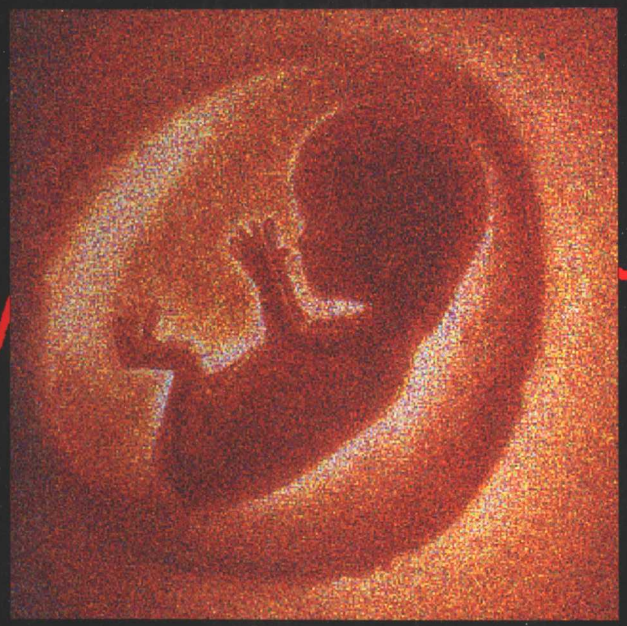
MORE EXERCISES IN FETAL MONITORING



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Preface

The widespread use of electronic fetal monitoring (EFM) and ultrasound for fetal surveillance has enhanced our understanding of fetal physiology and anatomy. These techniques have facilitated considerably our ability to diagnose potential abnormalities and at the same time have enhanced confidence in the diagnosis of normalcy. They have also permitted a better understanding of the limitations of the designations of "high-risk" and "low risk" pregnancy. In the ensuing material, I attempt to illustrate the problems of determining whether a pregnancy is at high or low risk. Indeed, I even classify as high risk those patients who are specifically called "low risk." The reader may not wish to indulge in this seeming persiflage, but my meaning should be clear. I believe we cannot meaningfully classify obstetrical patients as at high or low risk without incorporating fetal testing both before and during labor into the risk calculus. As many of the following tracings and commentaries suggest, too many low risk patients become high risk during labor. Low-risk and high-risk patients contribute almost equally to the census of neonatal intensive care units.

Anticipation, prevention, and timely intervention in the distressed fetus are the premise of fetal surveillance, but that is not what such monitoring does best. It best provides reassurance that fetal milestones of growth/maturity, oxygen availability, and neurologic function have been reached and that no intervention is necessary on behalf of the fetus. Pertinently, no single test, antepartum or intrapartum, informs us about all these parameters. The most popular antepartum tests are the Nonstress Test (NST) and the Biophysical Profile (BPP). The waning popularity of the contraction stress test (CST) parallels the evolving emphasis in fetal heart rate (FHR) pattern interpretation on the organization and types of responses to such intrinsic provocations as uterine contractions and fetal movement and such extrinsic provocations as vibroacoustic stimulation and maternal ingestion of food.

Fetal behavior receives only nominal attention in fetal monitoring textbooks, lectures, and workshops, where far more time is spent on decelerations, variability and the diagnosis and treatment of fetal distress. This emphasis on decelerations and variability and the search for hypoxia has resulted in a set of arbitrary guidelines for intervention and compromised the acceptance of electronic fetal monitoring. But the fetal condition cannot be truly appreciated by a "phenomenologic" approach to FHR patterns where conclusions are reached from an assessment of the number of accelerations, the type of decelerations, or the amount of variability. The term fetus, with its full complement of autonomic tone, is capable of producing a seemingly bewildering array of epochal heart rate patterns, including decelerations, which defy ready interpretation using a syntax that tends to view decreased variability as hypoxia and decelerations as a "step on the road to death." I believe we must stop focusing so narrowly on hypoxia and better appreciate the insights into fetal behavior as well as hypoxia that current surveillance permits. This book, the second in the series, is mostly about fetal behavior and its impact on FHR patterns during both antepartum and intrapartum testing.

This focus on fetal behavior does not attempt to minimize the potential significance of fetal hypoxia or its relationship to FHR patterns. Fetal asphyxia is a biochemical diagnosis with increased Pco^2 and base deficit and decreased Pco_2 and pH. Similarly, I believe that those definitions of fetal distress or fetal hypoxia that require significant neonatal distress and handicap as part of the definition should be abandoned.

