Fetal Heart Rate Monitoring

FOURTH EDITION

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Two Commerce Square
2001 Market Street
Philadelphia, PA 19103 USA
LWW.com

Printed in China

Library of Congress Cataloging-in-Publication Data
Fetal heart rate monitoring / Roger K. Freeman ... [et al.]. — 4th ed.
p. ; cm.
Rev. ed. of: Fetal heart rate monitoring / Roger K. Freeman, Thomas J. Garite, Michael P. Nageotte. 3rd ed. c2003.
Includes bibliographical references and index.
ISBN 978-1-4511-1663-2 (hardback)
I. Freeman, Roger K., 1935- II. Freeman, Roger K., 1935- Fetal heart rate monitoring,
[DNLM: 1. Fetal Monitoring. 2. Heart Rate, Fetal. 3. Fetal Heart. WQ 209]
618.3'2075—dc23

2011048155

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10 9 8 7 6 5 4 3 2 1

The authors dedicate this book in the hope that an in-depth, physiologically oriented understanding of electronic fetal heart rate monitoring (EFM) will result not only in the best possible outcome for babies and mothers but also in an understanding of the limits of this technology. Fetal heart rate (FHR) monitoring is after all a diagnostic, not a therapeutic, device, and no thermometer, EKG, or other diagnostic tool can do more than give us better information and help us make better decisions. It cannot in and of itself improve outcome. The unrealistic expectations that EFM has created and the frequency of abnormal FHR patterns have together led to a labor and delivery environment that is one of high anxiety, for both patients and their caregivers. Clearly, EFM very accurately identifies the fetus that is well oxygenated (category I patterns) and the fetus with hypoxia sufficient to result in death or damage (category III patterns), but the challenge continues to identify those patterns that will deteriorate and without timely intervention may lead to category III patterns. We hope that with a better understanding of EFM, we are not only able to optimize outcome and understand the limits of this technology but also to react more appropriately—minimizing interventions and making the labor and delivery unit the happy, fulfilling environment it should be.

Preface

The first edition of this book was published in 1981 when electronic fetal heart rate monitoring was becoming the preferred modality in most hospitals for intrapartum fetal surveillance. The retrospective and noncontrolled prospective trials comparing electronic fetal monitoring to nonintensive auscultation were encouraging. The second edition was published in 1991. At that time, questions about the validity of the modality and its potential impact on outcomes were emerging. Through randomized controlled trials, it was discovered that intensive auscultation appeared to give equivalent outcomes to electronic fetal heart rate monitoring. Electronic fetal heart rate monitoring was associated with increased operative delivery rates in some studies. It became apparent that the incidence of cerebral palsy had not changed with the introduction of electronic fetal heart rate monitoring. It was clear, however, that when comparing outcomes before electronic fetal heart rate monitoring and intensive auscultation to those outcomes after its advent, the incidence of intrapartum stillbirth in term pregnancies had declined by a factor of 3. This would suggest that the window may have moved, resulting in some previously stillborn infants surviving but with damage and some previously damaged infants surviving intact with a net result of no decrease in cerebral palsy. The third edition published in 2003 included a chapter on fetal pulse oximetry that is no longer available, so it is not included in this fourth edition.

Today, electronic fetal heart rate monitoring offers a more cost-effective means of surveillance than does intensive intermittent auscultation. Presently, most hospitals in the United States offer electronic fetal heart rate monitoring as the primary means of fetal surveillance. It therefore becomes incumbent on providers of obstetric care to have a good understanding of the technique and the interpretation of the data produced. This fourth edition provides the obstetrical clinician a framework within which to interpret and understand fetal heart rate tracings and their implications.

Since the last edition, the NICHD has had another consensus conference aimed at defining research guidelines for fetal monitoring. Such terms as reassuring and nonreassuring have been discarded in favor of the three-category classification that is described in detail in this edition. Furthermore, there are tracings that may suggest fetal central nervous system abnormalities that are not associated with any ongoing hypoxia and therefore would not benefit from intervention by delivery.

In the new classification, patterns are divided into three categories. Category I are patterns indicating normal oxygenation, and Category III patterns are those indicating sufficient hypoxia to result in damage or death. Category II patterns present challenges in management to determine when intervention may be beneficial. Since the last edition, fetal pulse oximetry has become unavailable, and fetal scalp blood sampling is seldom used. Therefore, at this time, most recommend using the presence or absence of FHR variability for intervention decisions in Category II patterns. Clearly, there remains a need for another modality to help interpret category II patterns.

The role of infection resulting in a fetal inflammatory response that is mediated through inflammatory cytokines has been identified as a major causative factor in the later development of cerebral palsy, especially in preterm infants. It is not clear what fetal heart rate patterns may be associated with such conditions or if there are any strategies that we may employ to avoid the damage resulting from this fetal inflammatory response. It is clear, however, that the neonatal encephalopathy resulting from this fetal inflammatory response may be indistinguishable from hypoxic ischemic encephalopathy. The authors have attempted to review this new area of concern.

We are especially proud to introduce Lisa A. Miller, CNM, JD, as our new fourth author who lends an additional perspective to our publication. The new chapter on risk management was a collaborative effort led by her. In addition, she has added perspective to all the other chapters.

We hope the fourth edition will provide obstetrical care providers with assistance in the management of their patients using fetal heart rate monitoring as a means of primary surveillance.

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CHAPTER 1

History of Fetal Monitoring

It is somewhat surprising that something as potentially accessible as the fetal heart was neither heard nor described until the 17th century when Phillipe LeGaust first depicted fetal heart tones in his poetry in an ancient French dialect. LeGaust was a colleague of Marsac, a physician of the province of Limousin, who is credited with first having heard the fetal heart.

Marsac's observation apparently went unnoticed until 1818, when Swiss surgeon Francois Mayor reported the presence of fetal heart sounds when he placed his ear on the maternal abdomen in an attempt to hear the fetus splash about in the liquor amnii. Three years later, French nobleman Lejumeau Kergaradec, apparently unaware of Mayor's report, described both the fetal heart tones and the uterine souffle. He suggested auscultation to be of value in the diagnosis of pregnancy and twins and in determining fetal lie and presentation.

As with many discoveries, the obstetricians of the time were slow to respond to Kergaradec's observations and recommendations. To convince clinicians of the value of Kergaradec's findings, Evory Kennedy of Dublin published an extensive book in 1833, *Observations on obstetric auscultation* (1). The text contains many anecdotal examples of cases in which auscultation was clearly beneficial. In addition, Kennedy described the funic souffle for the first time.

THE FETOSCOPE

Before the development of the fetoscope, much attention was paid to whether mediate (stethoscopic) auscultation using Laennec's instrument, or immediate auscultation, with direct application of the ear to the gravid maternal abdomen, was the more appropriate choice. Rauth and Verardini (2) suggested vaginal stethoscopy as more valuable in the early detection of fetal life. The development of the head stethoscope (fetoscope) is a story of controversy and professional jealousy. It was first reported, in 1917, in *The Journal of the American Medical Association* (Fig. 1.1), by David Hillis (3), an obstetrician then working in Chicago Lying-In Hospital. In 1922, J.B. DeLee (4), who was chief of staff at the same institution and who became a legend in American obstetrics for many contributions, published his report of a similar instrument. Although the order of publications is clear, DeLee claimed that he openly talked of this idea for many years preceding the Hillis publication. The instrument, which subsequently came to be known as the DeLee-Hillis stethoscope, has changed little since its early development.

DIAGNOSIS OF FETAL DISTRESS

Thirty years after Mayor first described heart sounds, Kilian (5) first proposed that changes in the fetal heart rate (FHR) might be used to diagnose fetal distress and to indicate when the clinician should intervene on behalf of the fetus. He formulated what is sometimes called "the stethoscopal indication for forceps delivery" and suggested that heart rates below 100 or above 180 beats per minute (BPM) and those with loss of purity of tone or distinct intermission, or in which only one tone could be heard, were indications for forceps application without delay. In 1893, Von Winckel (6) described the criteria of fetal distress that were to remain essentially unchanged until the arrival of fetal scalp sampling and electronic heart rate monitoring: tachycardia (heart rate >160 BPM), bradycardia (<100 BPM), irregular heart rate, passage of meconium, and gross alteration of fetal movement. Few studies either challenged or supported the validity of these auscultative and clinical criteria for fetal distress. It was not until 1968, when Benson et al. (7) published the results of the Collaborative Project, commissioned by the National Institute of Neurologic Diseases and Blindness, that these criteria came under serious scrutiny. The Benson study reviewed the benefits

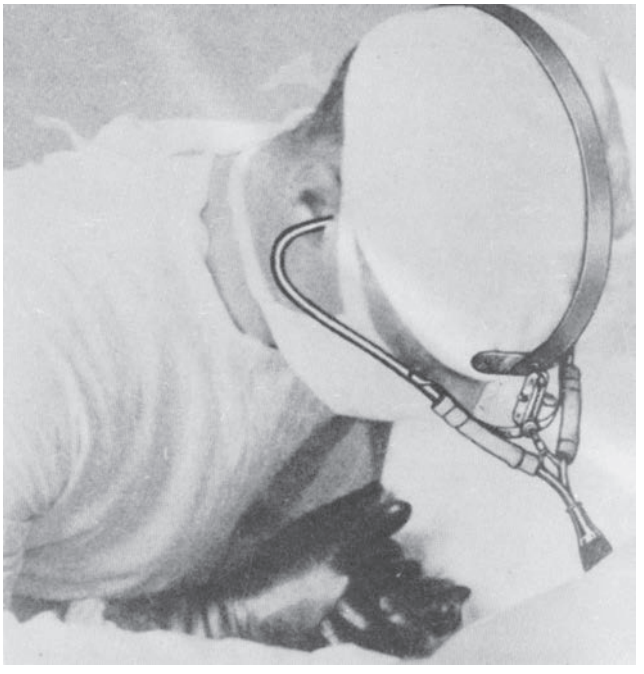

Figure 1.1. Original illustration of the head stethoscope or fetoscope. (From Hillis DS: Attachment for the stethoscope. *JAMA* 68:910, 1917.)

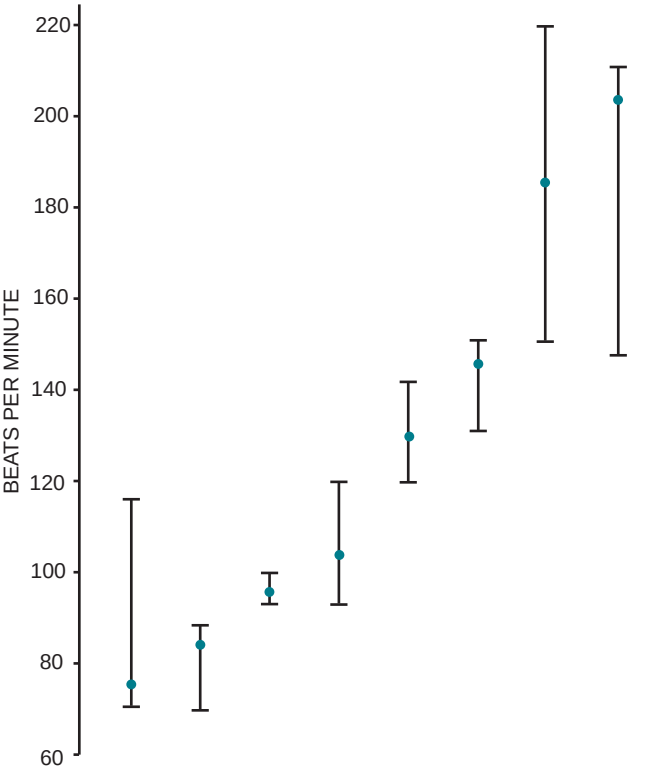

Figure 1.2. Range of error in auscultative counting of fetal heart rate (FHR) by 15 obstetricians asked to count from recorded FHR. Count as reported by obstetrician on vertical scale versus actual count on horizontal scale. (From Hon EH: The electronic evaluation of the fetal heart rate. *Am J Obstet Gynecol* 75:1215, 1958.)

of FHR auscultation in the management of intrapartum fetal distress in 24,863 deliveries. Benson concluded that there was "no reliable indicator of fetal distress in terms of fetal heart rate save in extreme degree." Ten years earlier, Hon (8) asked 15 obstetricians to count several rates from audiotape. He found a wide divergence in counting and pointed out the unreliability of human computation of the FHR (Fig. 1.2).

As new information has emerged, the term fetal distress has been determined as an inappropriate term to describe FHR patterns that may be associated with decreases in fetal oxygenation. Because a large proportion of such patterns do not result in neonates with signs of fetal hypoxia and/or acidosis, the term nonreassuring FHR pattern has been adopted to refer to such tracings. In addition, the original recommendations for intervention based on periodic FHR patterns alone are undergoing an evolution in which the presence of spontaneous or evoked accelerations, FHR variability, fetal pH, and fetal pulse oximetry allow the clinician to sometimes avoid intervention for nonreassuring patterns. These approaches will be discussed in later chapters.

With these serious doubts, and with the age of electronic technology fast making its impact on modern medicine, it was inevitable that obstetric research would turn to more sophisticated methods of fetal evaluation.

THE FETAL ELECTROCARDIOGRAPH

Cremer (9) recorded the FHR electronically for the first time in 1906, by means of abdominal and intravaginal leads. For the first half of the century, the application of fetal electrocardiography was used primarily for the diagnosis of fetal life. It was Southern (10) who, in 1957, suggested that certain fetal electrocardiograph (ECG) changes might correlate with fetal hypoxia. Shortly thereafter, Hon and Hess (11) reviewed all the applications of fetal electrocardiography, including fetal presentation, diagnosis of twins, antenatal diagnosis of congenital heart disease, diagnosis of fetal maturity, and fetal distress. They concluded that the ECG waveform was not of consistent value in any of these situations and, specifically, that in 75 cases of fetal distress "no consistent fetal ECG changes could be detected." Subsequently, Pardi et al. (12) used group averaging techniques to demonstrate ST segment depression with fetal hypoxia (Fig. 1.3). Unfortunately, this technique has not been developed to the point where it has become clinically applicable.

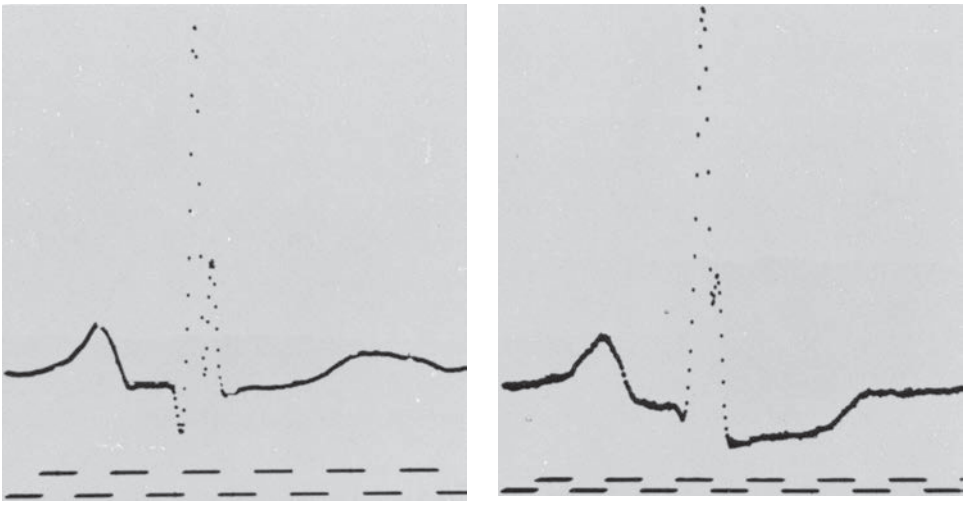

Figure 1.3. **Left:** An average of 25 electrocardiograph (ECG) complexes performed before the onset of a contraction: baseline 160 beats per minute. **Right:** Average of 25 ECG complexes performed immediately after the end of the same contraction, during a late deceleration. Notice the depression of the ST segment. Scalp capillary blood pH 7.34, Apgar score 4/9, umbilical artery pH 7.25. (From Crosignani PG, Pardi G: *Fetal evaluation during pregnancy and labor*. Academic Press, New York, 1971:235.)

The history of the development of electronic FHR monitoring, or cardiotachometry, is a complex merger of technologic development with empirical observations of those heart rate patterns associated with various causes of fetal distress.

The earliest preliminary report of FHR monitoring came in 1958 from Edward Hon (8), then at the Yale University School of Medicine. He reported on the continuous instantaneous recording of FHR via fetal ECG monitor on the maternal abdomen. He began to elucidate causes of fetal bradycardia and, more specifically, defined when bradycardia was indicative of fetal distress. In the years that followed, Hon, Caldeyro-Barcia in Uruguay, Hammacher in Germany, and their many coworkers reported their observations on the various heart rate patterns associated with fetal distress. Bradycardia and tachycardia were well-known signs of fetal compromise. In 1959, Hon (13) defined the type of variable deceleration associated with umbilical cord compression and proposed a mechanism for the hypoxic uteroplacental causes of delayed decelerations. In 1963, Caldeyro-Barcia et al. (14) reported their observations on the "prognostic significance" of similar heart rate decelerations, which they called type III and II. In addition, long- and short-term FHR variability was defined for the first time. Hammacher (15) subsequently described this parameter's significance in terms of loss of heart rate variability in association with fetal distress.

With many investigators throughout the world making similar observations, FHR terminology became extremely confusing. Hon, Caldeyro-Barcia, and many of their colleagues met at an international conference on monitoring of the fetal heart in December 1971 in New Jersey and in March 1972 in Amsterdam to discuss nomenclature and develop standards for FHR monitoring. They developed and agreed upon a common nomenclature. Efforts were made to agree upon universal scales and paper speed for fetal monitors, but these remained variable. Much of the subsequent history has been one of technologic development for the clinical application of electronic FHR monitoring. The first practical commercially available fetal monitor to be of clinical use was produced by Hammacher and Hewlett-Packard in 1968 using external tocography and phonocardiography (Fig. 1.4). Before this time, monitors were bulky and generally limited to research, although attempts were made to market some equipment for general use (Fig. 1.5). Technologic advances since that first generation of fetal monitors have allowed further and more accurate definition of FHR patterns and have provided the clinician with a practical tool. Direct ECG monitoring was realized with Hon's introduction of an electrode that could be applied directly to the fetal scalp (16). This was originally a modification of a surgical skin clip (Fig. 1.6). Subsequently, Hon developed a more convenient disposable spiral electrode (Fig. 1.7), which is widely used in the United States. Doppler ultrasound and external ECG and logic systems that provide adequate approximations of the real beat-to-beat heart rate have been more recent developments.

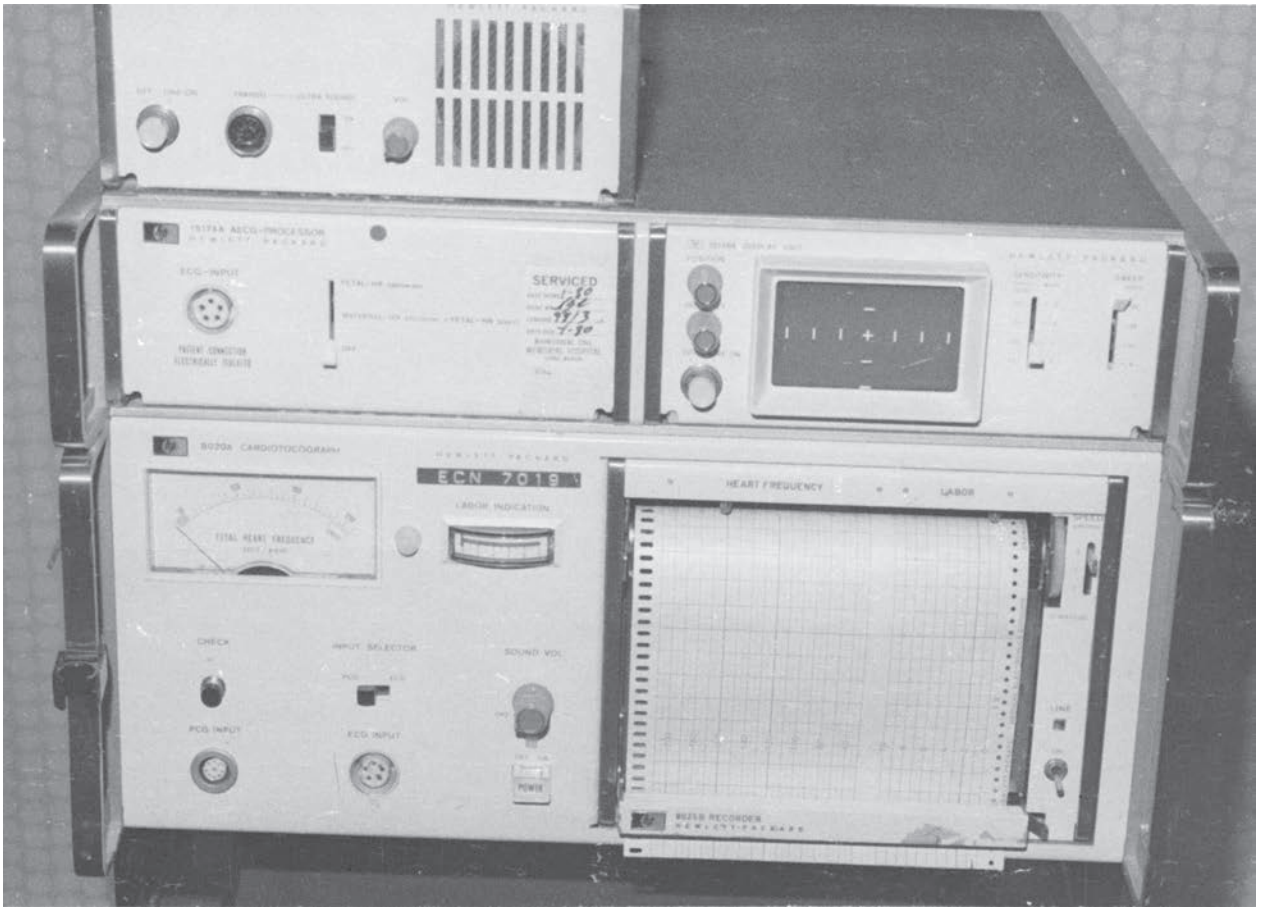

Figure 1.4. First generation of practical commercially available fetal monitors. The original model used external monitoring with phonocardiography only. The upper two modules were subsequently added, allowing the addition of external ultrasound and internal fetal electrocardiographic monitoring (Hewlett-Packard Model 8020A).

ERA OF QUESTIONING

Benson's report on the data collected by the Collaborative Project set the stage for rejection of auscultation and the boom in electronic cardiotachometry. By 1978, it was estimated that fetal monitoring was in routine use in over half of labors (18). Enthusiasm for its use, however, came without clear documentation of its efficacy and safety. There were many retrospective analyses of fetal monitoring and nearly all agreed to a beneficial effect, including reduction of intrapartum stillbirth rate and perinatal mortality as well as improved Apgar scores (19). Randomized controlled trials have now been reported (20–23). While mixed, they do not uniformly show electronic monitoring as beneficial and, indeed, suggest that such monitoring may substantially increase the rate of cesarean sections. Details of all these studies and an analysis of the risks and benefits of fetal monitoring are reviewed in Chapter 3. The era of consumer demand in obstetrics in particular, coupled with a period of heightened government intervention into cost, risks, and benefits of medical care, has provided further impetus for questioning electronic fetal monitoring.

Electronic fetal monitoring has become common practice for patients during labor, and antepartum electronic FHR monitoring is one of the best means currently available to assess the fetus at high risk for antenatal uteroplacental insufficiency. Further applications will depend on continuing evaluation and adjustment of current methodologies as well as the development of new technology. It is clear, however, that assessment of the human fetus for hypoxia is one end result of the development of electronic FHR monitoring.

Figure 1.5. This bulky machine was the first attempt at making a commercially available fetal monitor. (Courtesy of Epsco, Inc.)

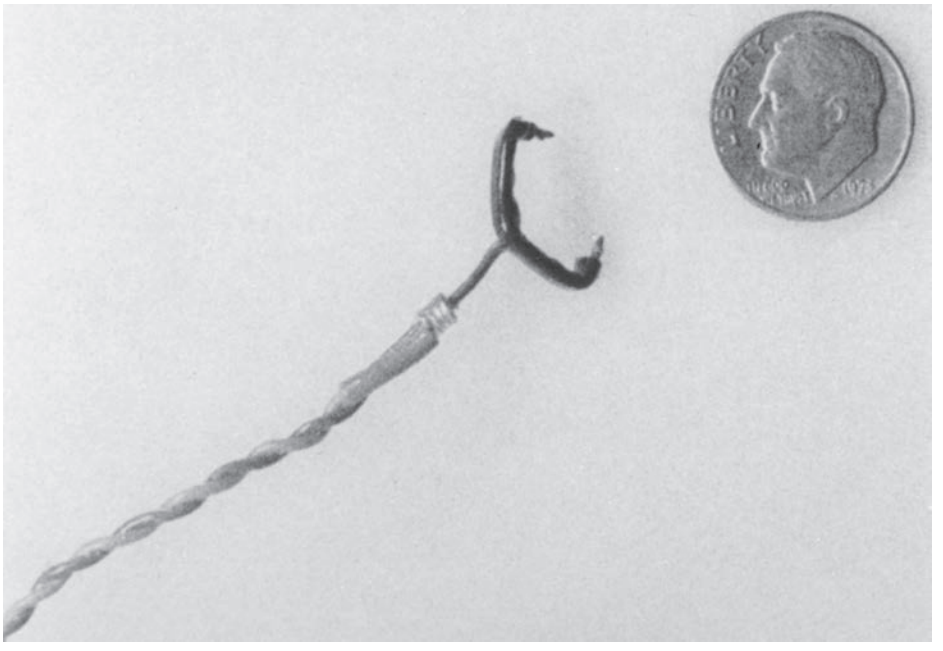

Figure 1.6. Vaginal fetal scalp electrode as described by Dr. Edward Hon (16). This is a modification of a Michelle surgical skin clip. A specially made long forceps and a vaginal speculum are necessary for application.

