine activity by the indirect system is obtained from a tocodynamometer, applied to the maternal abdomen, and triggered by the changing shape of the uterus during the contraction. Data thus obtained are quantitative only in respect to the frequency of contractions but semiquantitative or nonquantitative in respect to their duration and intensity. The indirect method has the distinct

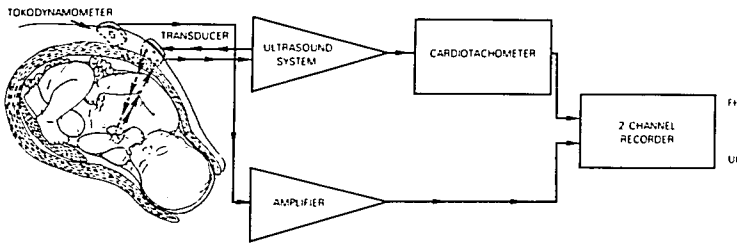

FIG. 10. Diagram of an indirect fetal monitoring system. Reproduced from Freeman RK: Intrapartum fetal evaluation, Clin Obstet Gynecol 17:84, 1974, with permission.

advantages of simplicity and non-invasiveness.

Fetal well-being is determined by interpretation of the recordings, taking into consideration the following variables: 1) absolute heart rate, 2) beat-to-beat variability, 3) periodic patterns, and 4) uterine activity.

Rate

By scanning a tracing the absolute FHR can be determined immediately. Normally it varies between 120 and 160 beats per minute. Persistently elevated rates may be associated with chronic fetal distress, maternal fever,[80] and administration of drugs such as atropine. Abnormally low rates may be encountered in fetuses with congenital heart block.

Beat-to-beat variability

The single best method of determining fetal well-being at present is provided by graphic evaluation of the beat-to-beat variability of heart rate computed from the "R" wave intervals of the fetal ECG.[81,82] The fetal heart rate is under the control of the cerebral centers, reflexes and the integrated parasympathetic and sympathetic nervous system.[83] When these divisions of the nervous system are functioning normally, variability will also be normal and the fetus is uniformly in good condition. Conversely, depression or damage of the fetal nervous system may result in minimal to absent variability (fig. 11), which frequently occurs as the manifestation of fetal hypoxia or drug effect.[84,85,86] Drugs most frequently associated with this phenomenon are: diaze-

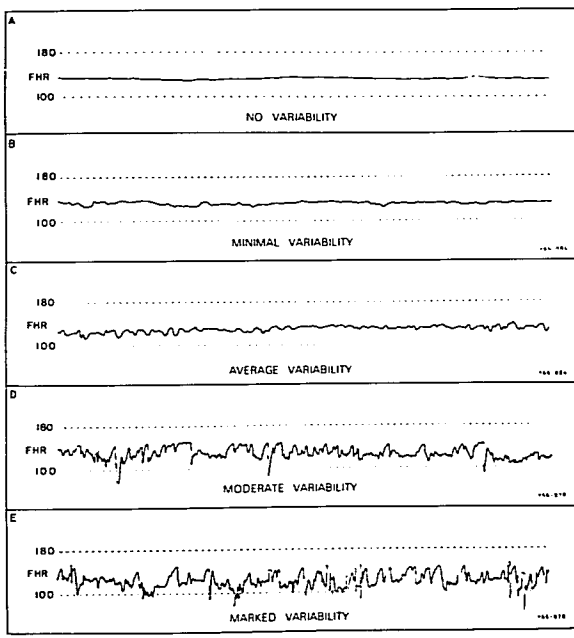

FIG. 11. Gradations of fetal heart rate variability. Reproduced from Hon EH: Biophysical intrapartal fetal monitoring, Clin Perinatol 1:153, 1974, with permission.

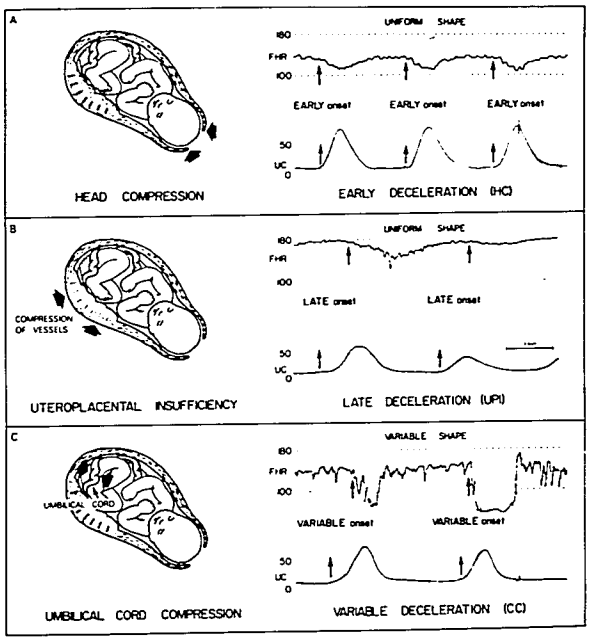

FIG. 12. Classification and mechanisms of fetal heart rate patterns. Reproduced from Hon EH: An Atlas of Fetal Heart Patterns, New Haven, Conn., Harty Press, 1968, p 49, with permission.

pam, local anesthetics, narcotics, and barbiturates. It is occasionally observed in an otherwise normal full-term fetus. Furthermore, this loss may normally be seen in a premature fetus and during fetal sleep cycles.