FHR patterns during labor will not fail to reveal *any* significant hypoxia from any source. Depending on the source and the rapidity, the previously normal fetus responds to hypoxia first with specific decelerations (late, variable, or prolonged), and then (unless the deceleration is sustained) a rise in baseline heart rate and loss of variability before it recovers. In the absence of these usually transient changes in baseline rate, decelerations should not be considered asphyxial. The fetus that demonstrates either prolonged deceleration without recovery or progresses to a high baseline rate and decreased variability with persistent decelerations is likely undergoing significant hypoxia and may ultimately be injured or die. But whether it is actually injured at the time will, alas, take some time to tell. The FHR patterns observed during acute hypoxia should be used to "predict" subsequent neurologic handicap. As a principle, injury must be separated from asphyxia. Simply stated, one cannot determine neurologic handicap in the presence of asphyxia any more than one would be confident in predicting subsequent neurologic integrity of a person being rescued from near-drowning. I am unaware of any published evidence that suggests that a briefly asphyxiated fetus who has recovered to its previously normal baseline rate and variability within a short period of time sustains injury during that episode.

In addition to responding to asphyxial provocations, the fetus is capable of producing discernible, epochal patterns of sleep (rest), wakefulness (activity), breathing, sucking, mouthing movements, and provides in these responses insight into its neurologic functioning and maturity. In the very premature fetus there is little to distinguish these episodes. Because of the relatively high baseline rate and limited autonomic tone and maturity of the premature fetus, behavioral patterns at this stage of gestation are far more chaotic and less well organized, and the deviations in heart rate they produce less dramatic than in the older fetus. As the fetus matures, first accelerations appear, then rest-activity cycles;

and as term approaches, behavioral patterns become more complex but more obvious. In this book the words "fetal behavior" refer to recurring FHR patterns seen on the monitor strip. During labor this is synonymous with the predictable waxing and waning of variability, accelerations, and even decelerations that appear despite the stress of uterine contractions.

To take advantage of our increasing understanding of fetal behavior patterns, I have redefined the reactive NST to include analysis beyond the frequency and amplitude of accelerations. As redefined, the reactive NST not only permits the conclusion about fetal well-being (absence of asphyxia, unstressed) but normal neurologic behavior as well. This does not exclude gross abnormalities of the central nervous system. I emphasize that the reactive NST pattern cannot be counterfeited. I have also added an intermediate test result to the NST. As the fetus deteriorates, it is variability, not accelerations, that are lost first. Movements and accelerations become isolated, variability diminishes, and the sleep phase of the sleep-wake cycles is prolonged until accelerations disappears. This represents *fetal malaise,* an effect of medication, immaturity, anomaly or deterioration.

What is abnormal behavior? And how does one anticipate neurologic deficit? Abnormal behavior is most readily estimated by the *persistent* lack of variability, and is even better appreciated by studying the fetus on real-time ultrasound. Abnormal behavior may develop as a result of genetic abnormalities, malformations, as well as medication or drugs, alcohol, or a deteriorating biochemical environment; occasionally hypoxia plays a role. It appears that most fetuses who are destined to develop neurologic handicap do not show abnormal FHR patterns. As will be seen in some of the tracings, fetal anomaly or injury sometimes produces unique or bizarre FHR patterns. All aspects of the tracing may be affected, including the behavioral pattern, the baseline rate and variability, and the pattern of accelerations and decelerations. It may be that prenatal or intrapartum FHR patterns may be the most reliable determinant of subsequent neurologic outcome. In most cases, when this pattern is discovered in labor, little can be done to change the outcome. In this respect FHR patterns before and during labor may provide insight into the timing of neurologic injury. On the other hand, observation of FHR patterns during labor may provide clues to the potential development of injury. But these potential benefits and insights of monitoring are secondary. The primary role of monitoring is, again, the reassurance that all is well with the fetus. With such reassurance, administration of oxytocin or epidural anesthesia and the timing of delivery is made safer thereby.

In the outline I refer to "placental insufficiency" in two defined senses. *Respiratory insufficiency* refers to compromise of the fetal oxygen supply, a potential development at any time, but more common during labor. *Nutritional insufficiency* refers to the inability to provide adequate nourishment to the fetus, and is manifested as diminished growth, and in some instances as diminished amniotic fluid volume.

Amniotic fluid volume has been incorporated into the biophysical profile (BPP) and our preferred scheme of antepartum testing as a measure of *nutritional placental function,* not diminution in oxygen availability. Diminished amniotic fluid volume, unrelated to an anomaly or ruptured membranes, develops as part of a generalized depletion of water from the fetal compartment: from the skin, the cord, the blood volume. It does not usually represent fetal hypoxia or deterioration in *respiratory placental function.* Nutritional and respiratory deficiencies of the placenta need not develop simultaneously. It may seem paradoxical that the fetus may not flourish nutritionally despite adequate oxygenation. The clinical model here is the postmature infant, who, through dysmature, usually shows no respiratory placental insufficiency in the form of late decelerations.