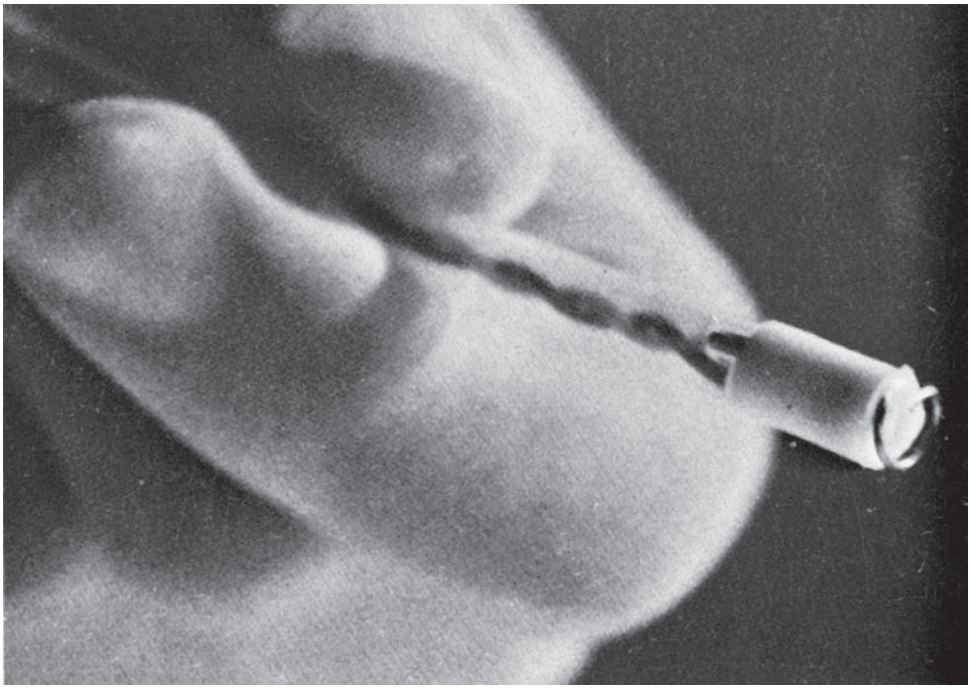

Figure 1.7. Spiral silver-silver chloride fetal scalp electrode described by Hon (17) in 1972. This is packaged within a plastic sheath, which allows direct application without the aid of a speculum.

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CHAPTER 2

Physiologic Basis of Fetal Monitoring

Clinical fetal heart rate (FHR) monitoring is an ongoing observation of human fetal physiology. The question being asked by the clinician is: What is the adequacy of fetal oxygenation? Because the FHR pattern appears to assume certain characteristics under the influence of various hypoxic and nonhypoxic influences, it becomes important for the clinician to have a basic understanding of the physiology of fetal respiratory exchange and the physiologic control of the FHR. In this chapter, we outline the principal factors involved in fetal oxygenation and carbon dioxide transfer as well as the basis for our current understanding of FHR responses to changes in fetal respiratory status.

THE ANATOMY OF MATERNAL-FETAL EXCHANGE

The placenta is an organ that functions as the fetus' extracorporeal life-support system. The placenta serves as the fetal lung (respiration), kidney (excretion), gastrointestinal tract (nutrition), and skin (heat exchange) and as a barrier against certain substances dangerous to the fetus. In addition, it is an endocrine organ that produces steroid (estrogen, progesterone) and protein (human chorionic gonadotropin, human placental lactogen) hormones. Very early in gestation, the blastocyst implants in the decidualized endometrium and the trophoblast cells invade the maternal circulation, creating a lake of maternal blood that bathes the trophoblast and developing embryo. As the gestation grows, a number of spiral arteries that supply blood to the endometrium are penetrated and provide the basic architecture as the placenta develops, with villi forming cotyledons arranged around these spiral arteries. The fetal chorionic villi develop many convolutions and float in the maternal blood supplied from the previously invaded spiral arteries. This maternal blood occupies an area referred to as the intervillous space, and it is between this space and the fetal capillary (contained within the chorionic villus) that maternal-fetal and fetal-maternal exchange occurs. The human placenta is thus referred to as a hemochorial type because the mother's blood comes into direct contact with the fetal chorionic villus (1). Oxygen, carbon dioxide, nutrients, waste products, water, and heat are exchanged at this level and must cross two layers of fetal trophoblast, the fetal connective tissue within the villus, and the fetal capillary wall (Fig. 2.1).

The uterine blood flow is supplied principally from the uterine arteries, but anastomoses occur between these vessels, other branches of the hypogastric arteries, and ovarian arteries. Significantly, the spiral arteries must traverse the full thickness of the myometrium to reach the intervillous space. Anything that affects maternal cardiac output will, of course, affect the flow through the spiral arteries. Additionally, as the

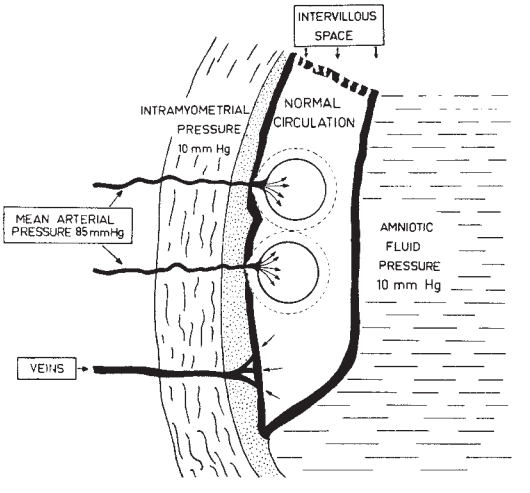

Figure 2.1. The uteroplacental circulation between uterine contractions is shown. Note that the intramyometrial pressure is less than the arterial pressure, allowing the arteries to remain open and provide a supply of blood to the intervillous space. (From Poseiro JJ, Mendez-Bauer C, Pose SV, et al.: Effect of uterine contractions on maternal blood flow through the placenta. In: *Perinatal factors affecting human development*. Pan American World Health Organization, Scientific Publication no. 185, Washington, DC, 1969:161–171, with permission.)

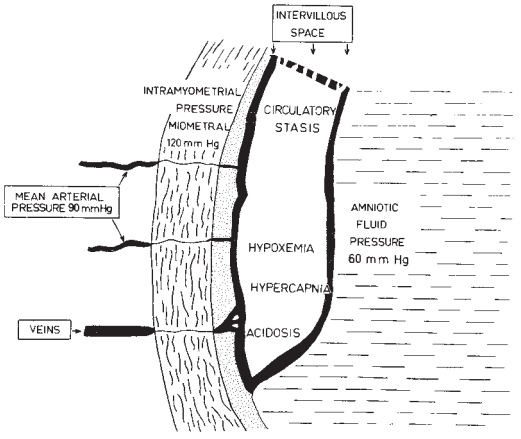

Figure 2.2. The uteroplacental circulation during a uterine contraction is shown. Note that the intramyometrial pressure exceeds the arterial pressure, causing circulatory stasis in the intervillous space. (From Poseiro JJ, Mendez-Bauer C, Pose SV, et al.: Effect of uterine contractions on maternal blood flow through the placenta. In: *Perinatal factors affecting human development*. Pan American World Health Organization, Scientific Publication no. 185, Washington, DC, 1969:161–171, with permission.)

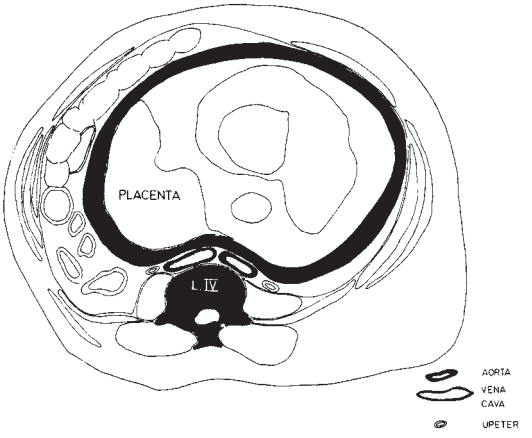

Figure 2.3. A cross section of a pregnant uterus lying on the maternal vertebral column with the interposed great vessels, which are subject to occlusion when the mother is supine. (From Poseiro JJ, Mendez-Bauer C, Pose SV, et al.: Effect of uterine contractions on maternal blood flow through the placenta. In: *Perinatal factors affecting human development*. Pan American World Health Organization, Scientific Publication no. 185, Washington, DC, 1969:161–171, with permission.)

uterus contracts, the intramyometrial pressure may exceed the intraarterial pressure, causing occlusion of these vessels and resulting in cessation of blood flow to the intervillous space (Fig. 2.2) (2).

Approximately 85% of the total uterine blood flow goes to supply the placental (intervillous) circulation and 15% supplies the extraplacental uterine musculature (3). The intervillous space blood supplies the vascular support to the placenta itself, and if the blood flow to any area is sufficiently compromised, an infarction of the placenta may occur (4,5).

For clinical purposes, the intervillous space circulation is probably maximal when the mother is at rest, in the lateral position. Although estrogen administration to the pregnant ewe will increase uterine blood flow, it is not certain that this results in increased intervillous blood flow (5,6). Therefore, although we know of nothing that will increase effective uterine blood flow above that found in the resting lateral position, there are many things that will decrease uterine blood flow.

FACTORS THAT DECREASE UTERINE BLOOD FLOW

Position

Changes in the mother's position may decrease blood flow to the uterus by at least two mechanisms. The supine position is characterized by an exaggeration of the lumbar lordotic curve, resulting in uterine compression of the vena cava, the aortoiliac vessels, or both. With vena caval occlusion, the return of blood to the heart is decreased, and this may result in a fall in maternal cardiac output, maternal hypotension, and decreased uterine blood flow (7,8). Compression of the aorta against the spine or iliac vessels as they cross the pelvic brim may result in decreases in uterine blood flow without maternal hypotension (Fig. 2.3). Abitbol et al. (9) showed that patients in the supine position had an increased incidence of late decelerations during labor and this correlated with a fall in femoral arterial pressure, a decrease in the amplitude of the big toe capillary pulse, and a drop in the fetal scalp blood pH, all of which were reversible when patients were returned to the lateral position. Simpson and James (10), using measurements of fetal oxygen saturation via fetal pulse oximetry, in a randomized trial, showed a 29% improvement in oxygen saturation with either lateral position compared to supine.

Exercise

Maternal exercise may serve to divert blood away from the uterus to supply somatic muscle groups, resulting in decreased uterine blood flow. Clapp (11) has shown that baseline FHR increases consistently following exercise in pregnant women. The tachycardia following exercise is not believed to be due to hyperthermia or increased fetal activity but rather is a sympathetic response to a period of reduced fetal oxygenation. Artal et al. (12) have described three cases of fetal bradycardia occurring during postexercise fetal tachycardia, suggesting that in those cases there was more profound hypoxia. These studies in humans tend to support the caution originally suggested in Emmanouilides et al.'s research into exercising pregnant sheep (13). Although there is evidence that exercise is not detrimental to the fetus where there is normal uteroplacental function (14), it is possible that some fetuses could be adversely affected by excessive maternal exercise.

Uterine Contractions

The spiral arteries that traverse the myometrium are subject to collapse as the uterus contracts and the intramyometrial pressure exceeds the spiral arterial pressure. During normal pregnancy, the uterus has certain inherent contractility (Braxton-Hicks contractions), and as labor begins, the contractions increase in frequency and intensity. If the uteroplacental oxygen exchange is normal, the Braxton-Hicks contractions and labor contractions do not appear to significantly compromise the total intervillous space blood flow. However, a normal fetoplacental unit may have its uteroplacental reserve exceeded if uterine activity is excessive, as with spontaneous or oxytocin-induced uterine hypertonus and/or tetanic contractions (15,16). Patients with abruptio placentae may have hypertonus and tachysystole, resulting in decreased intervillous space perfusion producing fetal hypoxia. Orgasmic coitus has been reported to be associated with increased uterine activity and prolonged FHR deceleration in three patients studied with simultaneous FHR and uterine contraction monitoring (17).

Surface Area

Anything that will decrease the effective surface area of the placenta will clearly increase the potential for fetal hypoxia. Abruptio placentae is a classic example of reduced placental surface area available for exchange. Patients with multiple placental infarcts, as may be seen in hypertensive disorders and prolonged pregnancy, are also subject to having the fetus suffer from uteroplacental insufficiency (UPI). There is recent evidence that hypercoagulable states in the fetus may result in thromboses of the fetal vessels in the placenta that may lead to growth restriction and decreased available oxygen to the fetus (18).

Anesthesia

The administration of conduction anesthetics carries the potential for reduced intervillous space blood flow secondary to maternal hypotension, resulting from the sympathetic blockade that may occur to a greater or lesser degree in all such patients. The pharmacologic correction of such hypotension with α-adrenergic agents may not restore uterine blood flow because the α-adrenergic agents will increase uterine circulatory resistance along with the other somatic components of total peripheral resistance that are responsible for the increase in blood pressure. For this reason, it is recommended that an agent such as ephedrine (a mixed α- and β-adrenergic stimulator) be used to restore maternal blood pressure after hypotension induced by the sympathectomy of conduction anesthesia (19). Usually, however, the uterine blood flow is easily restored in such situations by correcting positional factors and expanding maternal blood volume with increased intravenous fluids, thus obviating the need for any pressor agents.

Using isotope techniques, reports of increased uterine blood flow following epidural anesthesia have given rise to some questions regarding the classic understanding of sympathectomy caused by conduction anesthesia, resulting in diversion of blood from the uterus (20). Data using Doppler flow techniques have shown that diastolic flow in the arcuate arteries may actually increase in prehydrated patients following epidural anesthesia (21). The proposed mechanism involves a decrease in sympathetic tone in the uterine circulation, resulting in decreased resistance. It is not clear, however, whether this finding actually represents increased placental flow or merely opening of the nonplacental portion of the uterine circulation. The reason one must question data suggesting that epidural anesthesia increases uteroplacental blood flow is related to the frequent finding of late decelerations following epidural activation. Further research in this area is needed to clarify the exact relation of conduction anesthesia to uteroplacental blood flow.

Hypertension

Maternal hypertensive syndromes may result in decreased intervillous space blood flow as a result of either acute vasospastic or chronic atheromatous changes in the uterine arterial blood supply. If one lowers blood pressure in the hypertensive patient either intentionally with antihypertensive agents or unintentionally as a result of administering a conduction anesthetic, one runs the risk of diverting blood away from the intervillous space if the caliber of the uterine arterial circulation remains diminished as other vascular beds are dilated.

Diffusion Distance

The thickness of the placental membrane between the intervillous space and the fetal capillary may also decrease the transfer of oxygen. An example of a clinical entity demonstrating this phenomenon can probably be found in fetal erythroblastosis with placental edema. Perhaps this is also a factor in certain conditions of fetal dysmaturity where there is an increase in fibrin deposition between the intervillous space and the fetal capillary. Villous hemorrhage and edema in diabetics may also play a role in increasing the thickness of the placental membrane.

THE FETAL CIRCULATION

The anatomy and physiology of the fetal circulation are very complex. For the purposes of this chapter, we focus on the umbilical circulation and factors that affect placental exchange (Fig. 2.4).

The umbilical vessels are contained within the umbilical cord and are protected with an abundance of a substance

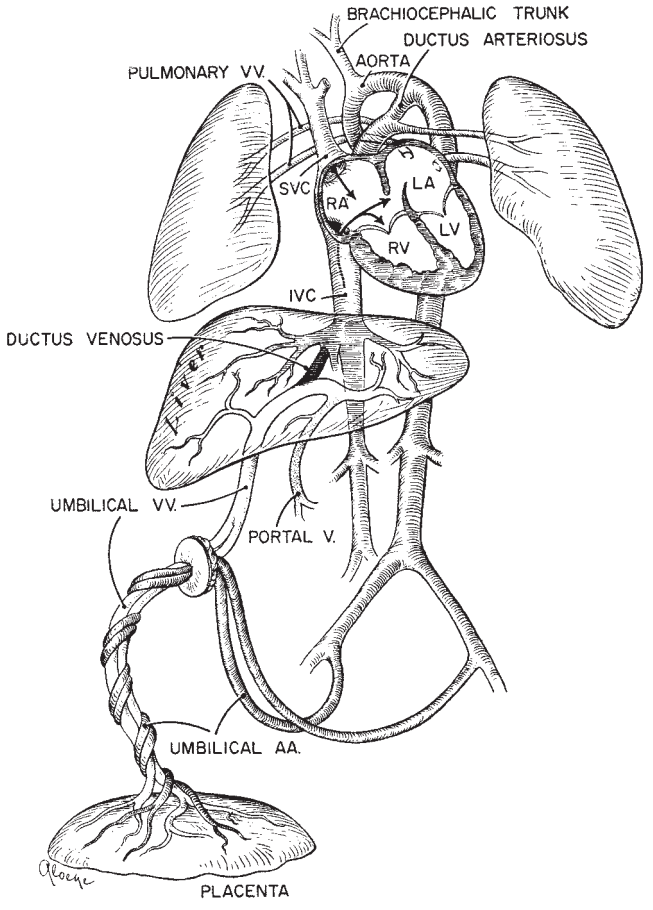

Figure 2.4. Schematic representation of the fetal circulation in the lamb. Note that the well-oxygenated blood returning from the placenta via the inferior vena cava (*IVC*) crosses into the left atrium (*LA*), while the superior vena cava (*SVC*) blood tends to *run into the right* ventricle (*RV*). (From Assali NS: Fetal and neonatal circulation. In: *Biology of gestation. II*. Academic Press, New York, 1968:254, with permission.)

called Wharton's jelly. Normally, there are two umbilical arteries that arise from the terminal ends of the fetal hypogastric arteries and a single umbilical vein that returns blood to the fetus from the placenta, channeling it partly through the liver and partly to the inferior vena cava via the ductus venosus. This well-oxygenated blood enters the right atrium and follows a course directing it mainly to the cephalic circulation, while blood returning from the upper body via the superior vena cava is channeled principally through the ductus arteriosus to the lower body and the placenta. Approximately 30% of the fetal cardiac output goes to the placenta, which comprises a low-resistance vascular bed. The oxygen content of the umbilical venous blood closely approximates that of the uterine venous drainage, which suggests that the pattern of umbilical and uterine flows functionally follows a concurrent relationship, although the flow relationships are clearly much more complex and *authorities* in the field are not in total agreement concerning this fact (22).

One must ask how the fetus can exist at a maximum pO_2 below that of the uterine vein (about 35 mm Hg), whereas the adult would be unable to survive under similar circumstances. The fetus has a number of unique characteristics that allow it thrive with such a low pO_2.

First, the fetal hemoglobin concentration is higher than that in the adult, allowing for a much greater oxygen carrying capacity. Second, the fetal cardiac output far exceeds that of the adult on a volume per unit body weight basis. Third, the fetal hemoglobin dissociation curve favors a higher saturation at a given pO_2 (Fig. 2.5). So, although the fetal pO_2 is lower, the fetus is able to compensate by having an increased oxygen content, due to the high hemoglobin concentration and the characteristic of the fetal hemoglobin dissociation curve. The more rapid circulation then increases the amount of oxygen that can be delivered to the fetal tissue per unit time (23).

Oxygen crosses the placenta by simple diffusion. If the relative flows on the two sides of the placenta are held constant, the differential pO_2 between the intervillous space and the fetal capillary determines the rate of maternal-fetal oxygen transfer. Thus, administration of high concentrations of oxygen to the mother may increase her pO_2 several hundred millimeters of mercury and the maternal-fetal oxygen gradient may be markedly increased by this maneuver (24). Moreover, if the fetal pO_2 is increased by only a few millimeters of mercury, the fetal blood oxygen content may increase

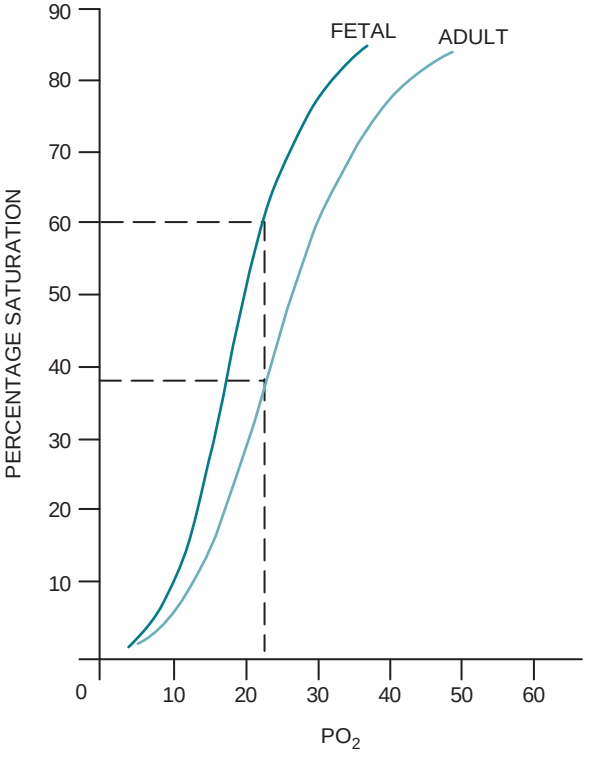

Figure 2.5. Adult and fetal hemoglobin dissociation curves.

significantly because, in either the physiologic or hypoxic range, the fetal hemoglobin saturation curve is quite steep (Fig. 2.5) (25).

Clinically, however, even though maternal hyperoxia may be of some help, most causes of fetal hypoxia are related to restriction of umbilical or intervillous space blood flow. Under conditions of diminished flow on either side of the placenta, changing the pO_2 gradient will not be of as much help as restoring the blood flow. It is still reasonable to administer oxygen to the mother (26) in such situations, but one must remember that restoration of flow is usually more important.

Fetal Circulatory Response to Hypoxia

Fetal circulatory responses to hypoxia involve redistribution of blood flow.

These include

- A redistribution of cardiac output resulting in preservation of blood flow to certain vital organs, including brain, myocardium, and adrenal glands, at the expense of flow to certain less vital organs including the parathyroid glands, lungs, liver, kidneys, intestines, bone marrow, and somatic muscles
- A loss of cerebral vascular autoregulation resulting in a pressure-passive cerebral circulation
- An eventual decrease in cardiac output resulting in hypotension and ultimately a decrease in cerebral blood flow (27–29). Fetal cerebral vascular resistance can decrease by at least 50% and maintain cerebral blood flow allowing for only a minimal decrease in oxygen delivery. With persistent hypoxemia leading to arterial hypotension, cerebral vascular resistance cannot maintain flow by vasodilation, leading to a marked reduction in cerebral blood flow. This can then lead to neuronal necrosis in the fetal brain.

There are also noncirculatory responses to hypoxia that are important in preserving neuronal integrity under hypoxic conditions. These include

- A slower depletion of high-energy compounds during hypoxia-ischemia in the fetus compared with the term infant or adult
- The use of alternate energy substrate, the neonatal brain having the capacity to use lactate and ketone bodies for energy production
- The relative resistance of the fetal and neonatal myocardium to hypoxia-ischemia
- The potential protective role of fetal hemoglobin

Because of these circulatory and noncirculatory responses to hypoxia, the fetus has considerable protection from neuronal damage, and even with severe hypoxic insults, most fetuses that survive have little or no central nervous system (CNS) damage.

CONTROL OF THE FETAL HEART RATE

The FHR, under physiologic conditions, represents the final product of intrinsic and extrinsic rate-determining or rate-modifying factors. Technically, the FHR represents the reciprocal of the interval between two successive beats. Most data on FHR use an electrical marker (specifically the peak of the fetal electrocardiogram [ECG] R wave) to signify the time of the beat. Unless otherwise stated, we will refer to the rate calculated from intervals between fetal ECG R waves. FHR changes constitute the basis for electronic fetal monitoring, and for this reason, one must look carefully at the factors that determine or modify the rate.

Schifferli and Caldeyro-Barcia (30) discovered that the baseline heart rate decreases with gestational age. At 15 weeks' gestation, the average baseline rate is approximately 160 beats per minute (BPM) (Fig. 2.6). Although premature fetuses may have a slightly increased rate over that found at term, within the limits of fetal viability (from 28 weeks to term), the average baseline FHR difference is only approximately 10 BPM. One must, therefore, be careful not to attribute a baseline tachycardia to prematurity when it may well be a sign of fetal compromise. Any baseline FHR above 160 BPM must be explained on some basis other than fetal prematurity.

Schifferli and Caldeyro-Barcia (30) further noted that if one administers atropine to a fetus, the resulting increase in heart rate is of greater magnitude as one approaches term, and that the postatropine heart rate is usually in the range of 160 BPM. Because atropine is a parasympathetic blocking agent, it would appear that the gradual decrease in FHR that

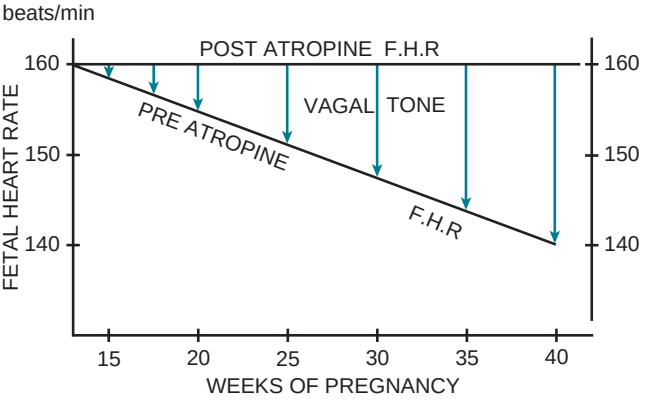

Figure 2.6. The preatropine fetal heart rate (FHR) shows that the average FHR decreases as gestational age increases. The postatropine FHR shows that the FHR after atropine administration rises to approximately 160 beats per minute, regardless of gestational age, indicating increasing vagal tone as gestational age increases. (From Schifferli P, Caldeyro-Barcia R: Effects of atropine and beta adrenergic drugs on the heart rate of the human fetus. In: Boreus L, ed. *Fetal pharmacology*. Raven Press, New York, 1973:264, with permission.)

occurs with increasing gestational age can be explained as an increase in parasympathetic tone (Fig. 2.6).