Studies comparing beat-to-beat variability with fetal acid–base analysis indicate a good correlation between the two techniques.[81,87,88]

The inherent error introduced into the computation of the FHR by phonocardiography or ultrasound cardiography renders these techniques unreliable for the determination of beat-to-beat variability. However, if flattening or smoothness of the fetal heart rate is detected with either of these systems, one should be suspicious of a potentially depressed fetus. Attempts to evaluate the integrity of the fetal nervous system by the use of continuous fetal electroencephalography are being made[89] but, to date, this method has been relatively unrewarding for widespread clinical use.

Periodic patterns

The second most important method of assessing fetal well-being is by the evaluation of periodic patterns. A transient alteration of fetal heart rate associated with a contraction is called an acceleration or a deceleration, and is generally known as a "periodic" pattern. Periodic patterns should be distinguished from baseline tachy- and bradycardia, which are deviations from the normal heart rate (120–160/min) occurring between uterine contractions. Fetal heart rate acceleration probably results from sympathetic activation

secondary to the stress of a contraction. Generally, acceleration is not a cause for concern.

There are three major forms of fetal heart rate decelerations, early, late, and variable (fig. 12).[84,90]

Early deceleration. This uniform wavelike deceleration or slowing of the fetal heart rate starts with the onset of contraction, reaches its lowest point at the acme, and returns to baseline level at the termination of the contraction. The FHR usually does not fall below 100 beats per minute. This deceleration is thought to be caused by vagal stimulation secondary to compression of the head. It is not ameliorated by increasing fetal oxygenation, but is blocked by the administration of atropine. Most importantly, this pattern is not associated with deterioration of fetal acid–base status or with poor neonatal outcome.

Late deceleration. This is a uniform wavelike slowing of the heart rate; however, the onset of deceleration is apparent after the onset of contraction. The slowest rate is reached after the acme of contraction, and FHR returns to baseline level after the contraction has terminated. The etiology of this pattern is thought to be myocardial hypoxia resulting from utero-placental insufficiency.[91] The severity of this phenomenon can be estimated by measuring the interval between the onset of the contraction and the initiation of FHR deceleration.[92] The longer the interval the better the condition of the fetus. The pattern of late deceleration can be corrected by improving fetal oxygenation. If this pattern is repetitive, continuous, and progressive in severity, there is a very significant correlation with a deteriorating fetal acid–base status. The primary factors contributing to the appearance of late decelerations include maternal hypotension, uterine hyperactivity, and chronic utero-placental insufficiency (diabetes, hypertension).

Variable deceleration. This is the most common periodic pattern observed in the intrapartum period. As the term indicates, it is variable in shape and onset. It may begin before, with, or after the onset of the contraction. Characteristically, the decelerative pattern is sharp, angular and saw-toothed in shape. Variable deceleration is often preceded and followed by a slight acceleration. Almost uniformly the FHR falls below 100 beats per minute. It is thought to be caused by umbilical cord compression and the resulting cardiovascular reflexes in the fetus. Atropine diminishes the severity of variable decelerations. Administration of oxygen to the mother is without effect. If the variable pattern is not severe and repetitive there is usually only minimal alteration of the fetal acid–base status and the condition of the newborn. If cord compression becomes more severe, prolonged and repetitive, fetal acidosis may develop. Should the FHR fall below 60 beats per minute, a brief cardiac arrest (lasting from 2 to 8 seconds) may occur. Rarely vagal stimulation may be of sufficient magnitude for the fetus to suffer a permanent cardiac arrest.[93]

Sinusoidal pattern. This fourth pattern has been reported by some investigators.[94,95] It is an undulating heart rate that may be observed in the ante- or intrapartum period and sometimes is seen in the fetuses of Rh-immunized mothers. Although the significance of this FHR pattern is unknown, it has been reported to occur in association with poor neonatal outcome.

Uterine Activity

During active labor, uterine contractions should occur every 2–3 minutes, with peak intrauterine pressures of 50 to 80 torr and resting pressures of 5 to 20 torr between contractions. With the insertion of a catheter into the amniotic cavity, reliable data pertaining to the intensity of uterine contractions can be obtained.[96] This form of continuous surveillance is invaluable in avoiding excessive stimulation of the uterus when oxytocin is used. It is also useful in evaluating the effect upon uterine contractions of drugs administered to the mother. For instance, uterine activity frequently increases significantly following a rapid intravenous injection of meperidine and promethazine.[97] Last, abnormally frequent uterine contractions and elevated baseline tone can aid significantly in the diagnosis of abruptio placentae in the presence of third-trimester bleeding.