Antepartum or intrapartum, test results based of FHR patterns show a relatively high false positive rate but low false negative rates. Such results have given rise to the well-known aphorism in perinatal medicine that "it is easy to make a good baby look bad, but difficult to make a bad baby look good." As a result there are numerous arguments in the literature over which test or which sequence or which criteria are "best." But which management strategy and which testing scheme is superior remains unresolved because no controlled studies have yet compared the various tests. Differences in methods, test criteria, and intervention strategy make comparisons of published data less than ideal. I believe that *testing is more important than the specific test used.* The more criteria used to define abnormality and the more often testing is carried out, the better the results. Testing also changes risk status in that the outcome of tested high-risk patients is better than that of untested, low-risk patients.

For its obvious but disputed benefits, electronic fetal monitoring cannot reliably predict outcome of problems unrelated to oxygen deprivation or those in which behavior is unaffected. Thus intrauterine growth retardation and congenital anomaly (even of the brain) usually escape detection on electronic fetal monitoring. Many hydrocephalic fetuses and even an occasional anencephalic fetus may produce seemingly normal behavioral patterns. On the other hand, there are compelling relationships between abnormal FHR patterns and the subsequent development of cerebral palsy and other forms of neurologic handicap.

The issue of routine testing has usually been deliberated as the value of the research for abnormality. This seems the wrong perspective. In an era when electronic fetal monitoring is thought to be "equivalent" to auscultation, the broadening inclusion of fetal behavior patterns into the analytic scheme seems almost anachronistic. Further, it

seems, well, awkward, to continue to espouse "routine" electronic fetal monitoring both before and during labor. Testing of the individual fetus is necessary, especially during labor, to define its risk status, and may be viewed as the "well-fetus" examination. In advocating routine intrapartum monitoring I do not advocate the attachment of this "tube anchor" to the patient for the duration of her labor. Rather, I advocate, along with many others, the use of electronic fetal monitoring as an admission test when the patient first arrives. If the fetus satisfies the criteria of well-being, the monitor may be removed, to be replaced under specific situations.

I present the ensuing outlines and tracings to facilitate the understanding of FHR patterns both before and during labor. I plead guilty to minimizing the distinctions between antepartum and intrapartum FHR patterns. While it was once believed that sleep-wake cycles and fetal breathing were diminished in labor, more recent studies reveal that the fetus does cycle during early labor.

Further, I encourage the reader to assess all aspects of the tracings and appraise not only the presence or absence of fetal hypoxia but also estimate such features as gestational age and fetal responsiveness (behavior) from the clues available. The reader should formulate an opinion before reading about the outcome or considering my interpretation of the tracing. This approach attempts to maintain the uncertainty of outcome that is always present in clinical medicine. FHR tracings do not always permit accurate prediction of outcome, but they always yield the opportunity for intelligent analysis, and even reasoned disagreement.

Finally, I dare to hope that these discussions will diminish the dread of medicolegal encounters involving the allegation that a fetus was negligently injured from "perinatal asphyxia." I expect the reader to take away the following messages: That a fetus is injured does mean that it was asphyziated. That a fetus is asphyxiated does not mean that it was injured. That a fetus is injured from asphyxia does not mean that it was reasonably preventable. A reasonable management scheme is based on one of several reasonable options and takes reasonable advantage of all of the clinical information, including the FHR tracing. Such an approach, properly documented, will satisfy the most demanding standard of care, irrespective of the outcome.

Barry S. Schifrin

Contents

INTRODUCTION

HIGH-RISK PREGNANCY

DEFINITION

Pregnancy with an increased risk of poor outcome.
Alternatively, a definable segment of the population which accounts for a disproportionate share of the poor outcome.

DESIDERATA

Numbers must be manageable.
Must define risk early enough to provide therapy.
Therapy must be available.
"Low-risk" must equal negligible risk.
Contemporary medicine has not fulfilled these desiderata.
The problem lies with the "low risk" not the "high risk."

MEDICAL DISORDERS

Hypertension
Diabetes mellitus
Heart disease
Renal disease

OBSTETRICAL DISORDERS

Labor complications
Erythroblastosis
Abruptio placentae
Preeclampsia
Genital tract anomalies
Hemorrhage
Trauma
Infection

SOCIAL/DEMOGRAPHIC FACTORS

Age, parity, race
Marital status, nutrition, child spacing
Socioeconomic class, emotional factors
"Low-risk pregnancy"

COMMENTS ON HIGH-RISK STATISTICS (Table 1)

A. Less than 50% of the population remained **low-risk** throughout pregnancy.

TABLE 1.
High-Risk Statistics*

Risk Status†	Patients, No. (%)	Mortality, No./1000	Morbidity, No. (%)	DQ Mean, 1 yr
Low/low	340 (46)	1/3	22 (6.5)	106
High/low	135 (18)	3/22	16 (11.8)	105
Low/high	144 (20)	5/35	35 (24.3)	88
High/high	119 (16)	16/145	42 (35.0)	91

*From Hobel et al: 1976. Used by permission.
†Prenatal/intrapartal.
DQ = developmental quotient.