Renou et al. (31) have shown that upon administering atropine directly to a human fetus during labor, three phenomena are observed. First, there is a modest increase in the baseline FHR, presumably due to blocking of the tonic parasympathetic effect described by Schifferli and Caldeyro-Barcia (30). Second, Renou et al. noted a loss of the variability of the FHR, suggesting that a continuous balance between the parasympathetic slowing effect and the sympathetic accelerating effect may have been disturbed. Third, they noted that a majority of patients demonstrated the appearance of FHR accelerations during uterine contraction. This suggests that, before release of parasympathetic tone by atropine, the accelerating forces were present but suppressed. Renou et al. then added a β-adrenergic blocking agent (propranolol) to these atropinized fetuses and noted abolition of the accelerations and a decrease in the baseline FHR (Fig. 2.7). It

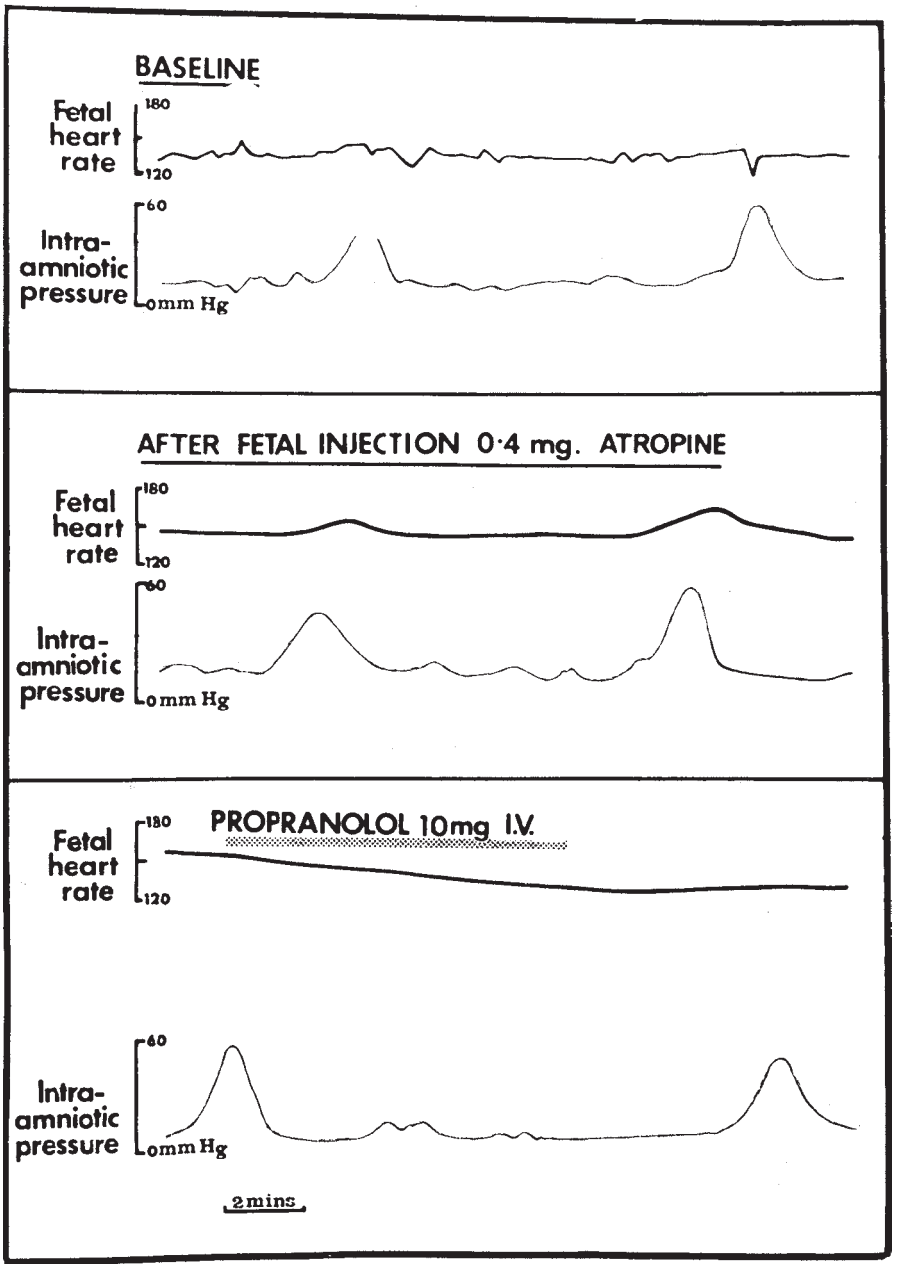

Figure 2.7. The upper panel shows a normal fetal heart rate (FHR)-uterine contraction tracing. The middle panel shows that after atropine administration to a human fetus, the FHR rises, the FHR variability decreases, and accelerations appear with contractions. The lower panel shows that after maternal propranolol administration, the FHR decreased and the FHR accelerations disappeared. (From Renou P, Newman W, Wood C: *Am J Obstet Gynecol* 105:953, 1969, with permission.)

would thus appear that the accelerating forces unmasked by atropine blockade were of β-sympathomimetic origin. With this information, it is then possible to think of the baseline FHR as a product of the modulated influences of the parasympathetic and sympathetic nervous systems. Furthermore, the baseline FHR variability probably represents an instantaneous product of these two forces that are constantly working in a push-pull relationship; the presence of good FHR variability probably requires the integrity of these two modulating forces.

Under normal circumstances at term, the rate determined by the atrial pacemaker and modulated by parasympathetic and sympathetic factors usually ranges between 120 and 160 BPM. In a fetus with heart block, the rate is usually in the range of about 60 BPM, which represents the intrinsic ventricular or nodal rate.

Parasympathetic impulses originate in the brain stem and are carried over the vagus nerve to the heart. Sympathetic impulses also originate in the brain stem and are carried via the cervical sympathetic fibers to the fetal heart. Sympathetic influences on the fetal heart may also come from humoral stimulation of the cardiac β-receptors via release of epinephrine from the adrenal medulla. In clinical practice, conditions such as fetal anencephaly, marked hydrocephaly, and anoxic brain damage may all be associated with an absence of FHR variability and a very "blunted" FHR response to stress. In the adult who experiences cerebral death, the heart rate, when recorded instantaneously, has no variability. These clinical examples support the notion that heart rate variability is largely under CNS influence. These examples further suggest that higher centers in the brain also play a role. Although these mechanisms of CNS control of FHR cannot be completely understood with current available data, clinically it is important to recognize that the FHR pattern does depend on certain CNS controls and that factors influencing the CNS such as drugs, brain damage, or cerebral hypoxia may also affect the FHR pattern. The FHR patterns associated with CNS dysfunction are discussed in more detail in Chapter 13.

FETAL STATE

The normal fetus has certain FHR changes that are related to the fetal state. It is well-established that, after about 32 weeks' gestation, virtually all normal fetuses demonstrate episodes of FHR acceleration associated with fetal movement. This has been called fetal reactivity and has been found to have a high association with fetal well-being. Before 23 weeks, fetal reactivity is rare, and it would seem that the appearance of FHR reactivity is related to the CNS maturation that is occurring at the beginning of the third trimester (32,33). Normal fetuses exhibit episodes of reactivity that last 20 to 40 minutes or longer (34). When the FHR accelerates, there is virtually always fetal movement, but the converse is not true (35). There are intermittent periods of low reactivity that correlate with fetal electroencephalogram (EEG) signs of deep sleep. During the periods of reactivity, fetal EEG measurements are suggestive of rapid eye movement sleep. Fetal state has a circadian rhythm with maximal reactivity occurring late at night (36). There is also a relation between fetal baseline heart rate and maternal heart rate that varies in a diurnal fashion. Brown and Patrick (37) have shown that normal fetuses rarely go more than 80 minutes without reactivity; when they do, they are very likely severely compromised. Drugs such as phenobarbital (38) and propranolol (39) will decrease reactivity, especially in higher doses. Also, fetuses with primary CNS abnormalities will often have decreased reactivity. Loss of accelerations appears to be a change that occurs later than the appearance of late decelerations in fetuses undergoing progressive hypoxia (40).

The Effect of Uterine Contractions on the Fetal Heart Rate

FHR monitoring consists of a series of observed changes in instantaneous heart rate with and without uterine contractions. Uterine contractions subject the fetus to an intermittent hyperbaric state. They also cause intermittent decreases in intervillous space blood flow, may influence cerebral blood flow under certain circumstances, and, depending on the location of the umbilical cord, may cause intermittent umbilical cord occlusion. These situations may all influence the FHR by giving rise to contraction-related or "periodic" FHR changes. The physiologic bases for these periodic changes are discussed below.

Early Deceleration

Pressure on the fetal head causes slowing of the heart rate. It is believed that this is due to local changes in cerebral blood flow (41), resulting in stimulation of the vagal centers. While the fetal head undoubtedly undergoes compression of a greater or lesser degree in all vaginal deliveries, the typical gradual onset, gradual offset FHR deceleration described as characteristic of fetal head compression is rather uncommon. Certainly during the second stage of labor, when pronounced variable FHR decelerations are commonly seen, it is not possible to say for sure that these decelerations may not be partially or entirely due to compression of the fetal head as it passes through the birth canal. Hon (*personal communication*, 1974, Los Angeles) studied neonates, using different-sized doughnut pessaries, and found that placing the circular pessary over the fetal vertex, when the inside diameter of the pessary was 4 to 6 cm in diameter, usually resulted in point pressure from the edge of the pessary over the anterior fontanelle and that this was associated with a FHR deceleration. This seemed to fit clinically with the fact that the FHR deceleration pattern that reflected the uterine pressure curve as a mirror image is usually found when patients are between 4 and 6 cm of cervical dilatation. This symmetric deceleration

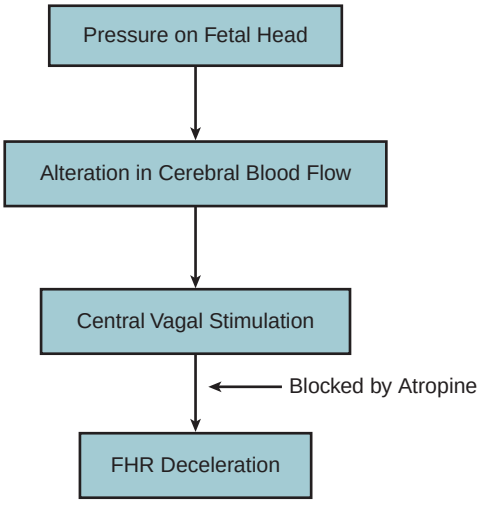

Figure 2.8. Mechanism of early deceleration (head compression).

pattern resulting from fetal head compression is called *early deceleration* and will be described in more detail in Chapter 6. Further studies have shown that early deceleration may be abolished or markedly altered by the administration of atropine, thus confirming the theory that this is a vagal reflex (42). There is good evidence that this deceleration pattern is not associated with hypoxia or acidosis (Fig. 2.8) (43).

Variable Deceleration

Variable decelerations are usually indicative of obstruction to umbilical vascular flow. The exception to this is that often in the second stage of labor, fetal head compression may cause decelerations that mimic those associated with umbilical cord compression. While the terminology for interruption of umbilical blood flow is often referred to as "umbilical cord compression," it is important to understand that there are other mechanisms that can cause obstruction to umbilical vascular flow. Teleologically, the normal protective mechanism for the fetus born without attendance is that there are three mechanisms for which result in cessation of cord flow at the time of birth. These include cord stretch at the time the baby is on the ground and the placenta still in the womb; cold, as the ambient temperature is almost always colder than the intrauterine/body temperature; and the rise in pO_2, as the baby begins to breathe. The corollaries for the fetus in labor are that at least two of these mechanisms can be responsible for interruption in umbilical blood flow and variable decelerations besides external compression of the cord. Certainly cord stretch can occur with a nuchal cord or a short cord as the fetus descends into the birth canal in the latter stages of labor, and this can be one, if not the dominant, mechanism for the appearance of variable decelerations as descent begins in the second stage of labor and when variable decelerations are commonly seen. Similarly, it has been observed that with amnioinfusion, that is, if cold or room temperature saline is infused rapidly, cord compression patterns or bradycardia can be seen. And as more traditionally described, the umbilical cord is easily compressed by entrapment between any part of the fetus and uterine wall or pelvis, and this most commonly occurs with uterine contractions or as the fetus moves.

Our understanding of the mechanism of FHR changes in association with umbilical cord occlusion began with the work of Barcroft (44). He studied exteriorized fetal goats and showed that there was an almost instantaneous rapid and profound decrease in the FHR when the umbilical cords of these fetal goats were occluded. Because the response was so rapid, he reasoned that it may be due to a neurologic reflex. He then repeated the experiment after interrupting the vagus nerves of these fetal goats and found that there was a delay in the onset of the FHR deceleration. This then suggested that indeed the initial rapid deceleration associated with cord occlusion in the animal with intact vagi did appear to be caused by a vagal reflex, but the fact that a delayed deceleration was observed even with interruption of the vagal nerves suggested a second mechanism for the delayed portion of the deceleration (Fig. 2.9).

If one examines the hemodynamic effects of umbilical cord occlusion, some clues concerning the mechanism for

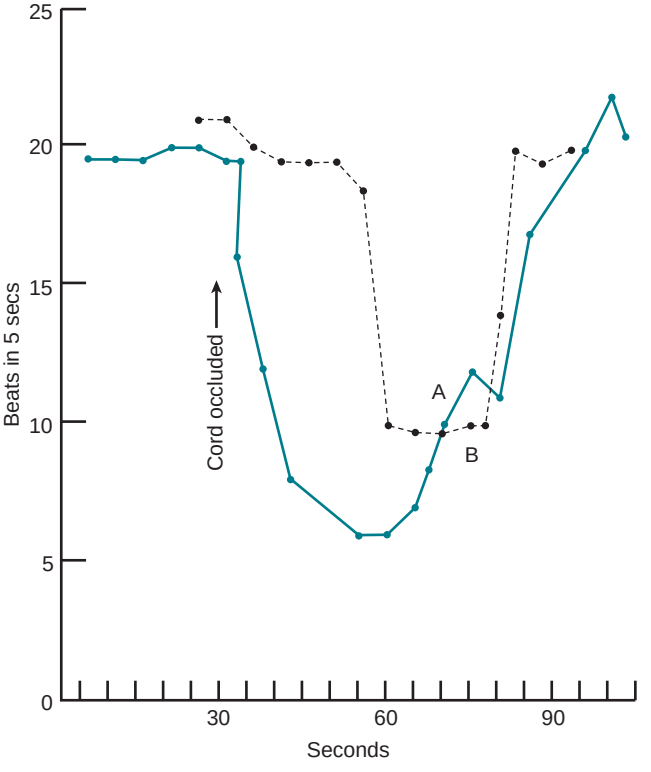

Figure 2.9. The solid line represents the fetal heart rate (FHR) after total umbilical cord occlusion in the intact exteriorized fetal goat. The broken line represents the FHR after total umbilical cord occlusion in the vagectomized exteriorized fetal goat. (From Barcroft J: *Researches on prenatal life*. Blackwell Scientific Publications, Oxford, 1946, with permission.)

the initial component of the FHR deceleration emerge. When the umbilical arteries are occluded, there is a sudden increase in total fetal peripheral resistance resulting from a cutoff of the low-resistance fetal placental circulation. This increase in peripheral resistance in the fetal circulation causes sudden fetal hypertension (45,46). Stimulation of fetal baroreceptors occurs instantly, sending reflexes up the afferent limb of the neural reflex. The baroreceptor impulses affect the central vagal nuclei and result in a parasympathetic outflow that produces a sudden slowing effect on the fetal atrial pacemaker. Fetal ECG changes during cord occlusion show a gradual shortening of the P-R interval, and with profound cord occlusion, the P wave disappears, resulting in a ventricular rate of about 60 BPM (47). With the release of the cord occlusion, the atrial pacemaker returns with a gradual lengthening of the P-R interval back to predeceleration values. Hon et al. (42) have shown that atropine administered to humans will greatly alter, if not abolish, this profound deceleration associated with umbilical cord occlusion. This tends to support Barcroft's hypothesis about the vagal reflex nature of at least one component of the FHR deceleration associated with umbilical cord compression.

This FHR deceleration pattern may bear no consistent temporal relationship to the contraction, presumably because the location of the umbilical cord may vary from one contraction to another. The pattern of cord occlusion has therefore been referred to as *variable deceleration*. Further description of the pattern will appear in Chapter 6.

Siassi et al. (48) have made suggestions, based on observations in neonates, that the afferent limb of this reflex bradycardia, due to umbilical cord compression, may result from changes in arterial pO_2. They noted that neonates on continuous heart rate monitors had heart rate changes similar to variable deceleration after apneic episodes. Because the deceleration seemed to follow the apnea, it appeared that one might explain this phenomenon by a decrease in the neonatal arterial pO_2 (paO_2), causing a chemoreceptor initiation of the afferent limb to the vagal reflex. Next, they looked at neonates on respirators and noted that the delay in the onset of deceleration after cessation of respiratory assistance was related to the level of the paO_2 before the cessation of breathing. The onset of the deceleration indeed appeared to occur at a critical paO_2. This observation would support Barcroft's theory and suggest that the more delayed deceleration was possibly due to hypoxia.

Siassi expanded these studies by returning to exteriorized fetal sheep. By connecting the fetal umbilical circulation to a membrane oxygenator, he was able to show that the onset of variable deceleration after umbilical cord occlusion could be delayed by first raising the paO_2 of the fetus via the membrane oxygenator. Because these sheep were anesthetized, however, one cannot say that the baroreceptor afferent limb does not play a role in variable deceleration. However, these experiments clearly point to the presence of a hypoxemic stimulus as at least having a role in variable deceleration. Certainly, the paO_2 decreases rapidly with total umbilical cord occlusion, but, fortunately, the paO_2 also increases rapidly with release of the occluded cord, accounting for the apparent clinical benignity of variable deceleration patterns in their mild-to-moderate forms.

Itskovitz et al. (49) showed that FHR responses in fetal lambs resembling variable decelerations did not occur until flow was reduced by at least 50%. With partial cord occlusion, there was no significant change in blood pressure and the variable decelerations were abolished by atropine. With partial cord occlusion, the deceleration was of chemoreceptor origin and was mediated via the vagus nerve. Only with total cord occlusion did they see an increase in fetal blood pressure, and they determined that, under those circumstances, the deceleration was of combined chemoreceptor and baroreceptor origin.

Along with the sudden increase in pressure and the sudden decrease in paO_2, there is an acute increase in the pCO_2 during umbilical cord occlusion. This may result in varying degrees of respiratory acidosis, and it is not clear what role this plays in the physiologic mechanism involved in the development of variable deceleration. It is not until cord occlusion is prolonged that a significant oxygen debt develops, resulting in fetal hypoxemia of a more than transient nature as evidenced by the development of metabolic acidosis reflecting significant anaerobic metabolism. When variable deceleration is severe, the second or late component of Barcroft comes into play. It is believed that this component is due to myocardial depression and represents significant hypoxemia and fetal metabolic acidosis (see the discussion of late deceleration below).

It has been suggested that lesser degrees of umbilical cord compression may result in only occlusion of the low-resistance venous return from the placenta. James et al. (50) and Lee et al. (51) have stated that this venous occlusion may result in a decreased return of blood to the fetal heart, decreased fetal cardiac output, and a compensatory FHR acceleration (Figs. 2.10 and 2.11). In the classic experiment by Lee et al. (51) (Fig. 2.10), the umbilical cord of the human fetus was exposed at the time of cesarean section but prior to delivery of the baby. The blood pressure and FHR were measured and the umbilical cord slowly and gradually compressed. The first change was an increase in the FHR (accelerations) as the BP of the fetus dropped due to compression of the less rigid umbilical vein and compromise in the return of blood flow to the fetal heart. This increase in heart rate is a well-known physiologic response to try to maintain cardiac output. With further compression, both the vein and now the umbilical artery were compressed, there was an increase in BP due to the increased peripheral resistance that occurs as the massive amount of blood flow usually going to the placenta is obstructed, and the FHR slows (deceleration), with the normal physiologic protective reflex to increase peripheral vascular resistance. As the cord compression is slowly released, the more rigid walled artery opens first and

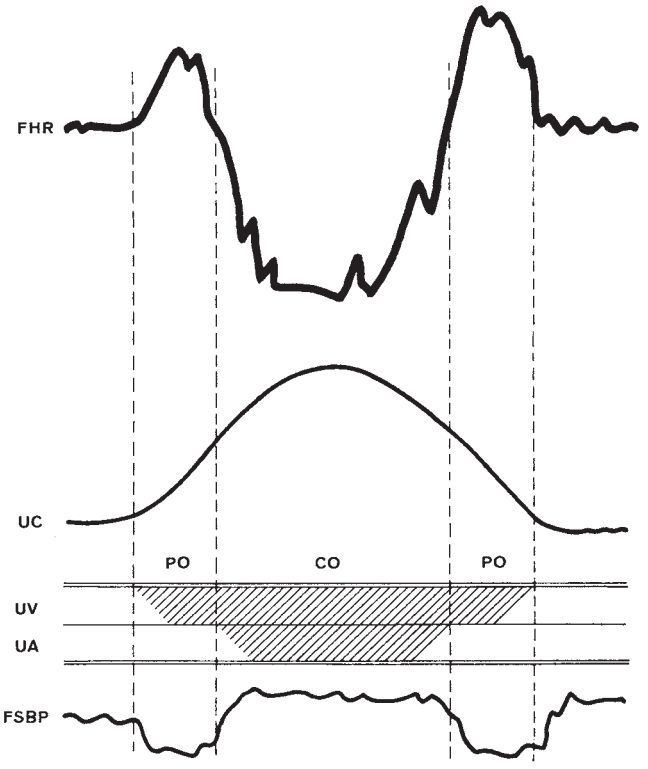

Figure 2.10. This figure represents fetal heart rate (*FHR*) and fetal systemic blood pressure (*FSBP*) occurring during compression of the umbilical vein (*UV*) and the umbilical artery *(UA)*. *UC*, uterine contraction. (From Lee CV, Di Loreto PC, O'Lane JM: A study of fetal heart rate acceleration patterns. *Obstet Gynecol* 45:142, 1975, with permission.)

the BP is then again low as it was when only the vein was compressed and the acceleration that often follows a variable deceleration is seen. Clinical evidence to support this concept comes from the observation that FHR acceleration is often, if not usually, associated with variable decelerations as a typical part of this FHR complex. Thus, it is clear that hypoxia is not necessarily required to produce variable decelerations as changes in blood pressure associated with umbilical cord compression alone can produce this pattern. Even further proof comes from observations with fetal pulse oximetry where even deep and relatively prolonged variable decelerations can be seen without a change in fetal oxygen saturation (Fig. 2.12).

Oligohydramnios may also be associated with variable decelerations even before the onset of labor. The protective nature of amniotic fluid is suggested by the fact that it is less common to see evidence of umbilical cord occlusion in antepartum patients when there are adequate amounts of amniotic fluid present. Gabbe et al. (52) showed that one was able to induce variable decelerations in laboring monkeys by removing amniotic fluid and to relieve them by restoring the amniotic fluid volume. Miyazaki and Nevarez (53) showed that saline amnioinfusion via a transcervical catheter to

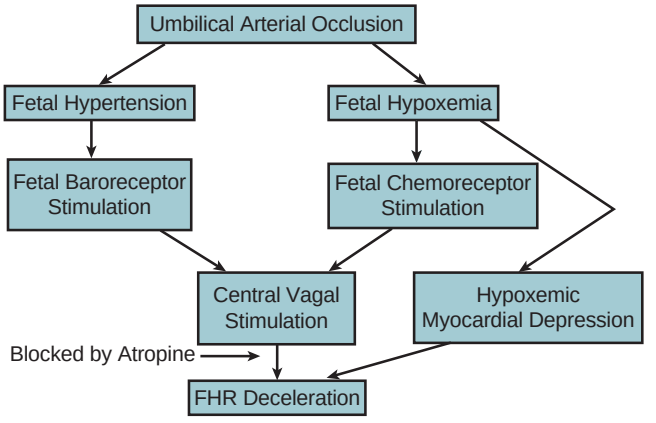

Figure 2.11. Mechanism of variable deceleration.

patients having variable deceleration in labor decreased the variable decelerations, and Nageotte et al. (54) showed, in a randomized prospective study on patients with preterm premature rupture of membranes, that prophylactic amnioinfusion decreased the incidence and severity of variable decelerations and resulted in improved umbilical arterial pH at birth. This technique will be discussed in more detail in Chapter 9.

In summary, the variable deceleration reflex appears to have both baroreceptor (hypertensive) and chemoreceptor (hypoxia) afferent limbs with the efferent limb being vagal. With severe hypoxemia and fetal metabolic acidosis, there may be a delayed deceleration component due to hypoxic fetal myocardial depression. With mild forms of umbilical cord compression, only the vein may be occluded, resulting in FHR acceleration (Fig. 2.10).

Late Deceleration

Earlier in this chapter, it was pointed out that intervillous space blood flow may be diminished by a number of causes. When intervillous space blood flow is decreased to a point that the fetus becomes hypoxemic, UPI is said to exist. Clinical UPI may manifest itself in the chronic form with intrauterine growth retardation, antepartum fetal death, or both; in the acute form, with the onset of fetal distress during labor, birth asphyxia; or in the extreme, intrapartum fetal death.

Before electronic FHR monitoring, there was little known by practitioners of the art of obstetrics about characteristic FHR patterns that are associated with UPI. Among the first to describe FHR changes associated with UPI were Hon (55) and Caldeyro-Barcia et al. (56). They pointed to a slowing of the FHR that was related to uterine contractions with a gradual onset, usually after the peak of the contraction and a delayed return to baseline, usually after the end of the contraction. This was referred to as *late deceleration* by Hon and a type II dip by Caldeyro-Barcia. This late deceleration pattern is believed to have both a reflex component and a hypoxic component somewhat similar to the mechanisms

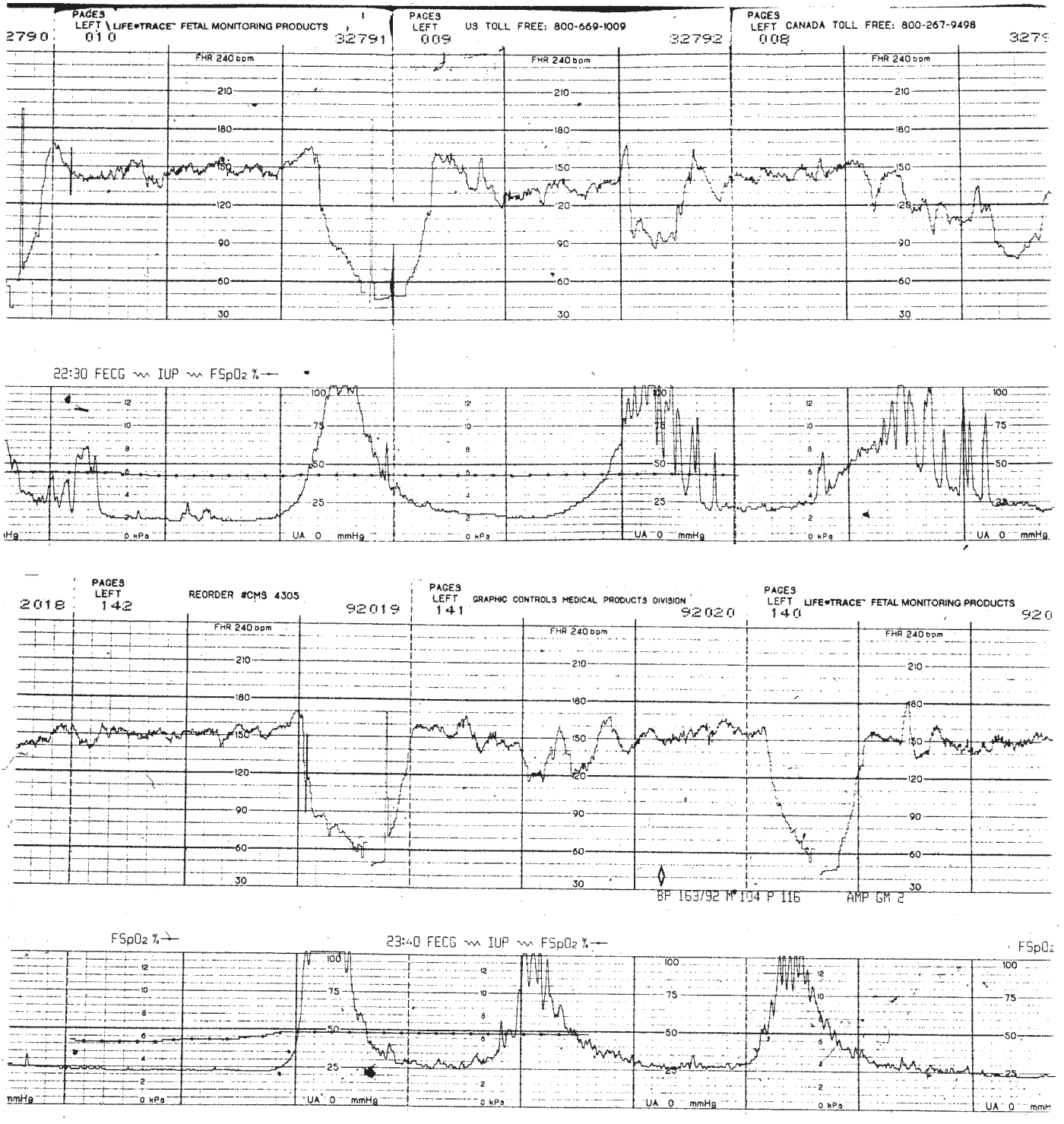

Figure 2.12. This tracing reveals a fetus having variable decelerations in the second stage of labor. The variable decelerations are descending as low as 50 beats per minute and lasting up to 1 minute. Remarkably, despite the depth and duration of the deceleration, the fetal oxygen saturation (the line superimposed on the contraction tracing) is unchanged from its normal level of 50% in this case. This illustrates that changes in peripheral vascular resistance are all that are needed to create a deep variable deceleration even without any change in oxygenation.

described in variable deceleration, but because of the nature and timing of the stimulus, the FHR pattern has different characteristics.