An attempt to correlate continuous FHR data obtained during 30 minutes preceding

delivery with the condition of the newborn has been made.[98] The ability to predict a vigorous infant (Apgar score 7 or better at 5 minutes of life) was as high as 99.2 per cent. In contrast, the birth of a depressed infant (Apgar score of 6 or less) was predictable in only 42.9 per cent of cases. In other studies, FHR variability patterns correlated with the incidence of RDS and death among low-birthweight infants.[99,100]

Fetal scalp sampling was introduced in our institution in 1963, and was followed, in 1969, by the adoption of biophysical monitoring. The two approaches to intrapartum fetal surveillance were integrated and the following general guidelines for management have evolved:

1) In high-risk pregnancies, maternal and fetal *p*H and blood gases should be determined, whenever possible, before instituting biophysical monitoring.

2) All patients should be monitored by direct techniques as soon as possible.

3) In the face of a mixed pattern, the most ominous pattern should indicate the course of action.

4) When loss of beat-to-beat variability cannot be explained by the prior administration of a drug and persists for more than 30–45 minutes, a sample of fetal blood should be collected for acid–base determinations.

5) Repetitive early decelerations in which the FHR does not fall below 100 beats per minute and which are not associated with fetal acidosis require no further investigation.

6) When repetitive late decelerations are detected, the following procedures should be initiated: a) stop oxytocin if it is being infused; b) administer oxygen at 6–7 liters per minute by a tight-fitting face mask; c) correct hypotension, if present, by alteration in maternal position, administration of a vasopressor (*e.g.*, ephedrine, 15–25 mg, iv), or expansion of intravascular fluid volume (*e.g.*, lactated Ringer's solution, 500–1000 ml, iv); d) collect free-flowing maternal venous and fetal capillary blood samples for acid–base determinations.

If these measures do not correct the pattern and the *p*H is less than 7.20 on two occasions, separated by 5- to 15-minute intervals, the fetus should be delivered immediately by the safest route. If the pattern persists and the *p*H is between 7.20 and 7.24, *p*H is redetermined every 15–20 minutes until the *p*H and the FHR pattern are corrected or the fetus is delivered. If the FHR pattern persists and the *p*H is 7.25 or greater, *p*H must be redetermined every 15–20 minutes until the FHR pattern is corrected or the fetus delivered.

7) When severe repetitive variable decelerations are noted, the following additional steps are to be taken: a) perform a vaginal examination to exclude a prolapsed cord; b) alter maternal position to left lateral, right lateral, Trendelenburg, reverse Trendelenburg, or knee chest—to alleviate the cord compression; c) determine fetal blood P_{CO_2} and base excess, in addition to the customary *p*H, in order to differentiate between respiratory acidosis and a more alarming metabolic acidosis. If the pattern is worsening and there is indication that severe metabolic acidosis is developing, the fetus should be delivered immediately.

Infants of diabetic and pre-eclamptic mothers are especially at risk and may die prior to the onset of labor. Since it became known that the FHR reacts in a characteristic manner to uteroplacental insufficiency during labor, it was only logical to attempt to detect fetal hypoxia in the antepartum period by indirect monitoring of the FHR following spontaneous or induced uterine contractions.[101,102]

The testing procedure requires the infusion of small doses of oxytocin in order to achieve three contractions within a 10-minute period.[103] Absence of late decelerations indicates that the respiratory function of the placenta is adequate and that the fetus is in good condition. Conversely, the presence of late decelerations suggests a fetus in jeopardy.[104–106] To date the contraction stress test is probably the best single test of fetal condition between the twenty-eighth and forty-fourth weeks of gestation.

A number of investigators feel that the condition of the fetus can be evaluated by the reaction of the FHR to stimuli such as pressure, noise and fetal movements. If the FHR responds with an acceleration, the fetus is felt to be in good condition. Lack of acceleration suggests fetal jeopardy and an oxytocin stress test should be performed.[107] Experience with

this form of antepartum FHR monitoring is limited and requires further evaluation.

Simultaneous recording of the fetal electrocardiogram and use of differential ultrasound cardiography for the determination of the pre-ejection phase of the fetal cardiac cycle has recently been introduced for the evaluation of myocardial performance.[108,109] The technique, which is applicable before or during labor, relies on determination of the measured interval between an electrical (*e.g.*, the "Q" wave) and a mechanical event (*e.g.*, the opening of the aortic valve). Prolongation of this systolic time interval indicates fetal deterioration.

The above-mentioned methods of ante- and intrapartum fetal surveillance are becoming ever more widely applied and appreciated. Their contribution to an improvement in perinatal outcome is reflected in the statistics from our own institution, where, in the years 1962–1974, the perinatal mortality among service patients decreased from 25 to 13 per 1,000. These results have been achieved in spite of an increase in the percentage of high-risk pregnancies.

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