B. Thirty percent of patients initially classified **low-risk** antepartum became **high-risk** during labor.
C. Intrapartum **high-risk** carries the greatest jeopardy: intrapartum **low-risk** carries the least.
D. Antepartum low-risk patients account for about 50% of intrapartum **high risk** and poor outcome.
E. In certain centers specialized care to specific **"high-risk"** gravidas has produced outcomes comparable or better than **"low risk."**
   1. Benefit of specific care.
   2. Poverty of definition of "low risk."
F. A semantical paradox:
   1. "Low risk" = gravidas called "high risk."
   2. "High risk" = gravidas called "low risk."
   3. "Lowest risk" = gravidas receiving "optimal care."
G. Gravidas who receive no care are at highest risk.
H. Is any pregnancy **"low risk"?**

LIMITATIONS OF AVAILABLE CRITERIA

A. Clinical estimation of fetal weight:
   1. Grossly inaccurate—examiner bias.
   2. About 50% of twins not anticipated.
   3. About 50% of babies with intrauterine growth retardation (IUGR) not anticipated.
   4. Accuracy of prediction of IUGR only about 50%.
   5. Accuracy unrelated to experience.
B. Ultrasound estimation of fetal weight:
   1. Better than clinical estimation.
   2. Average error about 10%.
   3. Error greater at extremes of birth weight.
C. The "weighting game":
   1. The accuracy of estimating fetal weight lies not with how closely you predict the fetal weight, but how well you assign the patient to a proper management scheme.

2. As the estimated birth weight at which we are prepared to intervene for fetal benefit decreases, it is only necessary to decide whether the fetus is too small to profit from aggressive care.
3. An example: Assume that you are prepared to intervene, on indication, in a fetus whose estimated birth weight is 650 g. If the fetus weighs 675 g but your estimate is 635 g and you do not intervene because the fetus is "too small," you have assigned the fetus to the wrong management strategy—despite an error of 40 g (or 6%).
4. There are only two questions to be answered:
   Is the fetus too young to profit from enlightened care?
   Is the fetus too large to deliver from below?

D. Clinical auscultation of the fetal heart:
1. Intermittent.
2. Errors introduced: technique, listener bias.
3. Detection of fetal distress depends on:
   a. Rate signifying distress: 120, 100, 80 bpm.
   b. Fetal baseline heart rate
   c. Detection of contractions
   d. Duration and amplitude of decelerations
   e. Onset and duration of counting
4. Cannot assess variability.
5. Confined to period between contractions.
6. Does not predict early distress during labor or before.
7. Does not predict deterioration or mechanism of distress.
8. Experience unrelated to accuracy.
9. Unrelated to outcome.
10. Randomized controlled trials show no benefit of auscultation in prediction of fetal condition irrespective of attention or scheme.
11. Cannot reproduce fetal heart rate (FHR) patterns from auscultation (Miller et al.).
12. Clinically unrealistic.
13. Impractical; too expensive to provide sufficient nursing.

TO IMPROVE STATISTICS

A. Understand limitations of available methods of fetal evaluation.
B. Risk status of the fetus must be tested directly before assigning any mother to "low-risk status."

ELEMENTS OF FETAL SURVEILLANCE

A. Growth/nutrition:
1. Sequential ultrasonic mensuration:
   a. Biparietal diameter (BPD), femur length, abdominal circumference.
   b. Head/abdomen ratio.
   c. Bowel pattern, epiphyses, etc.
2. Amniotic fluid volume (AFV):
   a. Diminution in AFV with IUGR not related to hypoxia.
   b. No chronic hypoxic model produces oligohydramnios.

B. Oxygenation:
1. pH, blood gases—experimental percutaneous umbilical blood sampling (PUBS)
2. Contraction stress test [CST] and breast stimulation test [BST])

C. Neurological integrity—behavior:
1. Non-stress test (NST).
2. Biophysical profile.

D. Placental insufficiency:
1. Poorly defined term, as a minimum:
   a. **Respiratory placental insufficiency**
      (1) Transport of oxygen, gases, maintenance of pH.
      (2) Requires ongoing maintenance at high level.
   b. **Nutritional placental insufficiency:**
      (1) Transport of nutrients.
      (2) May be curtailed transiently without embarrassment.
   c. Paradox: May have interference of nutritional function without impairment of respiratory function, e.g. postdate

E. Questions, questions, questions
1. Questions *to ask yourself* before you test:
   a. What am I testing for?
   b. Can the information be obtained clinically?
   c. Can an abnormality be predicted specifically?
   d. What is the risk of the procedure?
   e. Can a clinical decision be based on the result?
   f. Are we monitoring the inevitable?
   g. Is the test simple, safe, rapid?
   h. Is the stress tolerable, transient, quantifiable?
   i. Is the end point measurable, reproducible, sensitive?
   j. Does the end point depend on gestational age?
   k. How much does it cost?
   l. Do I have a plan for each possible result?
2. Questions *not to ask the fetus:*
   a. How old are you?
   b. How much do you weigh?
   c. What's your lecithin-sphingomyelin (L/S) ratio?
   d. Do you have any decelerations?
3. Questions *to ask the fetus:*
   a. How are you doing in there?
   b. Are you behaving yourself properly?
   c. Is there something your mother can do?
   d. Would you prefer to be somewhere else?
   e. Do you have a burning desire to meet a pediatrician?

f. Are you too large for safe vaginal delivery?
g. Are you too premature to benefit from modern care?