Work by Martin et al. (57) clarifies our understanding of the physiologic mechanisms involved in hypoxemic FHR changes. Martin devised a sheep model with an inflatable cuff that could be used to occlude the ewe's common hypogastric artery and was able to measure blood pressure, heart rate, and blood gases in this chronic fetal sheep preparation. Inflation of the implanted cuff around the common hypogastric artery resulted in cessation of blood flow through the uterine arteries. Intermittent compression could then simulate the changes in uterine blood flow that result from uterine contractions. The FHR changes noted with intermittent hypoxia in this model were a delayed FHR deceleration associated with transient fetal hypertension.

Treatment with phentolamine (α-adrenergic blockade) resulted in a loss of the hypertensive response and a decrease in the late decelerations. Fetal atropine administration (parasympathetic blockade) resulted in periodic FHR accelerations in response to intermittent hypoxia. These accelerations were blocked by propranolol (β-adrenergic blockade). With combined blockade (α-adrenergic, parasympathetic, and β-adrenergic), there was no change in FHR with intermittent hypoxia in the nonacidemic fetus. When the hypoxia was prolonged enough to produce acidemia, the initial hypertensive response was lost and the FHR deceleration occurred even in the presence of triple blockade, indicating that, with hypoxia and acidemia, the FHR deceleration is presumably due to direct myocardial depression.

With the knowledge of this work by Martin et al., it would appear that late deceleration is primarily a reflex change with nonacidemic hypoxia, but when hypoxia is severe enough to result in acidemia, the mechanism of late deceleration appears to be nonreflex or direct myocardial depression. Clinically, the use of fetal scalp blood sampling or fetal scalp stimulation may be of value to determine whether late deceleration is occurring in association with acidemia (Fig. 2.13).

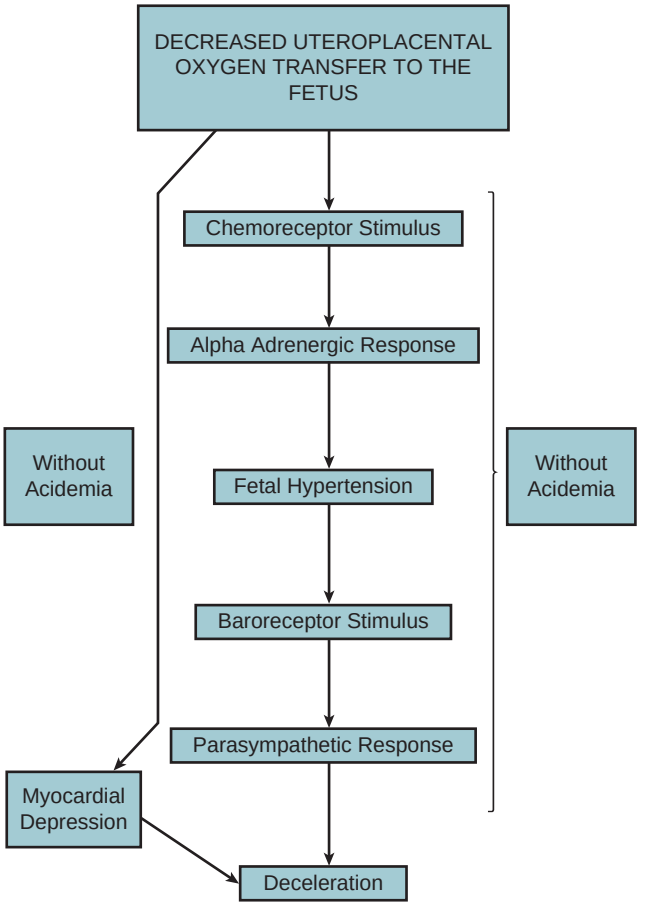

Figure 2.13. Mechanism of late deceleration.

Variability of the Fetal Heart Rate

The interval between successive heartbeats in the intact fetus is characterized by its nonuniformity. This beat-to-beat variability is known as short-term variability. Average interval differences are usually in the magnitude of 20 to 30 milliseconds or 2 to 3 BPM when converted to rate. When variability is diminished, the usual beat-to-beat interval differences average about 1 BPM or less.

The long-term fluctuations in FHR have a cyclicity of 3 to 5 per minute, and the amplitude is usually from 5 to 20 BPM. A long-term variability of <5 BPM is considered to be reduced (Fig. 2.14).

Parasympathetic influences tend to have a short time constant, resulting in more rapid decelerations than the longer time constant sympathetic influences that cause slower and more sustained accelerations. Druzen et al. (58) have shown that the parasympathetic system is more responsible for short-term variability, whereas the sympathetic effect appears to be strongest on long-term variability. Dalton et al. found that, even after double blockade with atropine and β-sympathetic blockade, 35% to 40% of FHR variability remained in fetal sheep. They interpreted this as suggesting some nonneural component to FHR variability (59). However, it is possible that there are some nonautonomic efferent neural components to variability that are operable in the presence of double autonomic blockade. With absent CNS function, as seen in some anencephalics, variability may be completely absent (60). Studies by Modanlou et al. (61) suggested that short-term variability appeared to be reduced early in the course of neonatal hypoxemia, with loss of long-term variability being a later change. Interestingly, with neonatal recovery, long-term variability reappeared first, and the return of short-term variability was delayed.

According to our understanding of the control of FHR, these changes in variability are probably related to changes

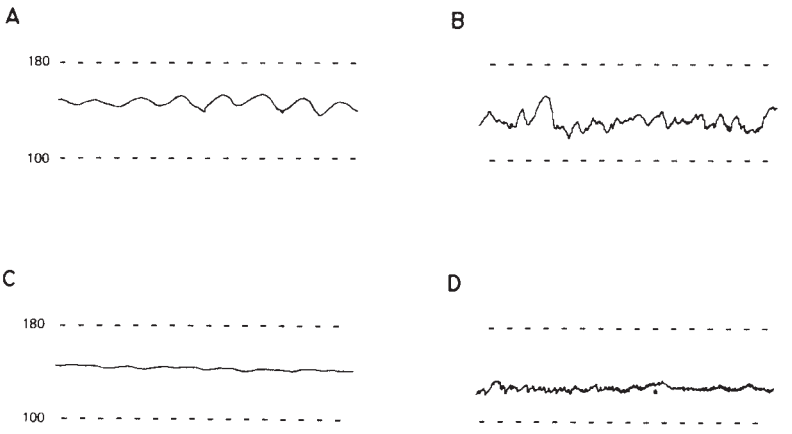

Figure 2.14. Components of fetal heart rate (FHR) variability for fetal electrocardiogram-derived FHR. **A:** Long-term without short-term variability. **B:** Long-term and short-term variability. **C:** No long-term and no short-term variability. **D:** Short-term without long-term variability. (From Zanini B, Paul RH, Huey JR: *Am J Obstet Gynecol* 136:43, 1980, with permission.)

in CNS status. Generally, the intact fetus has good short- and long-term variability. Drugs that depress the CNS or interfere with autonomic reflexes will tend to decrease FHR variability. There also appears to be a gradual increase in FHR variability as gestational age progresses. However, in the viable fetus of more than 28 weeks' gestation, one should not attribute loss of variability to prematurity alone, because most fetuses in the 28- to 32-week range will have reasonably good FHR variability but perhaps slightly less than the term fetus will have.

Although much attention has focused on the difference between short- and long-term variability, they usually go hand in hand—when one is reduced, so is the other. And in more recent effort to create uniform terminology, as will be discussed in detail in future chapters, the clinical distinction between short- and long-term variability has now been abandoned. Nevertheless, it is important to understand the physiologic basis of this very important feature of FHR patterns.

The major significance of FHR variability is that it may be affected by hypoxemia. Druzen et al. (58) showed that the earliest effect of fetal hypoxemia on FHR variability appears to be an increase in both long- and short-term variability. These investigators showed that mild hypoxia resulted in adrenergic discharge and fetal hypertension, causing stimulation of the fetal baroreceptors and a reflex vagal discharge. Thus, a general increase in autonomic tone during early fetal hypoxia results in an increase in both short- and long-term variability. Prolonged and severe fetal hypoxia with acidemia will reduce FHR variability, presumably due to the CNS effects of hypoxia and acidosis.

A curious pattern known as a sinusoidal FHR pattern is exceedingly rare. It is characterized by an absence of short-term variability and a uniform long-term variability pattern in the shape of a sine wave. It has been seen with chronic fetal anemia associated with erythroblastosis (62). It has also been reported with acute intrapartum fetal asphyxia (63), after alphaprodine administration (64), and with fetal-maternal hemorrhage (65). The physiologic basis for this pattern is not clear, but a report by Elliott et al. (66) showed that the pattern observed in the fetus with erythroblastosis was also seen in the neonate and did not respond to adequate neonatal oxygenation but disappeared after neonatal blood transfusion. They suggested that tissue hypoxia may have been relieved by increasing the hemoglobin concentration.

Work by Murata et al. (67) in fetal lambs indicates that anemic fetuses had increased levels of arginine vasopressin but that one could not produce a sinusoidal pattern simply by the infusion of this substance. However, with high doses of atropine or with fetal vagectomy, sinusoidal patterns were produced with high-dose arginine vasopressin infusion. Because cerebral ischemia may result in phasic vasomotor activity, perhaps through alteration in central parasympathetic function, these data suggest that the sinusoidal pattern may involve cerebral ischemia as well as increased arginine vasopressin levels. Certainly, this work suggests that the sinusoidal pattern is related to a change in the CNS control of FHR and implies cerebral ischemia. It is not clear, however, why the pattern is so rare and is usually associated with fetal anemia but can also be seen in hypoxemic fetuses with no anemia.

The FHR is believed to be under the direct control of the fetal autonomic nervous system. We are beginning to understand the reflex mechanisms involved with hypoxic changes in FHR patterns and variability. With early hypoxia, whether caused by cord compression or UPI, the FHR patterns are primarily of neural reflex origin, whereas with severe hypoxia and fetal acidosis, the periodic FHR changes are probably primarily due to myocardial depression. The correlation between physiologic studies and clinical observations remains incomplete, but, clearly, as we learn more about physiologic mechanisms involved in the control of the FHR, the clinical observations become more and more meaningful.

In general, fetal accelerations have the same significance as FHR variability. When the brain is unaffected by hypoxia or other factors that cause CNS depression and the fetus is active, FHR accelerations will be present. When the fetus is

inactive, due to sleep cycles or CNS depressants or due to acidosis, accelerations will be absent. However, unlike FHR variability, the fetus is often not moving and thus has no accelerations even in the absence of CNS depression. Thus, the presence of accelerations can be used to determine that the fetus is not depressed or acidotic, but with the absence of accelerations, the converse is not necessarily true.

FETAL ACID-BASE PHYSIOLOGY

Since the purpose of FHR monitoring is to detect fetal hypoxia and/or acidosis, the discussion of physiology would not be complete without at least a brief review of acid-base physiology. The first step is to understand the effect of hypoxia on the fetus, since hypoxia will come first and is

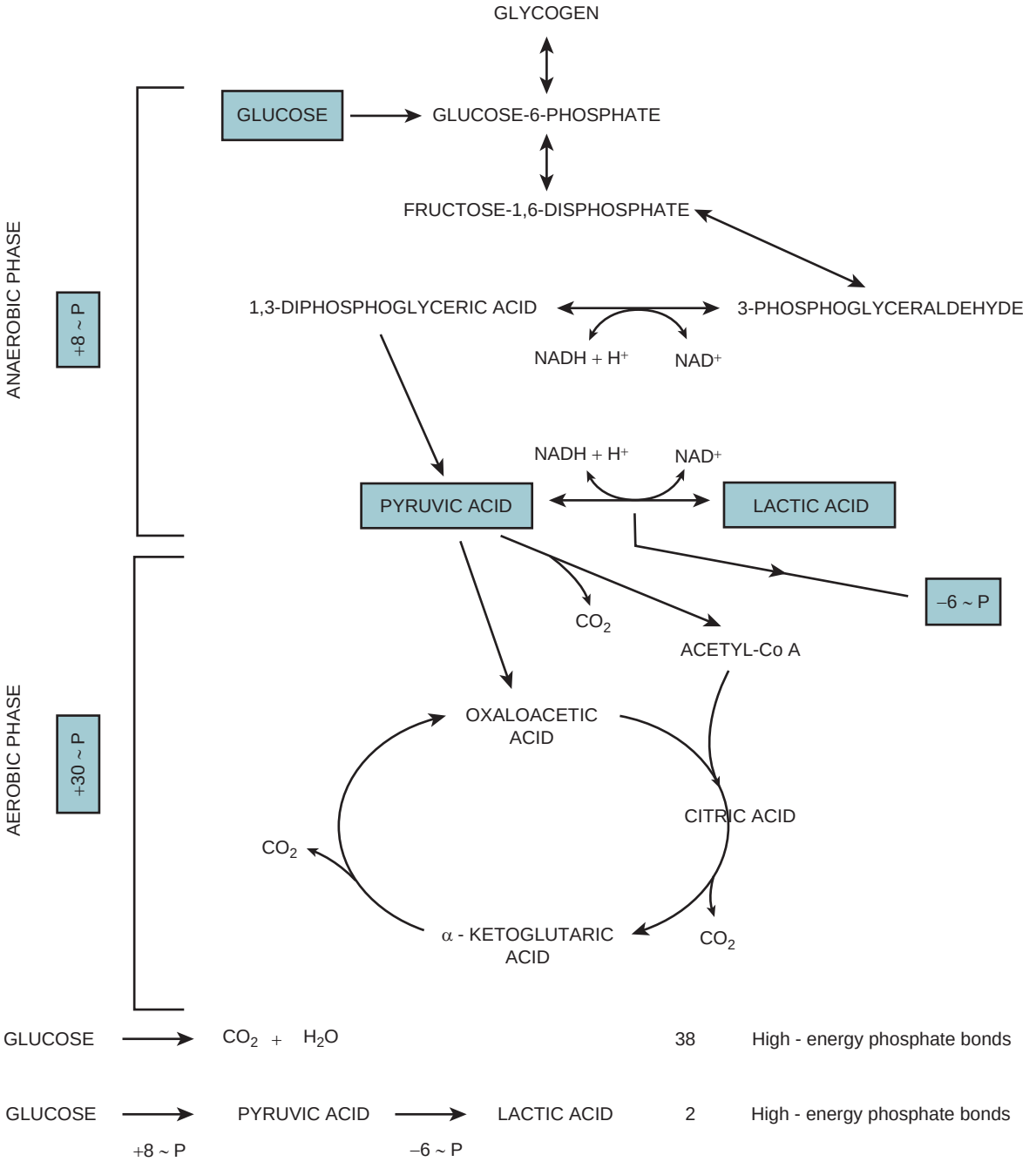

Figure 2.15. The metabolic pathway utilized in glucose catabolism shows that in the absence of oxygen (anaerobic metabolism), the end product is lactic acid, which will result in a metabolic acidosis if not metabolized to CO_2 via aerobic metabolic pathways. (From Hon EH, Khazin AF: Biochemical studies of the fetus I. The fetal pH monitoring system. Obstet Gynecol 33:220, with permission.) (68).

ultimately the cause of acidosis if it occurs. In the absence of oxygen, the fetus will be restricted to anaerobic metabolism, with the production of lactic and pyruvic acids. A brief review of glucose metabolism will serve to remind the reader that glucose is first broken down to lactic acid during the anaerobic phase of the carbohydrate metabolic pathway. There is only a minimal amount of energy produced at this point, but when lactic acid is converted to CO_2 in the presence of oxygen, there is a large amount of energy produced by this more efficient aerobic phase of glucose metabolism. When oxygen is absent, the lactic acid cannot be broken down and it accumulates, causing retention of hydrogen ions, resulting in *metabolic acidosis*. When oxygen is restored, the lactic acid is ultimately metabolized and CO_2 produced, which is easily transferred across the placenta to the maternal circulation. The accumulation of hydrogen ions and depletion of bases, primarily bicarbonate, can be returned to their normal state by diffusion across the placenta once normal aerobic metabolism is restored. However, this process is relatively slow as both H^+ and HCO_3^- are charged molecules, which diffuse across the placenta slowly.

On the other hand, when there is umbilical cord occlusion, whether complete or intermittent, there will first be accumulation of carbon dioxide, similar to airway obstruction in the adult, and only later, if the cord occlusion is prolonged and severe, will hypoxia result. The excess CO_2 is hydrolyzed and carbonic acid (H_2CO_3) is formed. The increase in H_2CO_3 forces the equilibrium toward the dissociated H^+ and HCO_3^- components, resulting in fetal *respiratory acidosis*. This respiratory acidosis can be easily and rapidly reversed when the umbilical cord occlusion is released and CO_2 is equilibrated across the placenta by the maternal circulation (Fig. 2.15) as CO_2, unlike bicarbonate, is rapidly diffusible across the placental membrane.

SUMMARY

An understanding of the physiology of oxygen exchange and the physiologic basis of FHR pattern changes due to normal and pathologic changes in the FHR is essential to a logical approach to management of the fetus in labor. Only with this understanding can correct interpretation and appropriate approaches to intervention occur. Subsequent chapters will build on this new understanding of the physiology behind the changes in FHR in labor and will be better understood if the information contained in this chapter is well understood and remembered.

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CHAPTER 3

Intrauterine Hypoxia and Other Causes of Neonatal Encephalopathy and Cerebral Palsy

Electronic fetal heart rate (FHR) monitoring has been extensively studied with respect to the effects of hypoxemia on the FHR. The relationship between uteroplacental insufficiency, umbilical cord compression, and FHR changes is useful in studying the mechanism of FHR change with respect to intrauterine fetal oxygenation. FHR changes also occur when the fetal central nervous system (CNS) control of FHR is impaired. This impairment may or may not have a hypoxic cause. The concept of CNS damage caused by intrauterine fetal conditions is no longer believed to be limited to hypoxia and trauma. Myriad associations have been described, and even though a substantial percentage of children with neurologic problems never have a precise etiology assigned, recent research has pointed to mechanisms other than hypoxia, including infection, hypercoagulable states, maternal thyroid disease, and a family history of neurologic abnormalities. It is generally agreed that intrauterine hypoxia that progresses to metabolic acidosis in a term fetus, proximate to birth, sufficient to result in later CNS damage, will always be associated with neonatal encephalopathy (1). Recent studies have also pointed to the fact that a substantial portion of cases of neonatal encephalopathy are not caused by intrauterine global fetal hypoxia. Furthermore, localized ischemia due to cerebral infarcts without global hypoxia may cause neonatal encephalopathy and later neurologic damage. Finally, while prematurity has the largest association with later cerebral palsy (CP), the patterns of neurologic deficits differ from those of children who sustained intrauterine damage at or near term.

INTRAUTERINE FETAL HYPOXIA

When the fetus is exposed to insufficient oxygen to allow the complete metabolism of glucose, lactic acid accumulates and results in metabolic acidosis. Acute impairment of the umbilical circulation will result in hypercarbia and respiratory acidosis due to decreased exchange of CO_2, but uteroplacental insufficiency may or may not be associated with hypercarbia because exchange of CO_2 at the placenta is more efficient than is exchange of oxygen. The term fetal asphyxia is overused and should be limited to fetal hypoxia accompanied by metabolic acidosis. Although it has become a popular legal theory, there remains no scientific basis for the notion that cerebral ischemia caused by the pressures of labor and in the absence of fetal hypoxia with metabolic acidosis is a cause of CP.

Fetal circulatory responses to hypoxia include redistribution of blood flow to the more vital organs resulting in preservation of circulation to the brain, myocardium, and adrenal glands. There is a resulting decrease in blood flow to the kidney, intestine, and muscle. Another consequence of hypoxia is a loss of cerebral vascular autoregulation resulting in a pressure-passive circulation. When the level of hypoxemia is near lethal, there is a decrease in fetal cardiac output resulting in hypotension and decreased cerebral blood flow (2–4). It appears that when fetal cardiac output declines, resulting in a decrease in cerebral blood flow below a critical level, neuronal necrosis results. Subsequent development of cerebral edema in the neonate further compromises cerebral blood flow, aggravating the degree of neuronal necrosis.

When fetal hypoxia occurs at sublethal levels, permanent damage to the fetus may result; however, the frequency with which this occurs in the intrapartum period and the ability of the clinician using fetal monitoring to prevent it are a subject of intense debate. References to perinatal brain damage can be found earlier, but the English orthopedic surgeon, Little (5), is credited with the first specific hypothesis suggesting adverse perinatal events as the main etiologic factors in infantile spastic palsies. He reviewed the histories of more than 200 cases of spasticity of congenital origin and presented his paper entitled "On the influence of abnormal parturition, difficult labours, premature birth and asphyxia neonatorum, on the mental and physical condition of the child, especially in relation to deformities" to the Obstetrical

Society of London. He concluded that these 200 cases had one thing in common, that is, some abnormal characteristic of parturition. The form of CP that he described has often been called "Little's disease." Little later went so far as to conclude that virtually nothing other than abnormalities of birth could cause the clinical picture he described (5). Subsequently, other neurologic problems including mental retardation, epilepsy, and behavioral and learning disorders have been attributed to various intrapartum problems.

Animal data corroborating the concept that perinatal asphyxia may cause profound sublethal neurologic damage can be found in the classic papers of Windle and Becker (6). Between 1943 and 1963, Windle (7) performed experimental asphyxiation of rhesus monkeys and evaluated the immediate, long-term, and neuropathologic effects. The monkeys were asphyxiated in one of two ways, both involving hysterotomy. Either the placenta and membranes were delivered with the fetus within the intact amniotic sac or the fetal head was covered by a fluid-filled sac and the umbilical cord was completely occluded. The fetal oxygen supply was completely interrupted for 5 to 20 minutes. Monkeys allowed to breathe in 6 minutes or less showed no neurologic deficits and no pathologic brain changes. Asphyxiation for more than 7 minutes produced at least transient motor and behavioral changes and relatively consistent brain pathology, along with necrosis of brain-stem cells in the inferior colliculi and ventrolateral thalamic nuclei with secondary glial proliferative scarring. The most profound changes were found in monkeys left anoxic for 12 to 17 minutes. Resuscitation was invariably necessary. More severe brain-stem lesions were created. Initially, the monkeys were hypoactive and hypotonic. Seizures, ataxia, and athetosis were often seen. The monkeys were observed for 8 years and, as they matured, most deficits gradually improved, leaving residual hypoactivity and clumsiness. Although this model indeed supported the hypothesis that perinatal asphyxia is associated with permanent neurologic damage, the pattern of damage did not seem to correspond with the mental retardation and spasticity seen most commonly in humans. In later experiments, Windle (7) subjected monkeys to prolonged labor with oxytocin, and the asphyxiated neonates did not show the midbrain and brain-stem lesions of acute total asphyxia but rather primarily cortical damage.

Myers (8) suggested that total asphyxia may not be the usual case in humans and that prolonged partial asphyxia is more likely. Myers was able to produce partial asphyxia in the rhesus monkey by various techniques including oxytocin-induced tachysystole, compression of the maternal abdominal aorta, maternal infusion of catecholamines, and inspiration by the pregnant monkey of reduced oxygen concentrations. These were controlled by maintaining fetal pO_2 at 5 to 9 mm Hg. Myers et al. (9) later demonstrated that late decelerations of the FHR were caused by this fetal hypoxemia. Fetuses were maintained in such partially asphyctic states for at least 1 hour, then delivered and resuscitated. The immediate effect on the newborn was flaccidity, which evolved after several hours into generalized hypertonus and decerebrate posturing, at which time the newborns began to have periodic generalized seizures. Many fetuses developed ileus and cardiogenic shock, and then died. A minority of fetuses survived. Extensive histopathologic examination of the brain led Myers to conclude that such prolonged partial hypoxia led to a vicious cycle of brain swelling, which caused decreased cerebral blood flow that further aggravated the brain swelling. In extreme degrees, such diminished blood flow led to total hemispheric cortical necrosis. In lesser degrees, cortical damage was seen in the middle third of the paracentral cerebral cortex and in the basal ganglia. Such lesions correspond closely with the intellectual deficits and spastic motor defects seen in humans in whom prolonged intermittent asphyxia is more common than acute total asphyxia as described by Windle.

ETIOLOGIC FACTORS IN CEREBRAL PALSY

Many authors have reviewed obstetric histories of children with congenital neurologic damage. Early in his career, Freud became interested in the etiology of CP. In his 1897 monograph, *Die Infantile Cerebrallahmung* (10), Freud concluded that one-third of the cases were the result of traumatic birth, one-sixth consequent to prematurity, one-sixth were of prenatal or postnatal cause, and one-third unknown. Interestingly, Freud questioned whether the real etiology of the damage was the birth process or if abnormalities seen at birth were a reflection of a previously existing abnormality. For example, Nelson and Ellenberg (11) found that a third of breech deliveries had major malformations, hence a conclusion that abnormal development was caused by breech delivery rather than the reality that existing fetal abnormalities contribute to the incidence of breech presentation. Torfs et al. (12) found a 3.8-fold increase in CP for breech-delivered children over those presenting as a vertex. Lilienfeld and Pasamanick (13) reviewed birth certificates of 561 congenitally spastic children and found a very high incidence of abruptio placentae and placenta previa. Eastman and DeLeon (14) reported an analysis of 96 obstetric records of infants in whom CP developed. Only 18 of these births were uncomplicated; 34 babies were premature. Of the 96 infants, 30% had apnea of more than 30 seconds at birth. Compared with controls, there was a doubling of third-trimester hemorrhage, a threefold increase in breech delivery, a fourfold increase in both anesthetic complications and fetal distress by auscultation, and a tenfold increase in shoulder dystocia among affected infants. Eastman's findings in a subsequent, more extensive review (15) of 753 cases are summarized in Table 3.1.

Finally, Steer and Bonney (16) in 1962 reviewed 317 patients with CP and concluded that 5% were a result of kernicterus, 8% were caused by congenital defects and neurologic infections, and 87% were cases "with possible obstetric causes."