NON-STRESS TEST

A. Technique for assessing fetal well-being by observing the fetal heart rate (FHR) response to spontaneous or induced fetal movement (FM). Includes epochal responses (rest-activity cycles).
B. Reactive pattern suggests:
   1. Normal neurological control.
   2. Adequate oxygenation (unstressed).
C. NST does not define:
   1. Nutritional growth failure.
   2. Anomalies.

CONTRACTION STRESS TEST

A. Technique for assessing fetoplacental respiratory reserve by observing the fetal heart rate (FHR) response to spontaneous or induced uterine contractions (UC).
B. Negative CST precludes hypoxia.
C. CST does not define:
   1. Nutritional growth failure.
   2. Anomalies.
   3. Pre-existing neurological injury.

BIOPHYSICAL PROFILE (BPP)

A. Technique for assessing fetal well-being by observing responses of fetal heart rate to fetal movement (NST), fetal body movement (FM), fetal breathing movements (FBM), fetal tone (TON), and quantifying amniotic fluid volume (AFV).
B. Placental grade included in some schemes.
C. Permits a general survey of intrauterine contents including presentation, position, BPD, placental localization, IUGR, anomalies.
D. Normal test suggests:
   1. Normal neurological control.
   2. Adequate oxygenation (unstressed).
   3. Nutritional adequacy.

INDICATIONS FOR TESTING

A. Patients at increased risk for placental insufficiency, e.g., diabetes, toxemia, hypertension, postdate.
B. When other examinations suggest fetal compromise:
   1. Suspect IUGR, oligohydramnios, multiple gestation.
   2. Meconium staining of amniotic fluid, etc.
C. When events or complaints dictate:
   1. Decreased fetal movement.
   2. Trauma, bloody amniocentesis.
D. Routine antepartum surveillance.

CONTRAINDICATIONS TO TESTING

A. NST/profile—none.
B. Contraindications to induction of contractions:
   1. Vaginal bleeding.
   2. Ruptured membranes.
   3. Previous cesarean section.
   4. Polyhydramnios.
   5. Multiple gestation.
   6. Incompetent cervix.
   7. Other contraindications to labor.

TESTING PROCEDURES

A. Patients should refrain from smoking; test after meals; position: semi-Fowler's, left lateral-tilt, avoid supine.
B. Carry out testing in a quiet room, free from distractions; take BP every 10 to 15 minutes; obtain first BP in either sitting or lateral position.
C. Position external FHR transducer for best recording.
D. Position external tocotransducer over the uterine fundus or fetal trunk or extremity (to record breathing movements).
E. Determine baseline FHR, variability, accelerations, decelerations, fetal movements, and uterine contractions.
F. Record name, date, time of day, medication, indication, vital signs, position, monitoring technique, etc.
G. If NST nonreactive after 20 minutes stimulate fetus:
   1. Abdominal palpation.
   2. Glucose-containing beverage to mother.
   3. Vibroacoustic stimulus (see below).
H. Vibroacoustic stimulation.
   1. Apparatus
      a. Electronic larynx (Western Electric)
      b. Acoustic stimulator
   2. Technique
      a. Apply to region of fetal head
      b. Single, short buzz
I. For CST, induce uterine contractions if:
   1. Spontaneous UC less than 3/10 minutes.
   2. No repeated late decelerations.

3. Nonreactive NST.
4. You are hoping to induce labor.

J. Oxytocin infusion:
1. Administer by constant infusion pump.
2. Initial rate—0.5 to 1.0 mU/minute.
3. Increase rate by 1 mU/minute every 15 to 30 minutes until there are three UCs lasting 40 seconds in 10-minute window.
4. Starting dosage and rate of oxytocin increase empirical; may need to start slower to avoid hypertonus.

K. Breast stimulation:
1. Ensure privacy.
2. Numerous variations on technique:
   a. Unilateral—bilateral.
   b. Exposed—unexposed.
   c. Warm towel—fingers only.
   d. Continuous—intermittent.
   e. Nipple roll—palpation—breast pump.
3. Minimum frequency of hypertonus with intermittent, unilateral palpation.

L. Discontinue oxytocin infusion or breast stimulation if:
1. Satisfactory test (positive or negative CST).
2. Unsatisfactory data.
3. Equivocal CST despite 1 hour of satisfactory UCs.
4. Infusion rate greater than 16 mU/minute (arbitrary); sometimes higher infusion rates necessary.
5. After 15 minutes of breast stimulation.
6. If contractions are:
   a. Less than 2 minutes apart or more frequently than three in 10 minutes.
   b. Last longer than 60 seconds.
7. Prolonged fetal deceleration.

M. Response to hypertonus:
1. Discontinue oxytocin or breast stimulation.
2. Lateral position.
3. Oxygen by face mask.
4. Check maternal vital signs.
5. Uterine relaxant (e.g., terbutaline).

N. Continue to monitor until contractions return to baseline level.

O. Irrespective of designation of test or stimulus applied (NST, CST, BST) evaluate for:
1. Movements and fetal responses thereto.
2. Contractions and fetal responses thereto.

P. BPP—perform general survey of intrauterine contents, including: presentation, position, biparietal diameter, placental localization; during this survey and for 10 to 30 minutes thereafter, FM, FBM are counted and TON determined.

Q. Monitoring twins:
1. Must monitor twins simultaneously.
2. Procedure facilitated by preliminary ultrasound.
3. If using two monitors, mark tracings simultaneously.

R. Documentation:
1. Record interpretation—and criteria.
2. Obtain official reading (if required).
3. Obtain consultation (if required).
4. Disposition—home, hospital, office, etc.
5. Save tracing—original, microfilm, or laser storage.
6. Annotate stimulation, etc.

COMPLICATIONS OF TESTING

A. CST:
1. Hypertonus, fetal distress.
2. Preterm labor (theoretical).

B. Vibroacoustic stimulation—all theoretical:
1. Produces sound and vibration:
   a. Represents energy input into uterus.
   b. Sound in utero may be louder than in air.
   c. Effects related to intensity, duration, and frequency of stimulation.
2. Mechanism of response may involve pain in fetus with release of catecholamines—disputed.
3. Generally regarded as safe:
   a. Apparently normal auditory function in babies
   b. Should probably avoid with oligohydramnios.

C. All: misinterpretation of results.

TIMING AND FREQUENCY OF TESTING

A. Begin testing at time when results will be acted on.

B. "Low risk"—at 32 to 34, 38, and 40 weeks (arbitrary).

C. "High risk":
1. Weekly, except:
   a. Semi-weekly:
      (1) Risk of oligohydramnios (IUGR, postdates).
      (2) Diabetes mellitus.
2. Daily: certain preterm premature rupture of membranes (PROM) unstable conditions.

D. No testing interval guarantees normal outcome.

RESPONSE TO ANTEPARTUM TESTS

A. Normal test results:
1. No fetal indication for intervention.
2. Repeat as clinically indicated.

B. Abnormal test results:
1. Continue monitoring.

2. Repeat test according to plan.
3. Induction of labor.
4. Cesarean section.

C. Documentation of thought process.

CLASSIFICATION OF RESULTS

NON-STRESS TEST

REACTIVE NON-STRESS TEST (NST-R)

Acceleration amplitude: At least 15 bpm from baseline to peak; before 32 weeks, use 10 bpm for 15 seconds.
Acceleration duration: At least 15 seconds from onset to return.
Acceleration frequency: Two or more FHR accelerations in 10 minutes. At least 2 accelerations must coalesce.
Variability: Average sleep-wake cycles.
Note: Auscultated accelerations acceptable.

NONREACTIVE NON-STRESS TEST (NST-NR)

Acceleration amplitude: Less than 15 bpm from baseline to peak.
Acceleration duration: Less than 15 seconds from onset to return, *or*
Acceleration frequency: Less than 2 in 10 minutes, *or* accelerations do not coalesce.
Variability: Persistently decreased.
Note: If intermediate classification used then NST-NR refers to: absent accelerations *and* decreased variability.

INTERMEDIATE NON-STRESS TEST (NST-I)

Acceleration amplitude: At least 15 bpm from baseline to peak.
Acceleration duration: At least 15 seconds from onset to return.
Acceleration frequency: At least 1 in 10 minutes; accelerations do not coalesce.
Variability: Persistently decreased.

SINUSOIDAL NON-STRESS TEST (NST-S)

Sine wave amplitude: 5 to 15 bpm (some greater).
Sine wave frequency: 3 to 6 cycles per minute (cpm).
Absent variability and reactivity; variant of NST-NR.

UNSATISFACTORY NON-STRESS TEST (NST-U)

Technically poor tracing: precludes detection of accelerations.

CONTRACTION STRESS TEST

NEGATIVE CONTRACTION STRESS TEST (CST-N)

Absent decelerations with three palpable UCs in 10 minutes.

POSITIVE CONTRACTION STRESS TEST (CST-P)

Recurrent late decelerations with three UCs in 10 minutes.