Thus, for many years, it has been the impression that CP was primarily caused by perinatal events. Fetal monitoring was developed with the hope that, by identifying fetal

TABLE 3.1 Obstetric background of 753 cases of CP (16)

Background	CP (%)	Control (%)
Postnatally acquired	8.5	
Premature (<2,500 g)	29.0	8.0
Twins (rate twin A = rate twin B)	7.0	1.0
Mid/high forceps	8.0	5.0
Breech	9.0	3.5
Resuscitated at birth	27.0	3.0
Hypoxia	11.0	4.0
Cord prolapse	3.0	.3
Abruptio placentae	4.0	1.3
Toxemia	5.0	2.0
Prolonged labor	2.4	1.6
Hemolytic disease	6.0	.3
Congenital anomalies	5.0	1.3

CP, cerebral palsy.

hypoxia early enough and with prompt and appropriate intervention, death and damage could be prevented (17).

Contrary to the classic theory of intrapartum hypoxia as the major cause of CP, epidemiologic studies have shown that only a small percentage of CP is attributable solely to intrapartum events and that most cases of CP with intrapartum factors also have antepartum risk factors. In a 1998 case-controlled review by Badawi et al., involving 164 term infants with moderate or severe neonatal encephalopathy compared with 400 term control infants without neonatal encephalopathy, antepartum and intrapartum risk factors for the development of CP were categorized. When adjusting for antepartum risk factors, they concluded that only maternal pyrexia during labor, occipital posterior presentation, an acute intrapartum event, instrumental vaginal delivery, emergency cesarean section, and general anesthesia were significant intrapartum risk factors for neonatal encephalopathy (Table 3.2). Such things as general anesthesia and emergency cesarean section are not believed to be causative in themselves but by association with the reasons for these interventions. They concluded that 70% of term or near-term neonates with neonatal encephalopathy had only antepartum risk factors with no evidence of adverse intrapartum events, 25% had evidence of antepartum risk factors and intrapartum hypoxia, and only 4% had only intrapartum hypoxia as a risk factor (18). A problem with this analysis assumes that if any antepartum risk factor is present, the cause cannot be only intrapartum. For example, if a patient has antepartum pre-eclampsia and enters labor with normal electronic fetal monitoring (EFM) and subsequently has a prolapsed cord with prolonged deceleration or bradycardia and profound metabolic acidemia at birth, this would not be an isolated intrapartum event. The small contribution of intrapartum asphyxia as a cause of CP probably accounts largely for the fact that intrapartum FHR monitoring has

TABLE 3.2 Risk factors for neonatal encephalopathy

Preconceptional factors	Antepartum factors	Intrapartum factors	Decreased risk
Increasing maternal age	Maternal thyroid disease	Intrapartum fever	Elective C/S
Unemployed, unskilled worker, or housewife	Severe preeclampsia	Prolonged rupture of membranes	
No private health insurance	Bleeding in pregnancy	Thick meconium	
Family history of seizures	Viral illness during pregnancy	Malpresentation and malposition	
Family history of neurologic disorders	Postdate pregnancy	Intrapartum hypoxia	
Infertility treatment	Growth restriction in the fetus	Acute intrapartum events	
	Placental abnormalities	Forceps delivery or emergency C/S	

Indicates risk factors that are significant by multiple logistic regression analysis of term infants with neonatal encephalopathy compared with term infants without neonatal encephalopathy.
C/S, cesarean section.
Adapted from Manning FA, Bondaji N, Harman CR, et al: Fetal assessment based on fetal biophysical profile scoring. VIII. The incidence of cerebral palsy in tested and untested perinates. *Am J Obstet Gynecol* 178:696–706, 1998.

been disappointing as a strategy to prevent CP (19,20), but the epidemiologic analysis of Badawi probably underestimates the actual intrapartum contribution. A recent population-based study of children with CP in California revealed that about one-third of 7,242 children with CP had an adverse intrapartum identifiable event compared to 12.9% in controls without CP. The effect was more pronounced in children born prematurely (36.8%) than children born at term (28.3%). Maternal and neonatal infections were also significantly more common in birth records of children with CP (21). While the various estimates of the contribution of acute intrapartum events to the development of CP continue to vary between 5% and 35%, it is clear that adverse intrapartum events are a definite contributor to later CP.

FORMS OF CONGENITAL NEUROLOGIC DAMAGE RELATED TO FETAL HYPOXIA

Cerebral Palsy

CP caused by intrapartum fetal hypoxia will always have evidence of encephalopathy in the neonatal period and will be of the spastic quadriparetic or dyskinetic type. Unilateral brain lesions are not likely to be due to global hypoxia as is seen in intrapartum asphyxia (22,23). A recent large population-based survey placed the incidence of neonatal encephalopathy at 3.8 per 1,000 term infants and the incidence of hypoxic ischemic encephalopathy at 1.9 per 1,000 term infants (24). In the collaborative project, the incidence of neonatal encephalopathy was 5.4 per 1,000 births weighing more than 2,500 g. Cerebral palsy, defined as "a persistent but not changing disorder of movement and posture, appearing in the early years of life and due to a nonprogressive disorder of the brain" (15), will develop in approximately 5 infants per 1,000 births, with a prevalence of one to two per 1,000 school-age children. As a result, CP affects 350,000 children in the United States today (14,25,26). Approximately 50% have mild intellectual retardation (IQ <70), and one-fourth are severely affected (IQ <50). One-fourth of children with CP have a seizure disorder (25). CP is classified according to the distribution of extremities involved (diplegia, paraplegia, tetraplegia, hemiplegia) and the type of movement disorder. Diplegia, a commonly used term, implies bilateral lower extremity involvement. Motor symptom classification describes the dominant movement disorder and includes spasticity, dyskinesia (athetosis), and ataxia.

Spastic diplegia is the abnormality most commonly associated with prematurity (27). Infants born prematurely are also more likely to have periventricular white matter damage (periventricular leukomalacia [PVL]) than infants born at or near term. PVL is frequently associated with intraventricular hemorrhage and ventriculomegaly (28). One study showed that abnormalities of the umbilical cord and frequent moderate variable decelerations seen in premature infants were more common in infants that were shown to have PVL (29). There is no evidence that intervention for moderate variable decelerations in a premature fetus is indicated in that PVL did not develop in more than one-fourth of premature fetuses that also had frequent moderate variable decelerations.

Mental Retardation

Estimates of severe mental retardation are surprisingly uniform from country to country at about 3.5 per 1,000 population (30). Mild retardation is somewhat more variable, occurring in 23 to 31 per 1,000, probably because of testing inaccuracy and the effect of environment in this group (30). Mental retardation is a much less specific result of perinatal asphyxia than is CP. Perinatal causes are estimated to be responsible for approximately 10% of cases of mental retardation (31,32); chromosome abnormalities and various hereditary disorders, 65%; infection, 5%; prenatal causes such as toxins and maternal disease, 10%; and the rest are unknown (33). Many studies, including the Collaborative Perinatal Project (34), have conducted prospective analyses of children determined to be asphyctic at birth, examining several criteria including Apgar score, neonatal apnea, shock, or acidosis. The vast majority (generally >90%) have normal IQs, and the mean IQ is usually only 5 to 10 points below average. Mental retardation without associated CP is not believed to be caused by fetal asphyxia (35).

Epilepsy

Although the potential for hypoxia to cause seizures in both newborns and adults is acutely clear, there is not a strong relationship between perinatal events and epilepsy. The Collaborative Perinatal Project did not demonstrate an increase in epilepsy in low-birth-weight infants or in depressed infants (36); however, epilepsy is found more commonly in infants with CP and mental retardation. Epilepsy without CP is not attributable to fetal asphyxia (35).

Behavioral and Learning Disorders

Because most depressed neonates do not have demonstrable intellectual impairment, many have questioned whether indeed such infants do eventually reach their full intellectual potential. The data suggest the opposite because many children born hypoxic and acidotic, with demonstrated intellectual impairment in infancy and preschool periods, will test in normal ranges later on. Whether this implies dissipation of the effects of hypoxia with catch-up intellectual growth or limitations of testing is open to question. Some of Windle's severely asphyxiated monkeys had structural brain defects despite apparent normal behavior. The subtleties of behavioral difficulties and learning disorders make the problem very difficult to analyze. Nichols and Chen (37) found that hyperactivity and learning disorders correlated weakly and inconsistently with perinatal asphyxia. Thus, current knowledge does not link isolated behavioral or learning disorders to fetal hypoxia.

OTHER FACTORS ASSOCIATED WITH NEONATAL ENCEPHALOPATHY AND CEREBRAL PALSY

Asphyxia is one cause of prenatal and perinatal neurologic damage. Other factors may be causative or contributive, or indeed, as pointed out by Freud (10), in babies apparently distressed and hypoxic at birth, there may have been precedent damage or anomaly unrelated to hypoxia.

Prematurity

The association of prematurity and perinatal neurologic insults and their sequelae have long been recognized. Shakespeare's King Richard III, then Duke of Gloucester, proclaimed:

> "I that am curtailed of this fair proportion,
> Cheated of feature by this dissembling nature,
> Deformed, unfinished, sent before my time
> Into this breathing world, scarce half made up—
> And that so lamely and unfashionable
> That dogs bark at me as I halt by them— …"

Little pointed out this passage in presenting his paper and documented a high association between spastic rigidity and prematurity.

Of all perinatal factors that are identifiable as being related to CP, prematurity has the highest correlation (11). Many of these data, however, come from 30 or more years ago. Improvements in neonatal care have decreased the incidence of neurologic damage in very-low-birth-weight babies, but survival rates have increased so much that the contribution of prematurity to CP rates becomes very difficult to analyze.

Prolonged Pregnancy

The risk for neonatal encephalopathy increases progressively after 39 weeks gestational age at birth. Badawi et al. (18) showed the risk at 40 weeks was increased 1.41 times; at 41 weeks, 3.34 times; and at 42 weeks, 13.2 times. Postterm birth had less impact when controlled for small for gestational age and maternal age (38).

Fetal Growth Restriction

Fetal growth restriction (FGR), also known as intrauterine fetal growth retardation (IUGR), may be caused by perinatal infections, teratogens, congenital anomalies, inadequate nutrition, or uteroplacental insufficiency in which a high incidence is associated with birth asphyxia. Fitzhardinge and Steven (39) observed 96 full-term growth-retarded infants (excluding anomalies and congenital infections) up to 8 years of age. CP occurred in 1% and epilepsy in 6%; however, "minimal cerebral dysfunction (learning difficulties, hyperactivity, and poor coordination) were found in 25%." Of these infants, 30% had speech problems and 40% poor school performance. In a case-controlled review, Badawi (40) found IUGR to be the most significant antepartum risk factor for the development of neonatal encephalopathy with an odds ratio of 4.37 between the third and ninth percentiles and 38.23 for FGR below the third percentile. Because this group of fetuses/newborns is known to have a high incidence of perinatal asphyxia, it is difficult to determine whether an antepartum respiratory and/or nutritional deficiency, intrapartum asphyctic insult, or some combination is responsible for such neurologic damage.

Traumatic Birth

The relatively high incidence of midforceps, high forceps, and breech deliveries in retrospective studies of neurologically damaged babies (14,15) suggests that birth trauma may have played a contributing role. In breech births, further evidence is provided by the reduced incidence of these problems with elective cesarean section (41). Because difficult operative vaginal deliveries have decreased, they are probably a less frequent cause of neurologic damage.

Prolonged Labor

Friedman et al. (42–44), in their series of studies concerning the effects of prolonged labor on adverse developmental outcome, have demonstrated that this also may have contributed to sublethal CNS damage but that this may not be as important as the means of delivery (i.e., midforceps). Because Friedman's work was largely done before EFM, it is difficult to know if the association with prolonged labor was due to fetal hypoxia or other causes such as infection.

Anesthesia and Analgesia

Drugs and anesthesia may contribute to neurologic damage in one of two ways. Regional anesthesias may cause maternal hypotension with resultant fetal hypoxemia from decreased uterine perfusion. Depressants, whether anesthetic agents or narcotics, may cause neonatal apnea. In a setting with availability of good appropriate neonatal resuscitation, this should contribute little to hypoxic-ischemic damage.

Genetic Factors

Congenital anomalies are associated with high incidences of CP, mental retardation, epilepsy, and developmental disabilities. Generally, researchers evaluating mental retardation report higher incidences of associated congenital defects than those studying CP. In a series of 1,410 autopsies from three hospitals for the mentally retarded, Malamud reports a 61% incidence of anomalies, with

Down syndrome the most frequent single cause (45). Fetuses with a family history of neurologic disease or seizures had a twofold to threefold increase in neonatal encephalopathy (35) and CP (11). This suggests a relation to genetic or early developmental causes. Some inherited metabolic abnormalities also are causes of neonatal encephalopathy and CP (46).

Fetal Infections

Congenital infections can be associated with CNS damage with or without microcephaly or hydrocephaly. Rubella, cytomegalovirus, syphilis, and toxoplasmosis (TORCH infections) are among well-known causes. As in anomalies, congenital TORCH infections are more likely to contribute to mental retardation and somatic growth retardation than to CP. They are estimated to account for 10% of mentally retarded children in developed countries (30).

The fetal inflammatory response associated with maternal fever during labor, chorioamnionitis, and funisitis has been implicated as a cause of later CP (47–49). It is believed that inflammatory cytokines can cause cerebral ischemia resulting in damage to the paraventricular area of premature fetal brains (50–54). These lesions appear as PVL and intraventricular hemorrhage. The relation between chorioamnionitis and CP in term fetuses has been demonstrated by Grether and Nelson (55,56). Cytokines have also been implicated in term fetuses (57). As our knowledge of infectious causes of CP increases, it may account for some of the large percentage of CP with unknown cause, and strategies for intervention may become evident.

Coagulation and Autoimmune Disorders

Infants whose blood contains indicators of coagulation or autoimmune disorders have a higher incidence of neonatal encephalopathy, stroke, and, later, CP, especially of the hemiparetic subtype (55,58,59). One study looking at placentas of children in whom CP developed showed that thromboses were the most frequent finding (60). This study also showed that some of these mothers had a history of pregnancy loss and some had histories of autoimmune disorders. Thyroid disorders in mothers are a potent risk factor for neonatal encephalopathy and may be related to autoimmune mechanisms (61). Neonatal encephalopathy resulting from cerebral infarcts due to coagulation disorders may closely resemble hypoxic ischemic encephalopathy. Neonates with encephalopathy should be evaluated for thrombophilias and autoimmune disease.

Toxins

Fetal neurotoxins may be the least understood causative factors in neurologic congenital diseases. Certainly, there are examples of environmental toxins (methyl mercury), drugs (folic acid antagonists such as methotrexate), and food and drink substances (alcohol) that are known to affect fetal neurologic development and cause serious mental retardation and even spasticity. Other environmental toxins may be responsible for a portion of the large group of cases of CP and mental retardation of unknown cause.

Antenatal Factors

The contributing role of antenatal factors is perhaps the most controversial and difficult to analyze of the factors involved in neurologic damage. Antenatal insult probably contributes to FGR, and when one observes the high incidence of antepartum bleeding and toxemia in the histories of children with neurologic damage, the contribution of prepartum damage becomes apparent. These babies often appear depressed at birth and have intrapartum FHR tracings that indicate fetal hypoxemia, but the damage may have occurred before the onset of labor.

Factors other than intrapartum hypoxia do seem very important in the pathogenesis of congenitally acquired neurologic damage. In assessing what contribution can be made with intrapartum assessment techniques, consideration must be given to such insults that may have occurred before the onset of labor. Approximately 75% of cases of CP have no history of neonatal depression or perinatal insults. Furthermore, a large percentage of neonates with encephalopathy and CP have causes other than global hypoxia. Therefore, the abnormally developing child with features consistent with CP should not be assumed to have suffered antenatal or perinatal asphyxia.

OTHER ORGANS AFFECTED BY HYPOXIC-ISCHEMIC DAMAGE

Redistribution of blood flow in response to fetal hypoxia affects non-CNS organs acutely. Neurologic damage is not the only sublethal result of intrauterine fetal hypoxemia. It is well known that the lungs, kidney, and gastrointestinal tract are also sensitive to hypoxic ischemia and that newborn sequelae may result; however, nonneurologic damage usually repairs itself (62).

Respiratory Distress Syndrome

Respiratory distress syndrome (RDS) is the leading cause of neonatal death and serious morbidity in prematurity. The primary etiologic factor is generally thought to be inadequate pulmonary surfactant. The synthesis of pulmonary lecithin (the major component of surfactant) is significantly diminished by hypoxia and acidosis (63). Even in infants with mature ratios of lecithin to sphingomyelin, depressed

and acidotic babies are more likely to have RDS (64–66). Hobel et al. and Martin et al. have pointed out that premature newborns, acidotic babies, and those with abnormal heart rate patterns suggesting hypoxia had both higher incidences of and higher mortalities from RDS. Furthermore, Martin found ominous FHR patterns to be even more predictive of RDS than Apgar scores.

Renal Damage

The association between hypoxia and renal damage, that is, anuria and renal failure similar to that in the adult, has been known since 1920. Animal data suggest such ischemic damage may be the result of the decreased renal blood flow after blood redistribution with hypoxia (67–69). Premature fetuses seem more susceptible to renal damage from ischemia.

Gastrointestinal Damage

Similarly, when blood flow is redistributed during periods of fetal hypoxia, blood flow to the fetal gastrointestinal tract is particularly diminished. Alward et al. (70) have shown that in animal fetuses, asphyxia could lead to decreased blood flow to the gastrointestinal tract, thus resulting in dilation of segments of the large and small bowel and scattered mucosal necrosis. Towbin and Turner (71), in looking at autopsies with other hypoxic-ischemic damage, found intestinal injuries due to venous stasis and infarction, with lesions ranging from focal mucosal necrosis to massive gross intestinal infarction. Necrotizing enterocolitis (NEC), seen most frequently in premature infants, may at least in part be the result of such damage. Preliminary data suggest abnormal FHR patterns are often seen in fetuses destined to develop NEC (72).

EVIDENCE THAT ABNORMAL FETAL HEART RATE PATTERNS CORRELATE WITH INTRAUTERINE HYPOXIA-ISCHEMIA WITH METABOLIC ACIDOSIS

Knowing the correlation of intrauterine fetal hypoxia, ischemia and metabolic acidosis, and other intrapartum events with sublethal neurologic damage, one must demonstrate the relationship of abnormal FHR patterns with hypoxia and metabolic acidosis. If Apgar scores are used to measure neonatal depression, one must show the association of neonatal depression with fetal hypoxia and acidosis. The latter was well demonstrated by Modanlou et al. (73). Biochemical changes, especially pH and base deficit, have a statistically significant correlation with depressed newborns (74).

While it is not within the realm of this chapter to discuss the mechanisms responsible for the genesis of FHR patterns, it has been established that many FHR changes are triggered by fetal hypoxemia, which can progress to metabolic acidemia and have been associated with neonatal depression. Barcroft (75) first demonstrated the typical variable-type deceleration caused by umbilical cord occlusion in the fetal goat. Subsequently, Lee and Hon (76) were able to reproduce this in the human fetus. Myers et al. (9) were able to reproduce late decelerations in the rhesus monkey by precipitating uterine artery hypotension.

Kubli et al. (77) described the relationship of human fetal scalp pH with various FHR patterns and demonstrated that the more severe the pattern, the more likely the fetus is to be acidemic. However, the correlation between pH and severity of FHR pattern abnormality had a large standard deviation. The correlation with fetal base deficit was much tighter. Paul et al. (78) further refined this work. They found that, with both acidosis and depressed Apgar scores, the FHR variability associated with late decelerations had a higher correlation than amplitude of the late decelerations. Many subsequent studies have supported the parallel relationship of abnormal FHR patterns and metabolic acidemia.

Many studies have linked FHR patterns to Apgar scores, showing generally that the abnormal tracings are more likely to result in a depressed baby at birth. Because there are really no studies in which fetal monitor patterns are blinded and not acted upon, intervention may have decreased the degree of correlation because in most patterns, a certain time is necessary from the onset of hypoxemia to the development of metabolic acidemia. Bisonette (79) has produced one of the most detailed retrospective analyses of FHR patterns and Apgar scores (Table 3.3). Schifrin and Dame (80) attempted to predict Apgar scores from FHR patterns obtained within 30 minutes of delivery without regard to clinical circumstances during labor. When a normal baby was predicted, they were nearly always right; when a depressed baby was predicted, they were more often wrong, but when a baby was born depressed, it was nearly always predicted (Fig. 3.1). Clearly, neonates with intrapartum hypoxia sufficient to cause death or damage will always have FHR patterns reflecting the severe hypoxia. Because there are other causes of neonatal depression besides intrapartum hypoxia-asphyxia, one would not expect to find 100% correlation between a predicted healthy baby and a normal Apgar score.

It can be concluded, therefore, that FHR patterns suggesting fetal hypoxia and/or acidosis do correlate with depression at birth. One must be careful to point out that (a) not all abnormal patterns are associated with poor outcome, (b) the extent to which intervention ameliorates or prevents adverse outcome is unknown, (c) EFM corresponds best and is most valuable when the pattern is normal, and (d) FHR patterns, by themselves, are not predictive of later neurologic outcome (81).

TABLE 3.3 Relationship between FHR pattern and 1-minute Apgar score

				Apgar 4–6 (moderate depression)		Apgar 0–3 (marked depression)	
FHR pattern	Number	Percent	Mean Apgar	Number	Percent	Number	Percent
Normal	322	45.0	8.2	16	5.0	3	.9
Uncomplicated baseline tachycardia	24	3.4	7.9	1	4.2	1	4.2
Uncomplicated baseline bradycardia	38	5.3	8.1	3	7.9	0	0
Uncomplicated loss of beat-to-beat variation	40	5.6	7.2	7	17.5	2	5.0
Complicated loss of beat-to-beat variation	11	1.5	6.7	2	18.2	2	18.2
Acceleration	52	7.3	8.4	1	1.9	0	0
Early deceleration	122	17.1	8.0	6	4.9	1	.8
Late deceleration	20	2.8	5.4	7	35.0	5	25.0
Variable deceleration with normal baseline	71	9.9	7.7	9	12.7	2	2.8
Variable deceleration with abnormal baseline	14	2.0	5.9	0	0	7	50.0

FHR, fetal heart rate.
From Bisonette JM: Relationship between continuous fetal heart rate patterns and Apgar score in the newborn. *Br J Obstet Gynaecol* 82:24, 1975, with permission.

CRITERIA NECESSARY TO ASSIGN ACUTE INTRAPARTUM ASPHYXIA AS A CAUSE OF NEONATAL BRAIN DAMAGE

In the third edition of this textbook, the criteria necessary to assign acute intrapartum hypoxia-asphyxia as a cause for neonatal brain damage were derived from a 1992 ACOG technical bulletin, and there were also references to the Task Force on Cerebral Palsy and Neonatal Asphyxia of the Society of Obstetricians and Gynecologists of Canada and the Perinatal Society of Australia and New Zealand. In 2003, the ACOG and AAP published a revised statement (82) in a publication entitled Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysiology. This publication was endorsed by

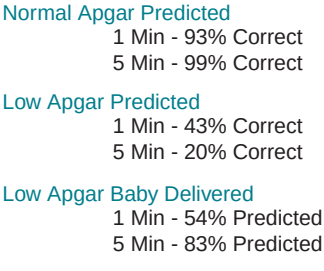

Figure 3.1. Reliability of prediction of Apgar score from fetal heart rate tracings. (From Schifrin BS, Dame L: Fetal heart rate patterns. Prediction of Apgar score. *JAMA* 219:1322, 1972.)

- The CDC
- The Child Neurology Society
- The March of Dimes
- The NICHD
- The Royal Australian and New Zealand College of Obstetricians and Gynecologists
- The Society of Maternal and Fetal Medicine
- The Society of Obstetricians and Gynecologists of Canada

While the criteria published in this monograph were similar to those in the third edition, there were some changes based on the most recent evidence, which follow.

CRITERIA TO DEFINE AN ACUTE INTRAPARTUM HYPOXIC EVENT AS SUFFICIENT TO CAUSE CEREBRAL PALSY

Essential Criteria (must meet all four)

1. Evidence of a metabolic acidosis in fetal umbilical cord arterial blood obtained at delivery (pH <7 and base deficit >12 mmol per L)
2. Early onset of severe or moderate neonatal encephalopathy in infants born at 34 or more weeks of gestation
3. CP of the spastic quadriplegic or dyskinetic type
4. Exclusion of other identifiable etiologies such as trauma, coagulation disorders, infectious conditions, or genetic disorders

Criteria That Collectively Suggest an Intrapartum Timing (within close proximity to labor and delivery, e.g., 0 to 48 hours) But Are Nonspecific to Asphyxial Insults

1. A sentinel (signal) hypoxic event occurring immediately before or during labor
2. A sudden or sustained fetal bradycardia or the absence of FHR variability in the presence of persistent, late, or variable decelerations, usually after a hypoxic sentinel event when the pattern was previously normal
3. Apgar scores of 0 to 3 beyond 5 minutes
4. Onset of multisystem involvement within 72 hours of birth
5. Early imaging study showing evidence of acute nonfocal cerebral abnormality

If the damaging hypoxic insult occurred prior to labor or during labor with full recovery from the hypoxic period, these criteria may not apply. It should also be noted that hypoxia and a fetal inflammatory insult may act synergistically.

THE IMPACT OF INTRAPARTUM ELECTRONIC FETAL MONITORING

Early studies of EFM indicated a correlation between FHR patterns and fetal pH, Apgar scores, and intrapartum death. Since the clinical introduction of intrapartum electronic FHR monitoring in the late 1960s and early 1970s, there have been a large number of both retrospective and prospective nonrandomized studies (83–95). Initially, the nonrandomized, largely retrospective studies indicated a decrease in fetal and neonatal deaths with electronic intrapartum FHR monitoring compared with intermittent auscultation, as was done in the early 1970s. Some studies showed better outcome in high-risk EFM fetuses than in low-risk fetuses monitored by intermittent auscultation. At that time, the intrapartum period was regarded as an especially vulnerable time for the fetus, and it was generally believed that most cases of CP were caused by intrapartum asphyxia.

The first randomized prospective trial of intrapartum electronic FHR monitoring was done by Haverkamp et al. (96), and much to the surprise of many, when compared to a one-on-one nurse listening every 15 minutes in the first stage of labor and every 5 minutes in the second stage of labor, outcomes including fetal and neonatal death, Apgar scores, and cord pH values were not different, with excellent results in both groups. The incidence of cesarean birth, however, was significantly increased in the EFM group. A follow-up study by Haverkamp (97), using fetal scalp blood pH before operative intervention, revealed that the excess cesarean birth rate decreased. Since then, there have been a number of prospectively randomized trials that have not shown any advantage to intrapartum EFM over intermittent auscultation with a one-on-one nurse listening every 15 minutes in the first stage of labor and every 5 minutes in the second stage of labor (98–102). One randomized prospective trial by Vintzileos et al. (103), done in Greece, suggested benefit to intrapartum EFM with no hypoxic perinatal deaths and nine per thousand in the intermittent auscultation group. This compared to .4 per thousand in the combined auscultation groups from the other prospectively randomized trials, suggesting either a different patient population or different auscultation techniques.

When these randomized prospective trials were completed, long-term neurologic outcome was evaluated in some of the study patients. In the Dublin study by McDonald and Grant with excellent follow-up, while there were more neonatal seizures in the auscultation group, the incidence of CP was not different between groups (104). A randomized prospective trial was then done by Luthy et al. (105) in preterm fetuses weighing between 700 and 1,750 g, and again, no difference in immediate outcome measures was noted. A 3-year follow-up study on these infants was done by Shy et al. (106), and interestingly, the EFM group actually had more cases of CP. This could have been due to the study protocol that required documentation of acidotic fetal scalp pH before intervention in the EFM group, resulting in longer delay to delivery after recognition of the abnormal pattern than in the auscultation group where no pH documentation was required.

The increased cesarean birth rate seen in some of the randomized trials with EFM without apparent benefit continues to be a concern. Because a high percentage of abnormal FHR strips are not associated with poor outcomes or metabolic acidemia, a technique that will safely reduce the cesarean birth rate among patients with FHR patterns that are concerning would be highly desirable. Fetal scalp blood pH sampling was such a technique, but because of the technical difficulty of this method, it has been largely abandoned. Fetal pulse oximetry was a promising technique but for reasons described in Chapter 9, it has been withdrawn from the market. Fetal ST segment analysis is discussed later in this book, and it appears to be promising as a secondary technique to aid in the management of questionable FHR patterns.

There have been no trials comparing intrapartum EFM to no monitoring. Another way to look at intrapartum EFM

is to point out that it is as good as having a one-on-one nurse listening every 15 minutes in the first stage of labor and every 5 minutes in the second stage of labor. Clearly, most busy labor and delivery units cannot provide that type of coverage for all laboring patients. It is the knowledge gained from EFM that is used to do intermittent auscultation, and, in fact, when auscultation is concerning, electronic monitoring is virtually always used before an intervention decision is made.

Thus, today we are left with a technique that clearly detects fetal hypoxia even in its earliest stages, but it appears to show no better outcome than intermittent auscultation with a one-on-one nurse listening every 15 minutes in the first stage of labor and every 5 minutes in the second stage. This then leaves us with some questions. Could most fetal asphyxia occur before labor, resulting in an abnormal pattern from the time of admission and a good correlation with outcome but no benefit from intervention? With sudden onset acute intrapartum hypoxia, perhaps fetal damage or death can occur so rapidly that even rapid intervention is not sufficient. Finally, perhaps neurologically abnormal fetuses are more likely to become hypoxic during labor, resulting in FHR patterns that detect the hypoxia but provide no benefit from intervention. Clearly, however, acute intrapartum asphyxia can result in fetal death or later neurologic damage, and in the fetus with a previously normal FHR pattern, acute damaging hypoxia with acidemia will be detected by FHR change, and the potential for intervention with prevention of death or damage exists.

The Effect of Electronic Fetal Monitoring and Dedicated Auscultation on Intrapartum Fetal Mortality

While questions remain regarding the relative value of continuous intrapartum EFM versus intermittent auscultation, it is clear that dedicated electronic or auscultatory fetal monitoring during the intrapartum period reduces the intrapartum fetal death rate when compared to no dedicated auscultation during labor. Intrapartum fetal death is a clear endpoint, which, prior to the adoption of intrapartum EFM, occurred at a rate between one and four per 1,000 in most obstetric populations. For this reason, a rather large study population would be required to determine the effect of EFM on the intrapartum death rate when compared to intensive auscultation as occurred in the randomized trials. However, it is clear that since the adoption of EFM and more intensive auscultation for routine fetal evaluation in labor, intrapartum fetal deaths are now relatively rare events. The first clinical studies were retrospective and noncontrolled. These studies included over 135,000 patients, and approximately one-third of these patients were electronically monitored (85–95). The intrapartum death rate in these studies was 1.76 per 1,000 in patients monitored during labor with auscultation and .54 per 1,000 in patients monitored with EFM (P <.001). The intrapartum fetal death ratio calculates to 3.26:1 for auscultated versus electronically monitored patients (Table 3.4). These data were restricted to infants

TABLE 3.4 Intrapartum fetal death and EFM: nonrandomized trials

Primary author	Year	No EFM	IFD	EFM	IFD	Ratio
Chan et al. (85)	1973	5,427	17	1,162	2	
Kelly and Kulkarni (86)	1973	17,000	15	150	0	
Tutera and Newman (87)	1975	6,179	37	608	1	
Edington et al. (88)	1975	991	4	1,024	0	
Koh et al. (89)	1975	1,161	4	1,080	5	
Shenker et al. (90)	1975	11,599	14	1,950	1	
Lee and Baggish (91)	1976	4,323	15	3,529	1	
Paul et al. (92)	1977	36,724	34	13,344	6	
Amato (93)	1977	2,981	12	4,226	1	
Johnstone et al. (94)	1978	9,099	13	7,313	3	
Hamilton et al. (95)	1978	4,353	11	4,399	1	
Total		99,842	176	38,785	21	
Rate		1.76/1,000		.54/1,000		3.26 (P <.001)
		Critical no. of subjects for P <.05 = 18,046				

EFM, electronic fetal monitoring; IFD, intrapartum fetal death.
From Antenatal Diagnosis. Report of a consensus development conference. NIH Publication no. 79-1973, Bethesda, MD, April 1979.

who weighed over 1,500 g at birth and who were corrected for congenital anomalies. These studies can certainly be criticized on several bases, including different time frames in the EFM versus the auscultated groups and no consistent protocols for the use of auscultation. In most of these studies, the electronically monitored patients were higher risk than the auscultated patients, and despite this, the intrapartum stillbirth rate was lower in the high-risk group that was electronically monitored. A recent ACOG practice bulletin no. 106 published in 2009 (107) indicated that while there is no definitive evidence that EFM reduces perinatal mortality or CP, it is still recommended on all high-risk patients.

In 2010, the ACOG issued another practice bulletin no. 116 (108) on management of intrapartum FHR tracings, which is referred to in later chapters. This publication outlines one recommended approach to management based on the three-tiered categorization of FHR tracings delineated in the 2008 NICHD statement (109).

Eight published randomized controlled trials have compared EFM to auscultation in both high- and low-risk patients and with one exception have shown no difference in intrapartum fetal death (96–103). In Vintzileos et al.'s study (103), there was a difference favoring electronically monitored patients and the high hypoxic mortality rate in the auscultated group that may not apply as discussed previously. It is significant that, with the exception of the study by Leveno et al. (102), both groups in each randomized controlled trial had a dedicated one-on-one nurse assigned to the patient and the auscultated groups were followed much more frequently than was commonly practiced in hospitals. This is substantiated by the low combined intrapartum fetal death rate in the auscultated groups: 1.05 per 1,000 (excluding the Vintzileos et al. [104] study). If the studies by Leveno et al. (102) and Vintzileos et al. (103) are eliminated, thereby including only those with one-to-one nursing providing the auscultation, the intrapartum fetal death rate drops to .4 per 1,000 (Table 3.5). Furthermore, in these studies, abnormal auscultative heart rates were often backed up by electronic FHR monitoring. It is thus evident that either EFM or dedicated auscultation will reduce the intrapartum fetal death rate compared to less stringent monitoring.

What Are the Risks of Electronic Fetal Monitoring?

There has been much written about the risk of invasive EFM and infection of the fetus and/or mother. Fetal infections have consisted almost exclusively of small scalp infections characterized by erythema and induration requiring no more than local medication and are reported in from .3% to 4.5% (110–115) of internally monitored labors.

Maternal infection in patients with internal EFM during labor has been said to be increased in several studies (96,111–113), whereas other studies have indicated no increased risk of maternal infection (85,98). When one examines the reports, it becomes obvious that infection is most pronounced if the patient is monitored and then delivered by cesarean section, as opposed to the patient who is monitored and then delivered vaginally. Scrutiny will reveal that the patient with long labor, many vaginal examinations, prolonged ruptured membranes, and obstructed labor requiring cesarean section is also the most likely patient to be internally monitored for a prolonged time. This association is probably responsible for the presumed cause-effect relationship between EFM and infection following cesarean

TABLE 3.5 Intrapartum fetal deaths: randomized prospective studies[a]

	Intermittent auscultation		Continuous EFM	
Study author	Cases	Deaths	Cases	Deaths
Haverkamp et al. (96)	241	0	242	0
Haverkamp et al. (97)	232	0	453	0
Renou et al. (98)	175	1	175	0
Kelso et al. (99)	251	0	252	0
Wood et al. (100)	432	0	445	0
McDonald et al. (101)	4,999	2	4,987	3
Totals	6,155	3	6,554	3

EFM, electronic fetal monitoring.
[a]Does not include study by Leveno et al. (102), which used selective EFM in the control group and Vintzileos et al. (103), where the hypoxic death rate in the auscultated group was an outlier.

section. Gibbs et al. (114) looked at all associated factors with postcesarean infection and, using multivariate analysis, found that internal monitoring had little or no effect on infection. In medical legal cases, poor-quality external fetal monitoring is a common deviation from the standard of care that results in plaintiff decisions. The reluctance to use a fetal scalp electrode and/or intrauterine pressure monitoring device contributes to this problem.

Effect of Electronic Fetal Monitoring on the Cesarean Section Rate

Perhaps the most discussed risk of EFM is the increase in the cesarean section rate that some have attributed to EFM (Table 3.6). Determining the exact contribution, if any, that EFM makes to the overall cesarean section rate is complicated by the fact that many other practices, such as routine cesarean section for breech presentations and abandoning midforceps, have changed during the same period that EFM has been on the increase. Five of the seven randomized controlled trials have shown an increase in the cesarean section rate in the EFM group.

Clearly, there are patients who are allowed to labor because of the fetal monitor, whereas before EFM, they would not have even been given a trial of labor. In our practice, this has been best demonstrated in diabetics, patients with clinical abruptions, elderly gravidas, and certain patients with previous intrapartum fetal death where the monitor reassures both the patient and the physician.

Williams and Hawes (116) looked at the impact of EFM on the cesarean section rate in California during 1977. They surveyed 324,085 births and determined that about half the patients in California had electronically monitored labors. They determined the expected perinatal mortality rates for each hospital based on the risk of the population served. They then determined those medical care factors that favorably or unfavorably affected those mortality rates. The most significant factors that favorably affected perinatal mortality were a high cesarean section rate and a high incidence of fetal monitoring. Other factors favorable to an improved perinatal outcome included a neonatal intensive care unit in the hospital, delivery by a trained obstetrician, a perinatal morbidity committee in the hospital, and occurrence of the birth in a nonprofit community hospital with a teaching program. The data, which include a large number of deliveries over a 1-year period from all types of hospitals, suggest that the overall impact of high technology with its probably higher rate of intervention would seem to be justified on the basis of improved perinatal mortality. However, since 1977, the cesarean section rate has more than doubled, and it has increased by 50% since 1996. It is hard to say this rise has been associated with additional benefit (117).

TABLE 3.6 Impact of EFM on cesarean section rates: randomized prospective studies[a]

Study author	C-sections (%)	
	Intermittent auscultation	Continuous EFM
Haverkamp et al. (96)	7	17
Haverkamp et al. (97)	6	11
Renou et al. (98)	14	22
Kelso et al. (99)	4	10
Wood et al. (100)	2.1	4
McDonald et al. (101)	2.2	2.4

C-sections, cesarean sections; EFM, electronic fetal monitoring.
[a]Does not include study by Leveno et al. (102), which used selective EFM in the control group.

THE IMPACT OF ANTEPARTUM FETAL HEART RATE MONITORING

There have been several randomized prospective trials comparing primary antepartum fetal surveillance using the nonstress test to no nonstress test surveillance, which have shown no benefit to this form of testing alone (118–121). None of these trials involved long-term follow-up. There is only one prospective randomized trial of antepartum surveillance that showed benefit. This was Neldam's study of fetal movement counting that showed a decrease in antepartum fetal deaths in patients instructed in fetal movement counting compared with patients with no fetal movement counting instructions (122). A 1983 study by Beischer et al. (123) did show that when a nonreactive positive spontaneous contraction stress was found, there was a 28% perinatal mortality rate, and 27% of surviving infants were found to have a neurologic handicap. Manning looked retrospectively at patients studied with biophysical profile surveillance and found fewer cases of CP when these cases were compared with patients not monitored with biophysical profiles (1.33 vs. 4.74 per 1,000) (124,125). Dayal, Manning et al. (126) reported that among 86,955 patients studied with the biophysical profile fetal-maternal hemorrhage was the most common cause of a false negative test. In August of 2007, the NICHD convened a workshop titled Antenatal Testing: A Reevaluation (127). A summary based on this workshop was published in Seminars in Perinatology in 2008, which pointed out the possible benefits and the problems with antenatal testing. The conclusion was that further research was indicated regarding

- The epidemiology of antepartum stillbirth and neurologic injury
- Normal fetal physiology and fetal responses to intrauterine insults

The technology and utility of existing and emerging fetal assessment methods
Maternal and fetal indications for antenatal testing
Cost and benefits of antenatal testing

Certainly, the potential for benefit of antepartum surveillance would seem high because a majority of stillbirths occur before labor, and even those fetuses with intrapartum hypoxia, more often than not, have evidence of antepartum risk factors. However, because of the high incidence of normal outcome in patients with abnormal antepartum testing, the predictive value of antepartum testing remains poor.

SUMMARY

Intrapartum fetal hypoxemia that progresses to metabolic acidemia is a clear although infrequent cause of fetal mortality and morbidity that may lead to long-term neurologic damage. Electronic fetal monitoring and dedicated intrapartum auscultation decrease the intrapartum fetal death rate when compared to practices before the introduction of either technique. Most cases of neonatal encephalopathy and CP are attributed to causes other than intrapartum hypoxia alone. Electronic FHR monitoring appears to be associated with an increased cesarean section rate in some settings. Our current understanding of fetal monitoring patterns shows substantial agreement with respect to patterns consistent with normal fetal oxygenation and with respect to patterns consistent with ongoing fetal hypoxia and acidosis. There is, however, much disagreement with respect to patterns in between.(128–130)

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CHAPTER 4

Instrumentation and Artifact Detection Including Fetal Arrhythmias

Instrumentation and Artifact Detection

INSTRUMENTATION

For the obstetrician or obstetric nurse to interpret fetal monitor tracings correctly, it is necessary to have some understanding of the processes involved in the acquisition and processing of data relating to fetal heart rate (FHR) and uterine activity. Electronic fetal monitors are designed to interpret accurately in most situations, but there are times when their output can be misleading unless the instruments' limitations are understood. Most errors we see in FHR interpretation are related to the quality of the data acquisition and presentation, and, for this reason, an understanding of this chapter is critical for the clinician using electronic fetal monitoring in the treatment of obstetric patients.

FETAL HEART RATE DERIVED BY DIRECT (INTERNAL) FETAL ELECTROCARDIOGRAPHY

The FHR monitor acquires, processes, and displays an electronic signal. To understand the significance of the FHR display, it is important to understand what the monitor can and cannot count. FHR tracings from a fetal scalp electrode (FSE) are obtained by measuring the interval between consecutive fetal R waves. Therefore, the fetal electrocardiogram (ECG) signal provides the clinician with a measure of the electrical activity of the fetal heart. It does not necessarily represent mechanical activity. The fetal ECG signal is acquired through a bipolar electrode that penetrates the skin of the fetal scalp (first pole) and that has a second conductor residing in the secretions of the maternal vagina (second pole). It is believed that the circuit is completed through the fetal umbilical cord, placenta, and the maternal circulation and that the potential difference (voltage) being measured is between the two poles. The original electrode was a modified skin clip, but now a spiral electrode is used.

The raw fetal ECG signal is amplified and fed into a beat-to-beat cardiotachometer (Fig. 4.1). The amplifier uses an automatic gain control circuit to boost the fetal ECG signal to a predetermined voltage before electronic counting. All electronic signals are equally strengthened noise as well as fetal ECG. Once the fetal ECG signal has been amplified, it is filtered, and the interval between R-wave peaks is measured. The interval between R waves is processed to a rate in beats per minute (BPM) by computing the reciprocal of the interval. With internal FHR monitors, a new rate is set each time an R wave is detected; as each new wave arrives, the rate is recalculated from the reciprocal of the previous R-R interval. The result of this rate calculation is the plotting of a series of square waves (Fig. 4.2). The minute rate differences between beats are referred to as beat-to-beat or short-term variability and can only be appreciated when the FHR is computed instantaneously by means of a beat-to-beat cardiotachometer. FHR changes with a cyclicity of 3 to 5 per minute are referred to as long-term variability, and those occurring in response to uterine contractions or fetal movement are referred to as periodic. Long-term variability and periodic changes can be detected with Doppler systems, but true short-term variability can be measured only with direct or abdominal fetal ECG systems. The newer fetal monitors have advanced technology, utilizing autocorrelation techniques, allowing for Doppler-derived signal data of significantly improved quality such that, although not exact, the FHR recording is very close to that obtained from direct fetal ECG and visually approximates true short-term variability in many patients.

Most fetal ECG systems will not record R-R intervals less than 250 milliseconds, which corresponds to a rate of 240 BPM. If the FHR exceeds 240 BPM, not even a direct fetal ECG system will count every beat and may halve or not print such rates. This occurs only with fetal supraventricular tachyarrhythmias (paroxysmal atrial tachycardia, atrial fibrillation, or atrial flutter), intermittent premature atrial contractions (PACs), or premature ventricular contractions (PVCs) (Fig. 4.3).

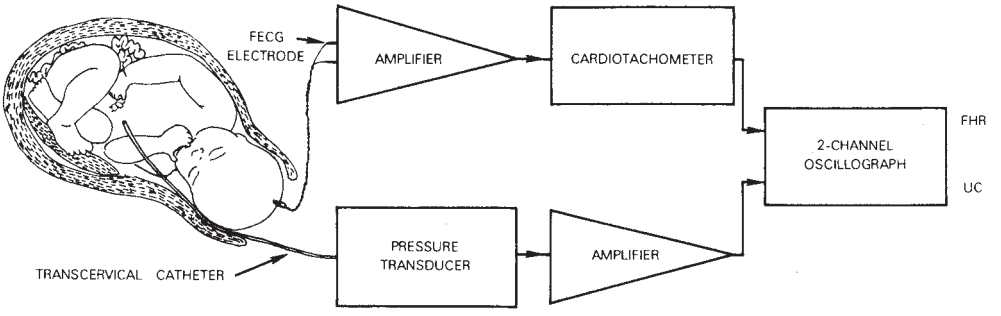

Figure 4.1. Schematic diagram of a direct fetal monitoring system. The fetal heart rate is obtained from a fetal scalp electrode and counted in a cardiotachometer. The actual uterine pressure is recorded directly from a transcervical intrauterine catheter. (FHR, fetal heart rate; UC, uterine contraction.)

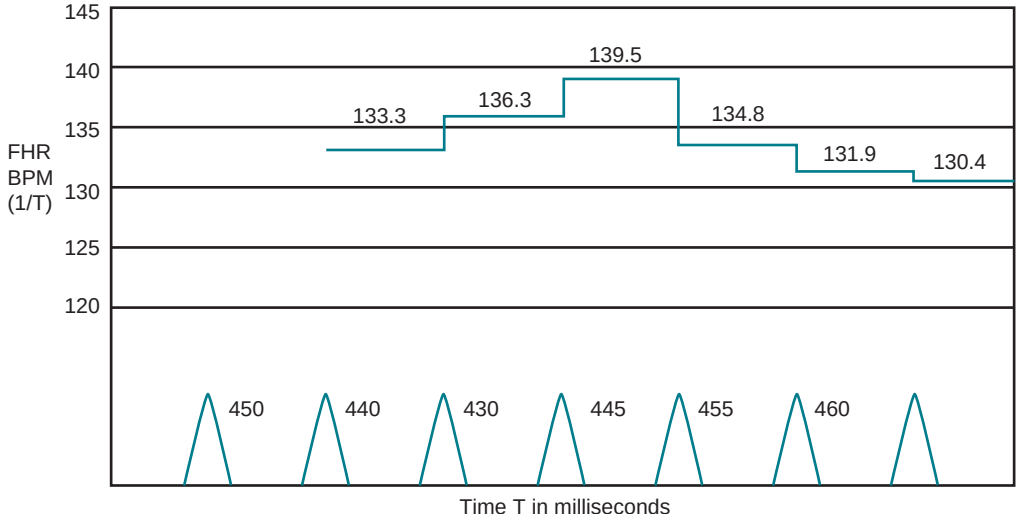

Figure 4.2. This schematic represents an enlarged segment of the fetal heart rate tracing. A new rate is set with the arrival of each R wave in the fetal electrocardiogram. This generates a series of square waves that indicate beat-to-beat changes.

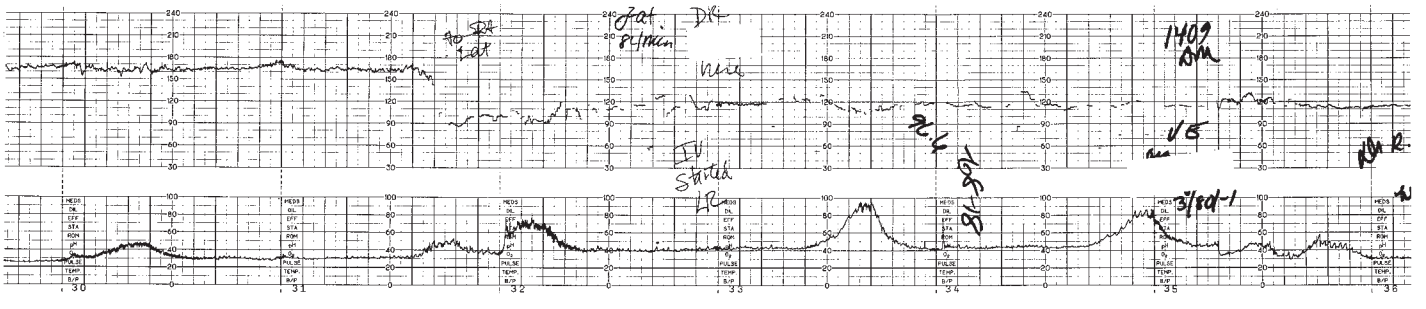

Figure 4.3. This is an example of a change in the atrial pacemaker. The fetus is known to have multiple cardiac abnormalities and presented in supraventricular tachycardia with nonimmune hydrops. Cardioversion was achieved successfully with digoxin and quinidine. This tracing is from early labor.

An additional instance that may cause confusion is the patient with a cardiac pacemaker. If the transmitted maternal pacemaker pulse is at a higher voltage than the fetal R wave, the scalp electrode may record the pacemaker signal (1,2).

In the absence of the fetal ECG signal, such as with a dead fetus, there will usually be no tracing. However, depending on the monitor and the existing maternal R wave, amplification of the incoming signal may continue until, on occasion, counting of the maternal heart rate (MHR) from the scalp of the dead fetus results (Fig. 4.4) (3,4). This can obviously lead to confusion. When there is any doubt, fetal demise should be confirmed with real-time ultrasound or comparison to maternal ECG or pulse oximeter signal, and in all instances of monitoring, there needs to be documentation of maternal pulse rate as different from the FHR.

FETAL HEART RATE DERIVED BY INDIRECT (EXTERNAL) DOPPLER ULTRASOUND

In the antepartum period, and often during the intrapartum period, it is neither feasible nor always necessary to use the direct fetal ECG signal to record the FHR. External monitoring using various biophysical modalities has

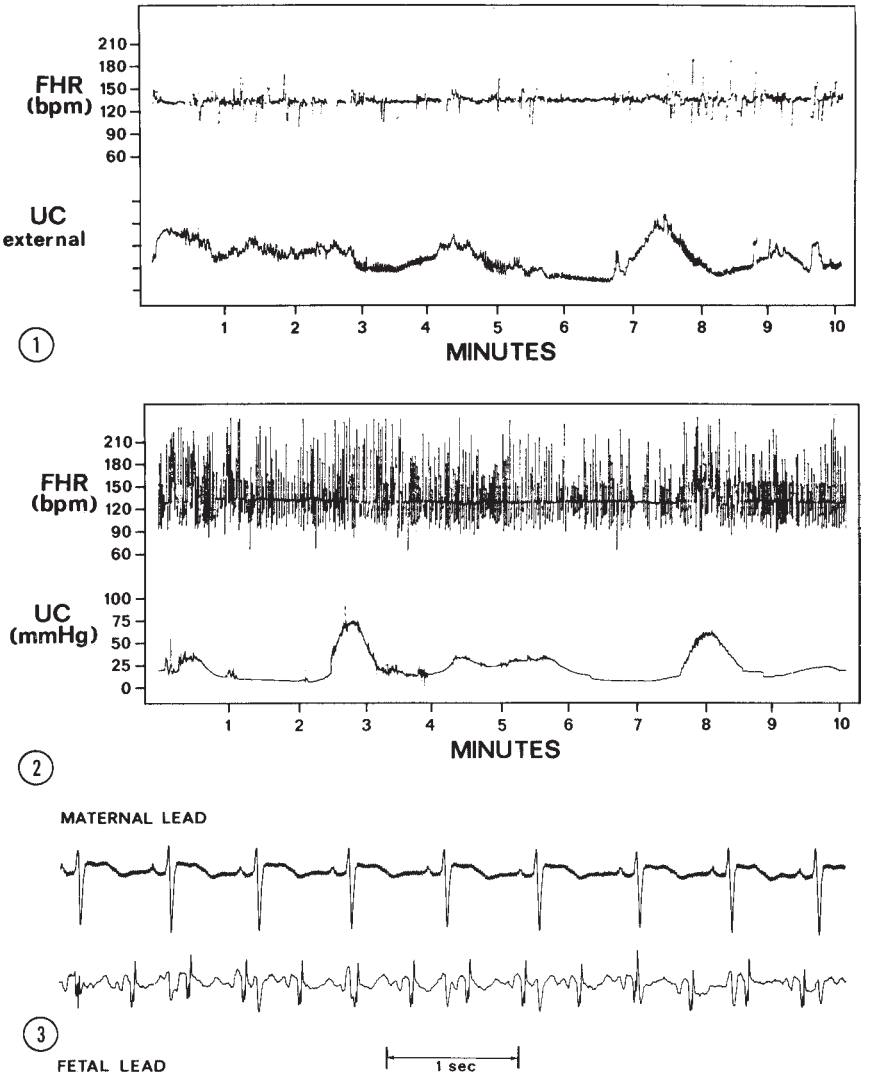

Figure 4.4. The **upper panel** shows the heart rate from a fetal scalp electrode (FHR) and maternal leads (MHR) with a dead fetus. Note the two rates are identical in detail. The **lower panel** shows the fetal scalp lead and the maternal lead electrocardiogram (ECG) tracing indicating that the dead fetus is transmitting the maternal ECG to the fetal lead. (From Klapholz H, Schifrin BS, Myrick R et al.: Role of maternal artifact in fetal heart rate pattern interpretation. *Obstet Gynecol* 44:373, 1974, with permission.)