EQUIVOCAL CONTRACTION STRESS TEST (CST-E)

Inability to define either a negative or positive CST within 1 hour of satisfactory testing.
Recurrent non-late decelerations.

UNSATISFACTORY CONTRACTION STRESS TEST (CST-U)

Technically poor tracing
or
Inability to obtain three UCs in 10 minutes within 1 hour.

BIOPHYSICAL PROFILE

FETAL BREATHING MOVEMENTS PRESENT (FBM-P)

One or more episodes of fetal breathing lasting at least 60 seconds within a 10-minute period.

FETAL BREATHING MOVEMENTS ABSENT (FBM-A)

No episode of fetal breathing within 10 minutes.

FETAL BODY MOVEMENTS PRESENT (FM-P)

At least three discrete episodes of limb or trunk movements within a 10-minute period. Simultaneous movements are counted as a single movement.

FETAL BODY MOVEMENTS DECREASED (FM-D)

Fewer than three discrete FMs in 10-minute period.

FETAL MUSCLE TONE NORMAL (TON-N)

Upper and lower extremities in full flexion.
Trunk in position of flexion and head flexed on chest.
At least one episode of extension of extremities or extension of spine with return to position of flexion.

FETAL BODY MOVEMENTS DECREASED (TON-D)

Extremities extended or partially flexed.
Fetal spine extended, hand open.
Fetal movement not followed by return to flexion.

AMNIOTIC FLUID VOLUME NORMAL (AFV-N)

Fluid evident throughout uterine cavity.
Largest vertical pocket of fluid > 2 cm.
Amniotic fluid index > 10.

AMNIOTIC FLUID VOLUME DECREASED (AFV-D):

Fluid absent in most areas of uterine cavity.
Largest fluid pocket < 2.5 cm in vertical axis.
Crowding of fetal small parts.
Amniotic fluid index (AFI) < 5 cm

AMNIOTIC FLUID VOLUME INCREASED (AFV-I):

Overt polyhydramnios; largest pocket > 8 cm.

FETAL BEHAVIOR

BEHAVIOR STATES: CLASSIFICATION BY FETAL ACTIVITIES

States 1F–4F (Table 2).
Organization normally present by 36 to 38 weeks.
Generally, FBM and FM don't occur simultaneously.
State affects variables used to test fetal well-being, FBM, FM, and FHR.

AS GESTATION ADVANCES

A. Heart rate patterns:
   1. Mean heart rate decreases and variability increases.
   2. Cycles more obvious, accelerations more pronounced, epochal; fewer decelerations with activity.
   3. Circadian rhythm:
      a. Peak between 0800 and 0900.
      b. Trough between 0100 and 0400—may reach levels down to 90 to 100 bpm.
   4. Before 28 weeks, epochal changes rarely dramatic.
   5. About 65% reactive at 28 weeks.
   6. About 95% reactive at 34 weeks.
   7. Nonreactive NST as function of prematurity applies only if there has been no previous reactive NST.

RESPONSES TO VIBROACOUSTIC STIMULATION

A. Fetal heart rate responses:
   1. Tachycardia, accelerations.
   2. Amplitude inversely proportional to baseline rate.
   3. Responses may last 30 minutes or longer.
   4. Abnormal response: bradycardia or decelerations.
B. Ultrasound responses:
   1. Startle.
   2. Head movements, sucking, swallowing.
   3. Alteration of fetal state.
C. Responses influenced by:
   1. Fetal state:
      a. State 1: most easily aroused.
      b. State 2: least responsive.
   2. Duration, intensity, and frequency of stimulus.

TABLE 2.

Fetal Behavioral States

State	Baseline	Variability	Accelerations	Body Movements	Breathing Movements	Eye Movements
1—quiet sleep	Stable	Decreased	Rare	Brief, absent	Infrequent, regular	Absent
2—REM sleep	Stable	Increased	Episodic with fetal movement	Episodic, gross truncal flexion/extension	Frequent, irregular	Present
3—similar to state 1	Stable	Average	Absent	Absent	Infrequent, absent	Present
4—active sleep	Unstable tachycardia	Increased	Large	Continuous gross truncal	Present, irregular	Present

D. Vibroacoustic stimulation
   1. Before 32 weeks:
      a. No increase in accelerations, tachycardia
      b. Startle response—brief
   2. After 32 weeks:
      a. Increases number of body movements
      b. Decreases respiratory movements
      c. Habituation to response

E. Mechanisms—not proven:
   1. Vibration stimulus.
   2. Auditory pain.
   3. Catecholamine release—disputed.

EFFECTS OF DRUGS ON BEHAVIOR

A. Barbiturates, tranquilizers:
   1. Decrease the incidence of REM sleep.
   2. Prolong periods of HVECOG.
   3. Decreased variability, isolated accelerations.

B. Cocaine:
   1. Atypical, disorganized, or bizarre behavior.
   2. Disrupted behavior in response to stimulation.
   3. Sustained hyperpnea, recurrent yawning.
   4. Hypertonic and hyperirritable.
   5. May never attain organized state BEHAVIOR by term.
   6. Difficult to arouse, difficult to console.
   7. Lack of habituation.
   8. Persistently nonreactive NST.
   9. Persistently reactive NST.
   10. Mimics "all or none" or "wired" neonate.

FETAL MOVEMENTS

A. Detection:
   1. Seen by ultrasound as early as 6 to 7 weeks.
   2. Felt by patient 16 to 20 weeks (quickening).
   3. Peak activity 28 to 32 weeks, declines thereafter.
   4. Wide range of normal activity, diurnal variation.
   5. Patient detection varies (about 75%).

B. Movements become more complex, sustained as fetus matures.

C. Physiology:
   1. Occur during LVECOG and HVECOG—frequent.
   2. Abolished by hypoxia, medication, smoking, etc.
   3. Stimulated by contractions.
   4. Coincide with accelerations.
   5. Begins as early as 7.5 weeks gestation.
   6. Later stretching or rolling movements.
   7. Gross movements episodic—about 10% of time.
   8. May be absent for as long as 75 minutes.
   9. Interchangeable with FHR accelerations.

FETAL BREATHING MOVEMENTS

A. Frequently associated with rapid eye movements (REM).

B. Associated with lower rate, increased variability; sometimes regular oscillatory pattern.

C. Occasionally associated with "late decelerations" if UC present.

D. Usually not associated with fetal movements.

E. Episodic—apnea up to 120 minutes in normals; usually less.
F. During LVECOG.
G. Circadian rhythm in healthy fetuses.
H. Increased:
    1. Glucose infusion (fasting).
    2. Hypercapnea.
    3. Smoking.
    4. Prostaglandin synthetase inhibitors.
    5. Normal fetus develops tolerance.
    6. After meals and during sleep.
I. Decreased in:
    1. Hypoxemia (normocapnea).
    2. Asphyxia—gasping.
    3. Alcohol—not reversed by glucose.
J. Recognized in human as early as 10 weeks.
K. Gestation dependent:
    1. Between 24 to 28 weeks' gestation: 10% to 20%.
    2. Beyond 30 weeks' gestation: 30% to 40%.

FETAL TONE

A. Least reliable parameter and last one to go.
B. Hard to detect with decreased AFV.

AMNIOTIC FLUID VOLUME

A. Increased:
    1. Diabetes, Rh isoimmunization, hydrops fetalis (most).
    2. Anomalies—genitourinary (GU), neurologic.
    3. Miscellaneous.
B. Decreased:
    1. Ruptured membranes.
    2. Hypertensive disorders, IUGR—asymmetrical, postdate.
    3. Anomalies—GU.
    4. If prolonged—acquired pulmonary, renal, orthopedic changes.
    5. Miscellaneous.

UTERINE CONTRACTIONS

A. Composed of high-frequency, low-amplitude oscillations with occasional high-amplitude, low frequency contractions (Braxton-Hicks).
B. As gestation advances, low-amplitude oscillations disappear, UC become more frequent, eventually evolve into labor.
C. Hypertonus/tetany:
    1. Spontaneous—about 2%.
    2. Oxytocin infusion—about 5%.
    3. Breast stimulation—about 5%, depending on technique.
D. Behavioral responses to uterine contractions:
    1. Initially stimulate fetal movement.
    2. Later stimulate fetal breathing.

DECREASED VARIABILITY

A. Decelerations absent:
    1. Rest state.
    2. Medication.
    3. Late hypoxia—with unstable baseline.
    4. Neurological deficit—injury or anomaly.
B. Decelerations present—asphyxia.
C. Occasionally "sinusoidal."

INCREASED VARIABILITY

A. With frequent movements; actually reactivity.
B. With low baseline, especially postdates.
C. After variable decelerations.
D. Usually not ominous sign.

SINUSOIDAL PATTERN

A. Consider diagnosis only in the absence of reactivity anywhere.
B. Variant of decreased (short-term) variability.
C. Characteristics:
    1. Amplitude: 5 to 15 bpm.
    2. Frequency: 3 to 6 cpm.
    3. These features same as normal, with variability.
D. Mechanism:
    1. Uncertain.
    2. Possibly related to endorphin release:
        a. May be induced by narcotics.
        b. May be relieved by naloxone HCl (Narcan).
        c. Probably not vagal effect.
E. Clinical classification:
    1. Ominous pattern:
        a. Rh isoimmunization or fetal anemia.
        b. Neurologic injury; usually other features.
        c. Preceding death; other ominous features.
        d. Usually sporadic, sometimes persistent.
    2. Benign pattern:
        a. No identifiable cause.
        b. Narcotic administration.
        c. May be episodic or persistent.
    3. Congenital anomaly:
        a. Seen with cardiac, CNS anomalies.
        b. Usually persistent.