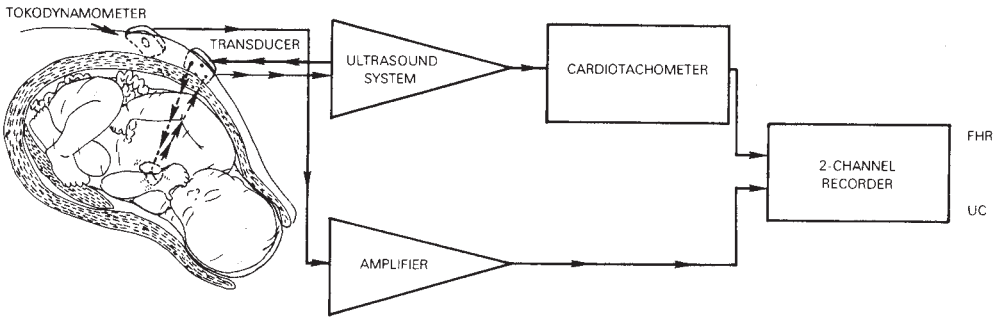

Figure 4.5. Schematic diagram of an indirect fetal monitoring system. The fetal ultrasonogram is obtained from an abdominal wall transducer, conditioned, and then counted by a cardiotachometer. The semiquantitative uterine activity is measured by an external tocodynamometer. (UC, uterine contraction.)

evolved to a point where it currently represents the most frequent form of FHR monitoring. Doppler ultrasound is the method most commonly used to indirectly record FHR (Fig. 4.5).

The principles underlying the use of Doppler FHR monitoring are described.

Ultrasonic signals can penetrate human tissue. When the transmitted ultrasonic beam encounters an interface of increased density, a portion of the signal is reflected. The angle of reflection varies according to the angle of incidence of the beam. A portion of the signal will be transmitted to the next interface. If the interface is moving, the reflected signal undergoes a frequency change (Doppler shift). The frequency increases if the reflecting interface is moving toward the signal source and decreases if the reflecting interface is moving away from the signal source. An example commonly used to describe the Doppler shift is the audible change in pitch (frequency) noticed by a stationary observer of the whistle from a rapidly moving train. As the train approaches, the whistle gets both louder and higher in frequency. As the train passes and moves away, both loudness and pitch rapidly decline.

The fetal monitor Doppler transducer contains a transmitter, or signal source, and receiver. With all of the first-generation fetal monitors and many second-generation monitors, the signal is transmitted and the reflected signals received continuously by multiple crystals contained in the transducer. A transducer innovation employed by second-generation monitors is pulsed Doppler. The pulsed Doppler transducer alternates the emission of ultrasound waves with the reception of the reflected waves, resulting in a decrease in both the amount and time of exposure of the fetus to ultrasound energy. Ultrasound waves of sufficient intensity will generate heat. Intensities of less than 100 mW/cm^2, regardless of the length of exposure, generate no heat. The amount of energy generated by fetal monitors is only a small fraction of this, with the continuous ultrasound transducers generating intensities of 5 to 12 mW/cm^2 and pulsed ultrasound transducers generating 1.5 to 5 mW/cm^2.

As long as the reflecting interfaces are not in motion, the reflected signal has the same frequency as the transmitted signal. However, if the reflecting interface is the surface of a moving organ such as the fetal heart, there will be a frequency change (Doppler shift) in the reflected signal. The electronic circuitry of the fetal monitor senses this frequency change and converts it to an electronic signal. This signal can then be used as a marker of the fetal heart beat as well as for the creation of fetal heart sounds produced by the monitor. This is the sound that is heard using a Doppler device. A similar shift is created if the Doppler signal is being reflected by any movement such as fetal blood, maternal vessels, or fetal movement. It is important to understand that with Doppler technology, it is not the actual fetal heart being heard but rather a sound that is created by the device in response to frequency changes generated by a moving interface.

This biphasic signal is immersed in noise created by fetal movements, arterial blood flow, maternal movements, and random muscle contractions. The signal actually received is a composite consisting of bursts with various amplitudes and frequencies. In addition, the actual signal created by the fetal cardiac motion is greatly affected by the position and movement of the transducer with respect to the fetus. With older monitors, the quality of the Doppler-created FHR tracing is directly related to the orientation of the signal to the fetal heart, the amount of fetal movement, and the degree of constant attention by nursing personnel of maintaining an adequate signal while caring for the patient. One potential source of error occurs when the Doppler signal is actually maternal and not FHR (Fig. 4.6). The maternal pulse should be checked to confirm correct recording of FHR at the initiation of fetal monitoring, with any switching of FHR modes or with any abrupt decrease in FHR,

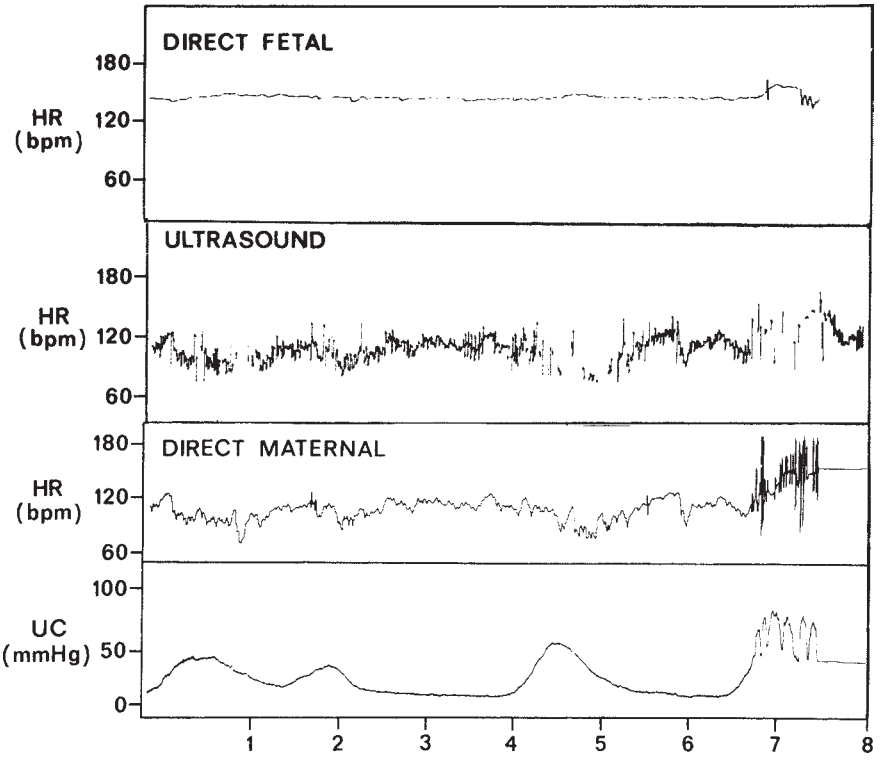

Figure 4.6. Simultaneous recording of heart rate (HR) from the direct fetal scalp electrode (**upper tracing**), abdominal Doppler (**middle tracing**), and direct maternal (**lower tracing**). Note that the abdominal Doppler signal is recording the maternal heart rate from a maternal vessel in the abdomen. (UC, uterine contraction.) (From Klapholz H, Schifrin BS, Myrick R, et al.: Role of maternal artifact in fetal heart rate pattern interpretation. *Obstet Gynecol* 44:373, 1974, with permission.)

especially in a setting of noncontinuous tracings. Newer monitors sometimes do not produce noncontinuous tracings when the signal switches from fetal to maternal, making the transition more difficult to identify (see section on signal ambiguity).

With the evolution of autocorrelation in many of the newer monitors, great advances have been made in both signal quality and continuity. While new wide-beam ultrasound transducers decrease signal loss due to fetal movement, they increase the chance of recording MHR (see section on signal ambiguity).

FIRST-GENERATION SIGNAL PROCESSING

Fetal monitors obtain the FHR indirectly by use of Doppler ultrasound. To produce an FHR tracing, several modulations of the reflected signal need to be used. As previously discussed, amplification and filtering of the incoming signal within certain frequencies extracts FHR signals from those produced by other moving structures. The filtered signal is converted to an electrical waveform by the transducer, and it is this waveform that is used to generate and display the FHR. Detection of fetal motion with Doppler signal is the same with both the older and newer monitors. It is the process of signal conversion to FHR that differs.

First-generation monitors calculate heart rate by electronic integration and peak detection of the returning Doppler signal. The highest point of the waveform is detected and recorded as a heart beat, even though it may not appear at the same time in each waveform. Despite various electronic logic and filtering processes, this often results in an apparent increase in short-term variability due to a false reproduction of the actual interval from one heart beat or R wave (contraction) to the next (Figs. 4.7–4.9). With signal fading, inconsistent signal shape, and changing signal peaks, the resultant FHR may be, at best, a poor reflection of the true variability of consecutive R-wave intervals. The beat-to-beat variability of the FHR calculated from the fetal ECG usually averages 1 BPM or less (5,6), but the Doppler signal using the first-generation monitor technology often displays a variability much greater than that and can be very misleading.

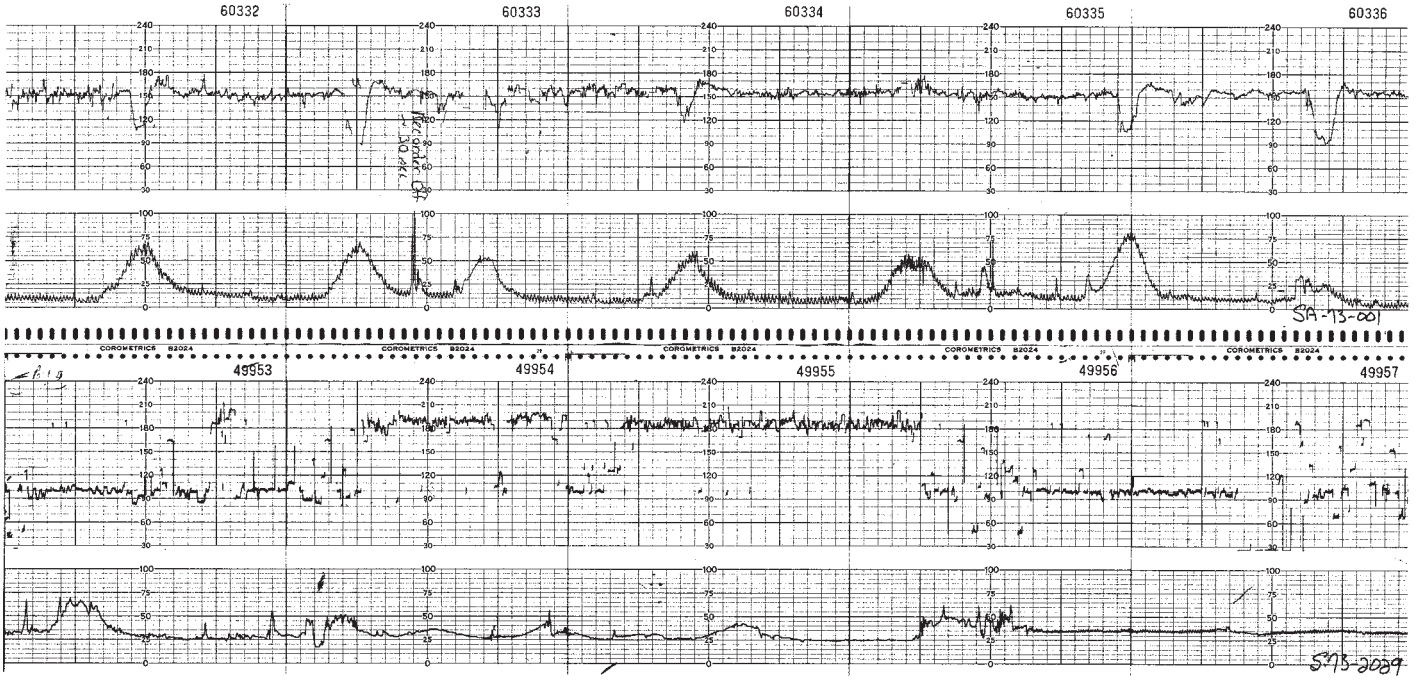

Figure 4.7. Tracing from a Doppler system showing halving of the fetal heart rate at rates over 180 beats per minute.

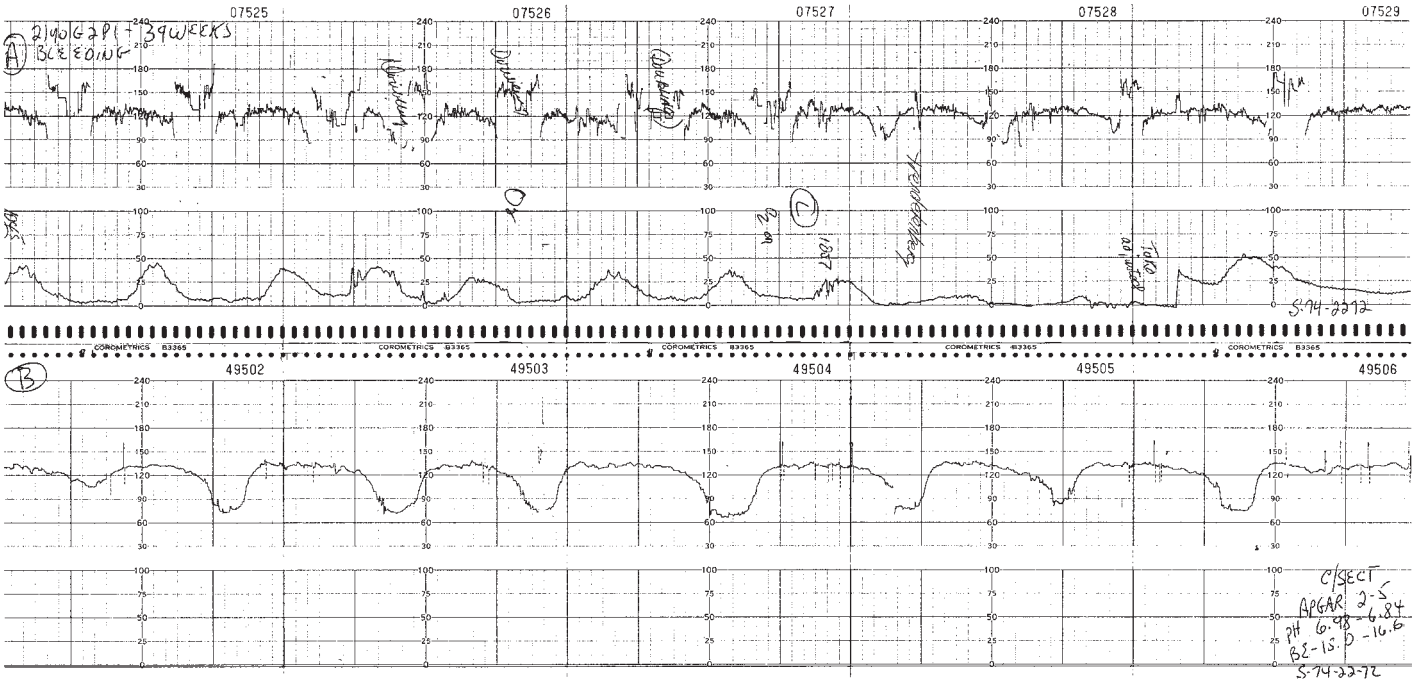

Figure 4.8. The **upper tracing** is taken from a Doppler signal source. At first glance, it appears to be a poor-quality erratic tracing, but upon closer examination, the late decelerations can be seen. The fetal heart rate (FHR) doubles whenever it goes below 90 beats per minute, putting the trough of the late decelerations above the baseline. The **lower tracing**, a continuation of the **upper tracing** after the fetal scalp electrode signal source was begun, shows the deep decelerations that were previously unrecognized because of the artifactual doubling of the FHR by the Doppler logic system.

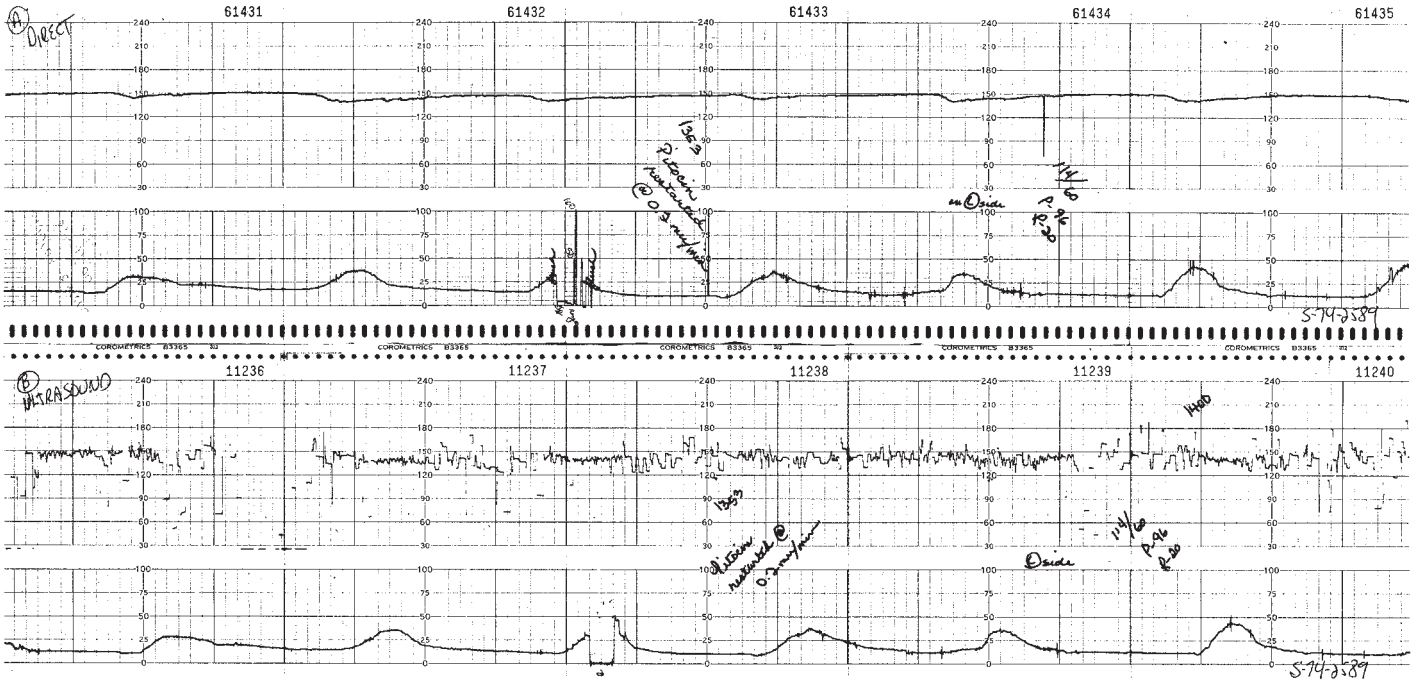

Figure 4.9. Simultaneous recordings with Doppler (**lower tracing**) and direct fetal electrocardiogram (**upper tracing**) signal sources. Note the apparent increased variability and obscuring of periodic changes on the Doppler tracing.

AUTOCORRELATION

Although not new in concept, the application of autocorrelation to FHR technology has been made possible by the introduction of high-speed microprocessor integrated circuitry. The received pattern is broken into very short second envelopes of time made up of 200 to 300 digitalized points (Fig. 4.10). With each consecutive new heart beat waveform, the microprocessor compares the digitalized points to the equivalent points in the preceding envelope. Essentially, autocorrelation is a

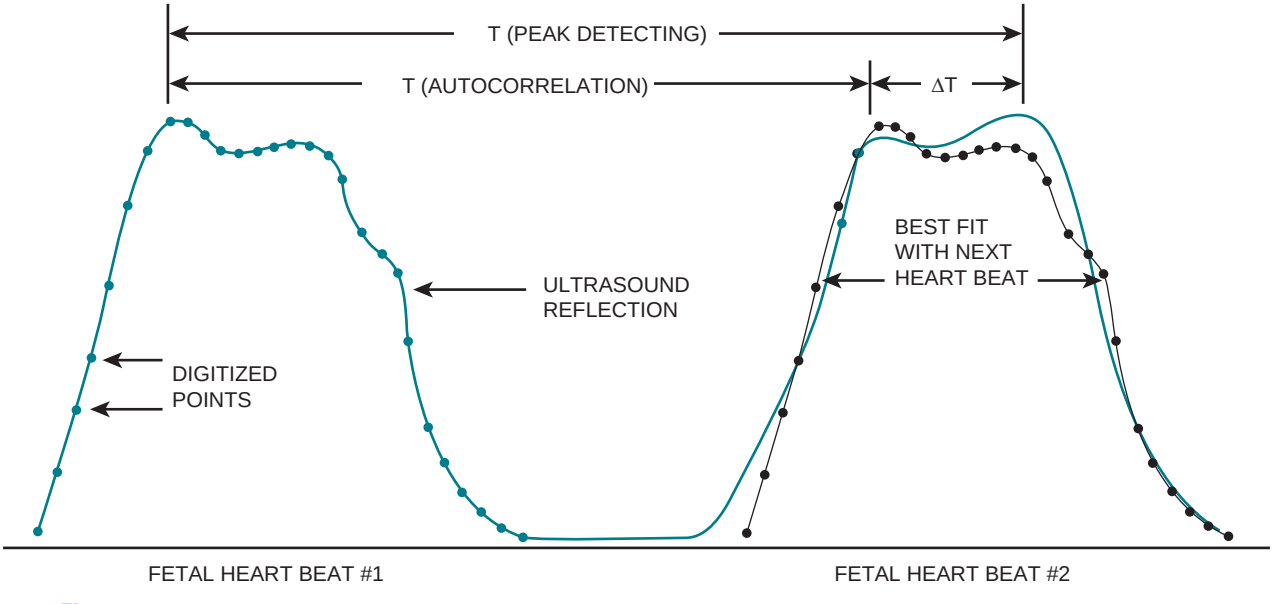

Figure 4.10. Schematic representation of consecutive fetal heart beat waveforms demonstrating autocorrelation versus peak detection. (Reproduced with permission of Hewlett Packard Corporation, Bohlinger, Germany.)

signal-processing scheme that compares the incoming signal with a time-delayed version of previous signals. Important information has a regular form repeated over time, while random noise has no such regularity. Artifactual waveforms and older points are discarded. The resultant tracing represents a point in each cardiac cycle more accurately than can be done with first-generation monitors. Varying waveform amplitude problems are, therefore, minimized, and markedly less false variability is produced (Fig. 4.11).

As with first-generation monitors, interpretation of the FHR from newer monitors using autocorrelation must be done cautiously. Despite apparent improvement in signal interpretation, autocorrelation is still not a true measure of short-term variability. The possibility for signal loss, doubling, halving, or recording of MHR or other movements must be kept in mind when reading changes in FHR monitor strips (6,7). Because autocorrelation enhances signal-to-noise levels and periodic phenomena, in the absence of fetal cardiac motion, it may produce a false signal and resultant "heart rate" (Fig. 4.12). When doubt is present, confirmation with auscultation or direct FSE must be done.

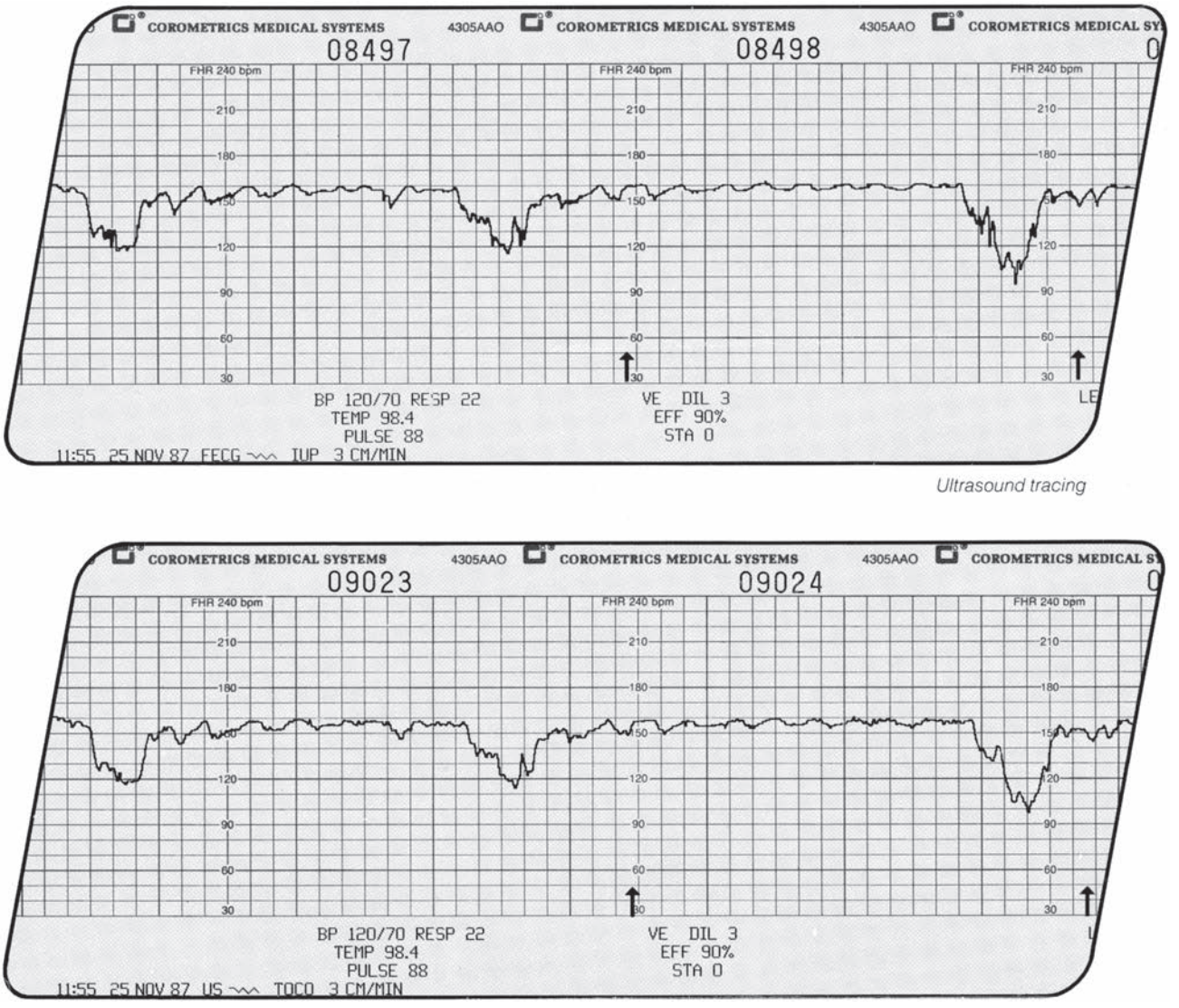

Figure 4.11. Example of simultaneous intrapartum fetal heart rate tracings comparing the external monitor with the direct fetal electrocardiogram internal monitor. Autocorrelation has improved the quality of the tracing and eliminated much of the signal loss or error present with many older monitors. (Courtesy of Corometrics Medical Systems, Wallingford, CT.)

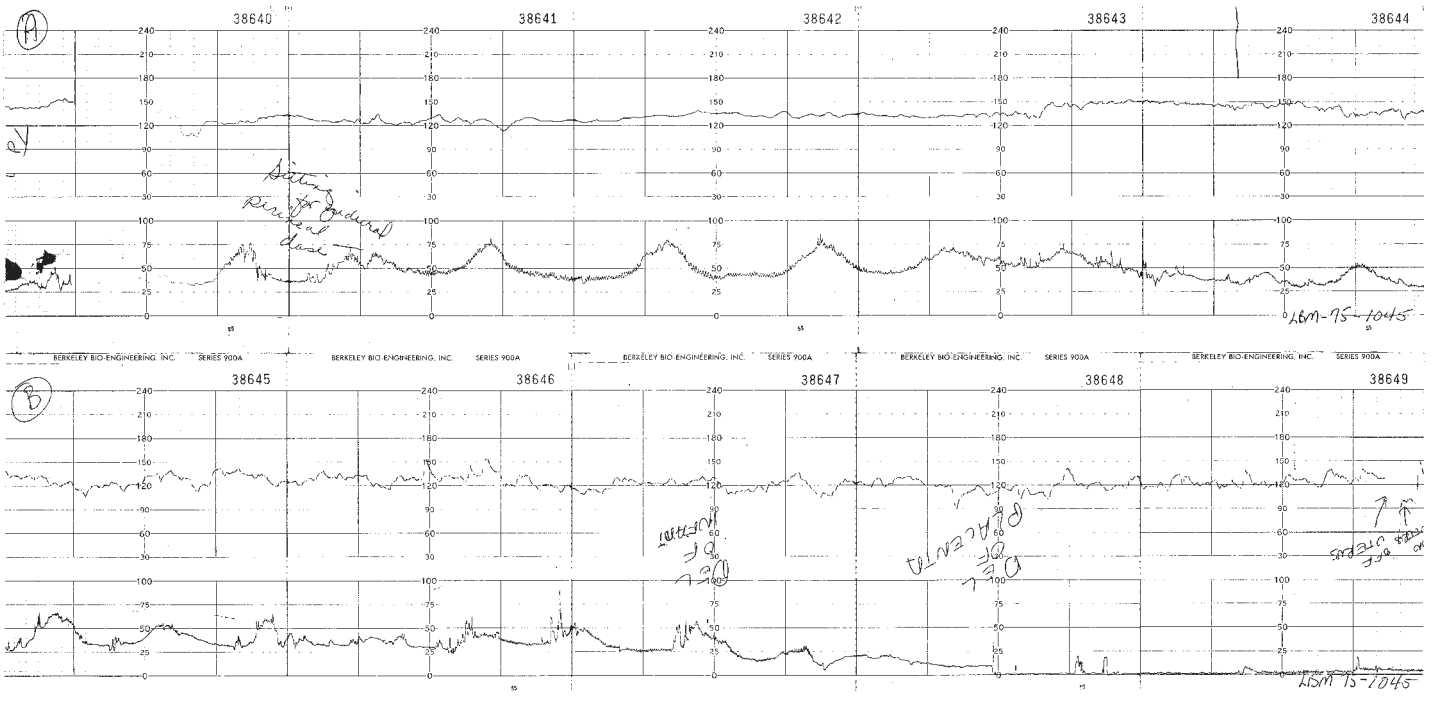

Figure 4.12. This tracing shows a Doppler record from a monitor with an excessive amount of electronic logic. Note in the **lower panel** that, even after the delivery of the baby and placenta, an apparent fetal heart rate can still be seen on the tracing.

ABDOMINAL FETAL ELECTROCARDIOGRAPHIC-DERIVED FETAL HEART RATE TRACINGS

Abdominal fetal ECG signals were first recorded by Cremer in 1906 (8). The original application of this method was to diagnose fetal life. Later, Larks and Dasgupta (9) and Hon and Hess (10) showed that the presentation of the fetus could be predicted by the polarity of the fetal QRS complex in relation to the maternal abdomen. With a breech presentation, the fetal ECG complex would be similar in polarity to that of the mother; with a cephalic presentation, the R wave would be opposite that of the maternal polarity. In addition, abdominal fetal ECG tracings could be used to diagnose twins by comparing the fetal complexes and determining two separate rate patterns among the fetal complexes. From the maternal abdomen, the fetal ECG complex is much smaller than the maternal ECG (Fig. 4.13). Often, if the electrical noise level is high, one may not be able to see a fetal ECG signal.

Due to the weakness of the fetal ECG signal before 30 weeks' gestation, the interference created by the electromyographic muscle noise of the maternal abdominal wall, and the frequency of coincidence of maternal and fetal ECG signals, abdominal ECG plays little role in modern FHR monitoring other than in arrhythmia detection.

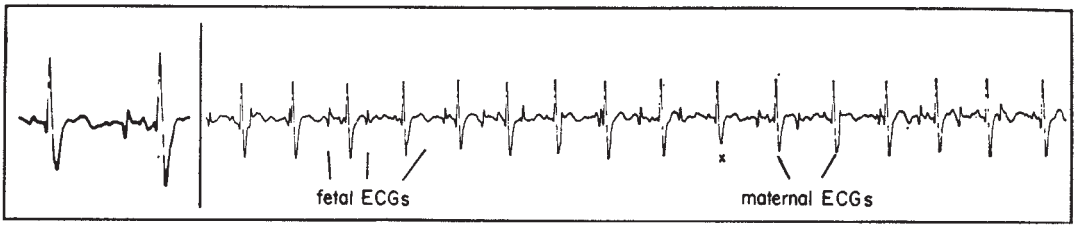

Figure 4.13. An abdominal tracing showing the large maternal electrocardiogram complexes and the clear but much smaller fetal complexes. (From Hon EH, Lee ST: *Am J Obstet Gynecol* 87:804, 1963, with permission.)

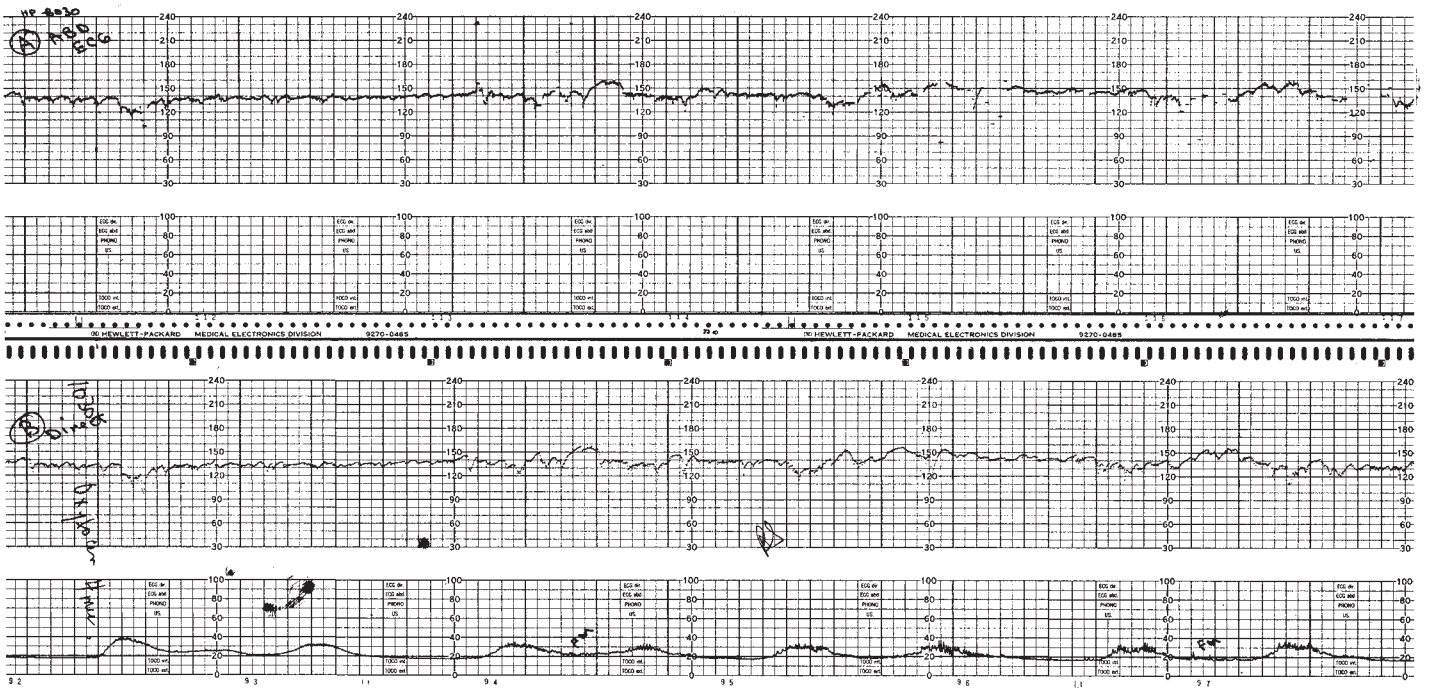

Figure 4.14. Simultaneous fetal heart rate tracings derived from abdominal electrocardiogram (ECG) (**upper panel**) and scalp ECG (**lower panel**).

However, before the introduction of autocorrelation and improved Doppler technology with the newer monitors, abdominal fetal ECG-derived tracings were the most accurate recordings of FHR signal in many patients (Fig. 4.14). Several attempts have been made, with varying degrees of success, to reliably obtain FHR variability indirectly using the abdominal fetal ECG modality (11,12). Today abdominal FECG signal source for FHR tracings are no longer used in the United States.

PHONOCARDIOGRAPHICALLY DERIVED FETAL HEART RATE

Phonocardiography was the first method used to record FHR electronically. As the fetal heart beats, closure of the valves may be detected by listening with a suitable stethoscope through the mother's abdominal wall. With ventricular systole, the closure of the atrioventricular (AV) valves produces the first heart sound. This mechanical energy may be sensed by a microphone and amplified, producing an electrical signal that may then be reconverted to sound or used to produce a phonocardiogram, an oscillographic tracing of the heart sounds. The amplified electrical signal can also be used as a counting source for an FHR monitor. The phonocardiographic signal is clearer than the Doppler signal, resulting in less artifactual "jitter." For this reason, phonocardiography historically was widely used for antepartum FHR monitoring.

The main drawback to phonocardiographically derived FHR systems is that they are extremely sensitive to ambient noise such as maternal bowel sounds, voices in the room, certain air-conditioning systems, and, especially, noise produced by any motion of the microphone or of the bed clothing against the microphone. In addition, any fetal kicking or motion produces a very loud noise that will saturate the automatic gain system on the monitor's amplifier, resulting in complete loss of recording for several seconds while waiting for the amplifier to reopen. For this reason, a manual gain control offers a great advantage when using abdominal fetal phonocardiography for recording heart rate. Also, because of the high sensitivity to ambient noise, the technique is unsatisfactory for monitoring during the active phase of labor (Fig. 4.15). The current role of phonocardiographic FHR recording is quite limited but should be considered if abdominal fetal ECG and Doppler do not produce satisfactory recordings. Today, it would have to be considered below Doppler in a ranking of preferred methods of antepartum FHR recording. Both abdominal fetal ECG and phonocardiographic FHR are rarely employed means of fetal monitoring but are of historic significance.

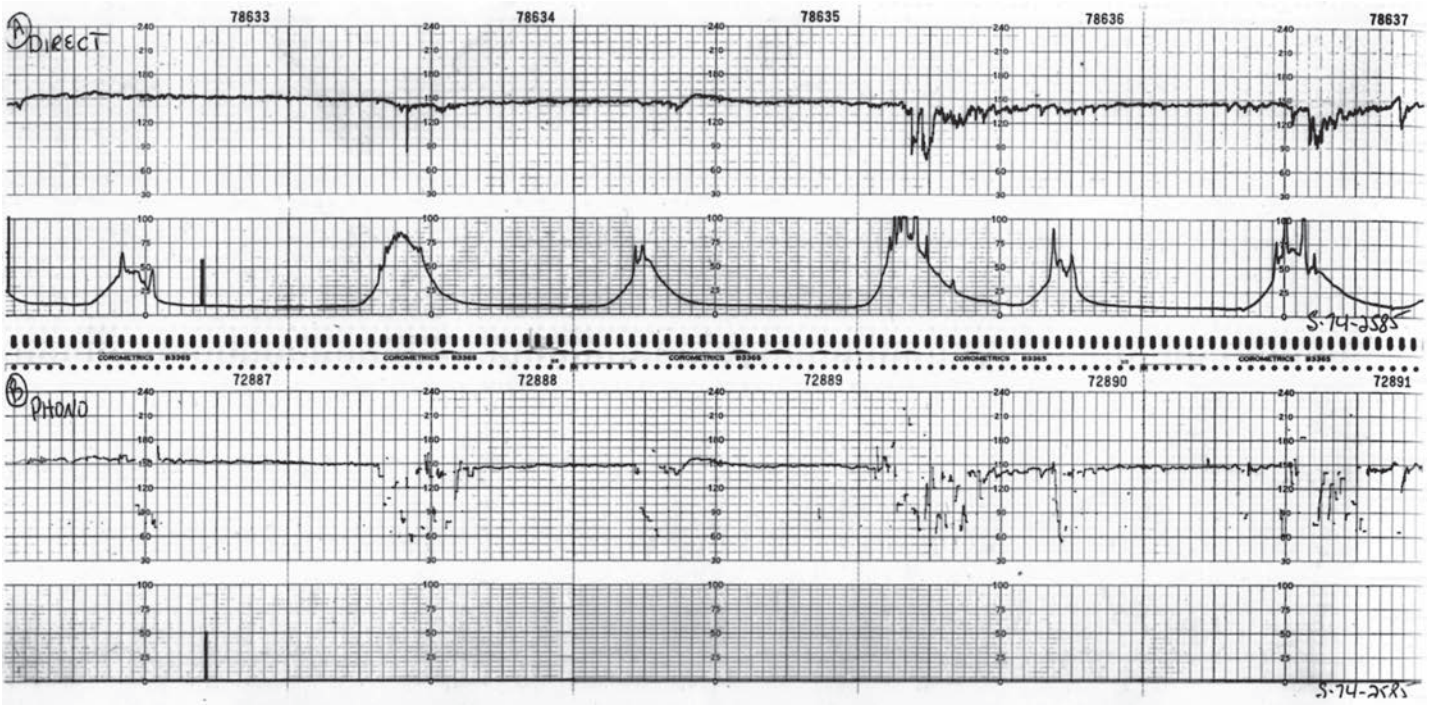

Figure 4.15. Simultaneous fetal heart rate tracings derived from phonocardiographic (**lower panel**) and scalp electrocardiogram (**upper tracing**). Note the signal loss during contractions with the phono recording.

SCALING FACTORS

The choice of vertical and horizontal scaling directly affects the appearance of the FHR and uterine contraction tracings. In the United States, the standard factors are 30 BPM/cm on the vertical scale and 3 cm/minute on the horizontal scale. In Europe, standard factors are 20 BPM/cm (vertical) and 1 or 2 cm/minute (horizontal). The European scaling factors accentuate apparent FHR variability and tend to make periodic changes appear more abrupt than American scaling factors. Although US clinicians find 1 cm/minute tracings are harder to read than the same tracings at 3 cm/minute, the slower rate of tracing is commonly used in Europe, South America, and certain centers in this country. Figure 4.16 shows how paper speed can significantly alter the appearance of a tracing.

APPLICATION OF MONITORING DEVICES

Uterine Contraction Monitors

Clinically, uterine contractions can be monitored by two techniques: external tocodynamometry or intrauterine pressure measurement. Both methods have advantages and disadvantages, and one or the other is more applicable in certain clinical situations. This section will deal with the methodology involved in the clinical application of these techniques.

Intrauterine Pressure Monitoring

The pregnant uterus is a closed, fluid-filled space. Hydrostatic pressure within the uterus should be equal at all points. Intrauterine pressure has historically been determined with the use of an open-ended, fluid-filled catheter placed through the cervix and externally attached to a strain gauge transducer. Pascal's law dictates that assuming such a monitoring system is a closed system, the baseline tone as well as the intrauterine pressure during a contraction will be transmitted directly to the external strain gauge pressure transducer. With such a system, both technical and logistic problems exist, such as catheter occlusion by solid matter, kinking or entrapment of catheter between the uterus and the fetus, as well as introduction of artifact secondary to maternal movement and catheter manipulation (13). An alternative type of intrauterine pressure device was introduced in 1987. This device has a micropressure transducer located at the tip of the catheter, which is inserted through the cervix into the uterine cavity. Such a device eliminates most of the problems associated with the fluid-filled devices used for uterine pressure monitoring. Most devices currently employed to monitor intrauterine pressure come from one of the manufacturers of these transducer-tipped catheters (see Figs. 5.3 and 5.4).

The pressure within the uterine cavity is directly proportional to the uterine wall tension and inversely proportional

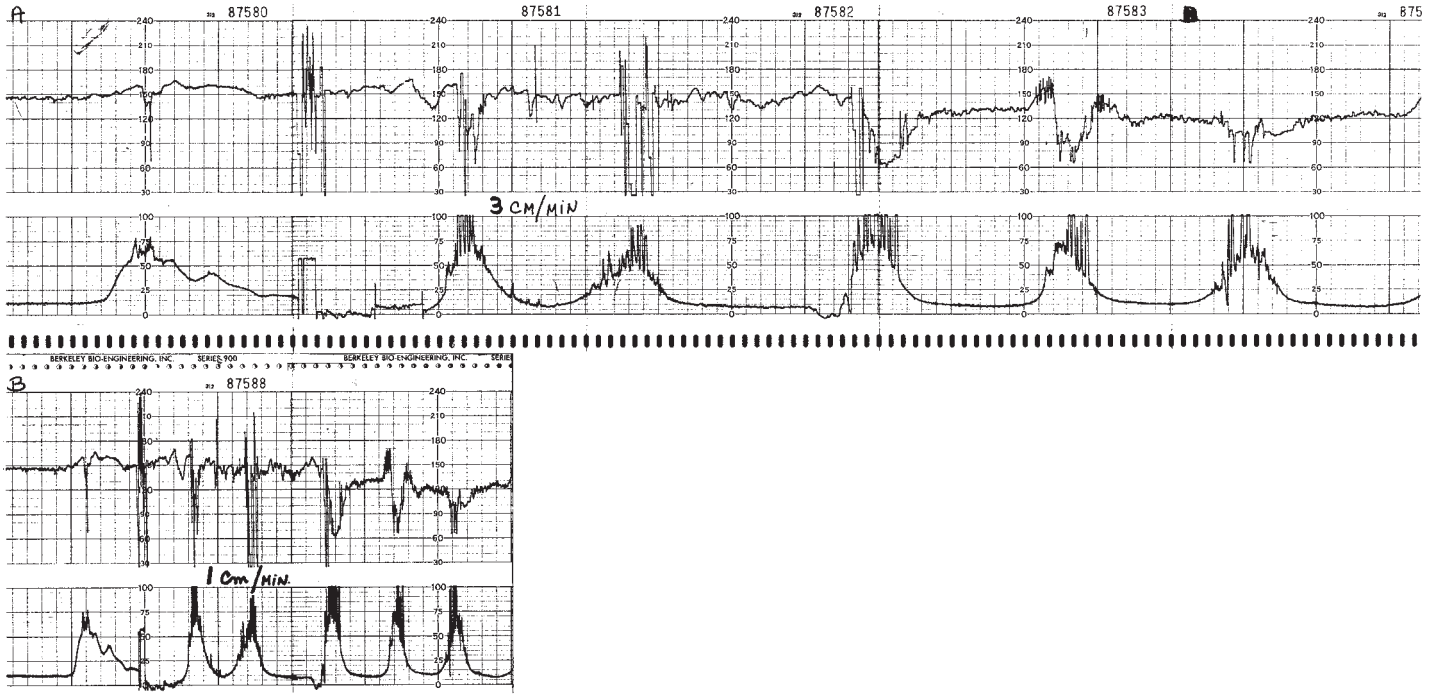

Figure 4.16. Tape-recorded fetal heart rate and uterine contraction patterns were played back simultaneously to monitors running at 3 cm/minute (**upper tracing**) and 1 cm/minute (**lower tracing**).

to the diameter of the uterus. Thus, the larger the uterus, the lower the intrauterine pressure for a given uterine wall tension. Clinical support of this concept is reflected in the fact that pressure in the nonpregnant uterus may exceed 200 mm Hg during menstrual contractions, whereas intrauterine pressure with twins may never exceed 35 or 40 mm Hg, even in active labor. The usual pressures observed in the pregnant uterus during active labor at term are in the range of 50 to 100 mm Hg at the peak of contractions, with a baseline tone of 5 to 12 mm Hg.

Insertion of the uterine pressure catheter is accomplished by introducing it, while within the sterile introducer tube, just inside the uterine cervix and next to the presenting part (Fig. 4.17). The flexible intrauterine catheter is usually advanced into the uterus quite easily, but on occasion, resistance will be met. In this situation, one should move the introducer 90 degrees in its orientation to the cervix and try again. Usually, one begins posterior to the presenting part, but the catheter can be inserted anywhere in the 360-degree circumference of the cervix. When the catheter advances easily, it should be inserted to the mark on the catheter that should be just visible at the introitus. All systems for measuring intrauterine pressure must be zeroed, which is the referencing of the pressure to 0 mm Hg while the system is open to air. Any reconnection of the patient to the monitor requires rezeroing. It is expected that there will be a baseline uterine tone, so if the monitor is registering zero or a negative number, the calibration is incorrect. Replacement of the catheter is necessary if a good recording does not result from simple manipulation.

Another modification of the intrauterine pressure catheter allows for amnioinfusion while simultaneously recording contraction strength directly (see Fig. 5.5). The accuracy of pressure monitoring is not lessened with this catheter and allows for continuous amnioinfusion without

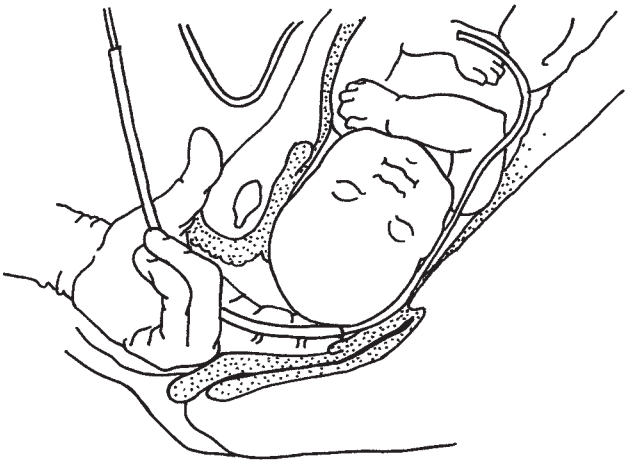

Figure 4.17. Technique of transcervical insertion of uterine catheter. Note that the introducer is only inserted about 1 cm inside the cervix.

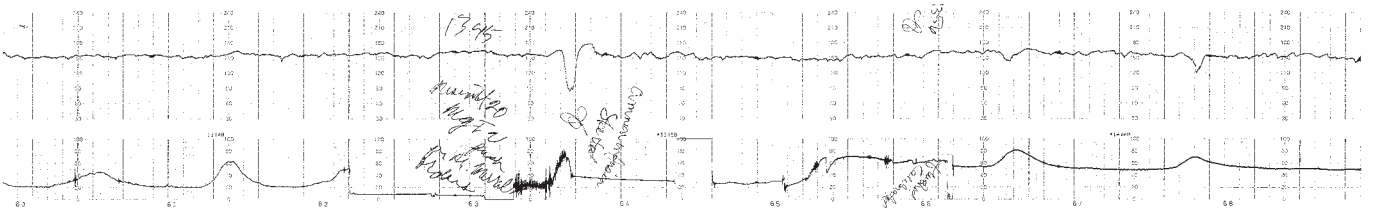

Figure 4.18. Amnioinfusion effect on uterine tone. Note the significant change in the baseline uterine tone associated with initiation of amnioinfusion. This results from the hydrostatic pressure of the infusate and must be considered in assessing the actual intrauterine pressure.

artifactual elevation of baseline uterine pressure or a need for the placement of a second catheter for infusion. If one is using a single-lumen catheter for pressure readings and simultaneous amnioinfusion, there is an elevation of the apparent baseline uterine tone (Fig. 4.18). However, this is actually the result of the hydrostatic pressure from the infusate and is readily confirmed by interrupting the infusion and confirming true baseline and peak contraction strength.

The Spiral Fetal Scalp Electrode

The most commonly used fetal electrode consists of a spiral or corkscrew-shaped device placed inside two concentric tubes, with the wires trailing through the center tube (Fig. 4.19). After clearly identifying the presenting part, the whole assembly is inserted through the vagina and cervix against the fetus. The inner tube is then rotated clockwise one full turn so that the electrode tip penetrates the fetal presenting part (Fig. 4.20). The wires are then attached to the leg plate, which is usually placed on the mother's thigh (Fig. 4.21). The leg plate is then attached to the fetal monitor.

If the signal is not adequate, efforts should be made to be certain that the electrode is attached to the fetal presenting part and that the leg plate is appropriately grounded.

Precautions that should be taken when inserting an internal electrode include the following:

1. Use sterile technique.
2. Do not attempt placement if unsure of the exact site of placement.
3. Do not place over facial structures.
4. Avoid the genitalia in breech position.
5. Never try to reinsert an electrode by twisting manually. Sometimes the spiral is stretched out, and the depth of insertion may exceed the safe depth set on nonstretched electrodes.
6. Do not rotate the spiral more than 360 degrees, as tissue injury may result.
7. Do not use in a patient who has active herpes, hepatitis, or human immunodeficiency virus infection.

Figure 4.19. Example of the fetal spiral electrode. (Tyco Healthcare-Kendall LTP, Chicopee, MA.)

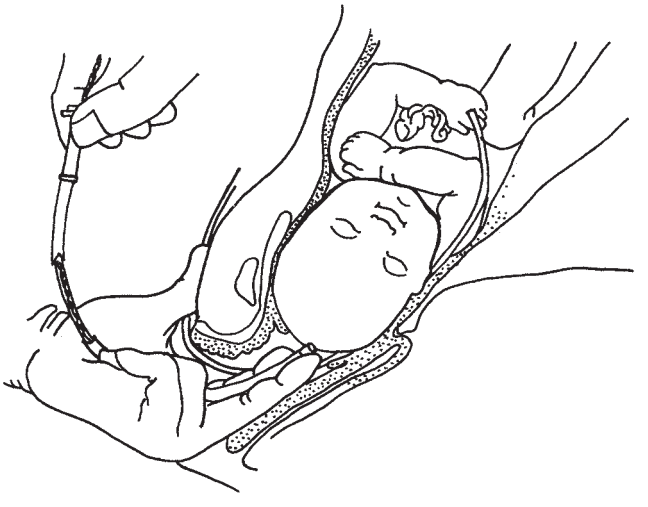

Figure 4.20. Technique for application of the spiral electrode.

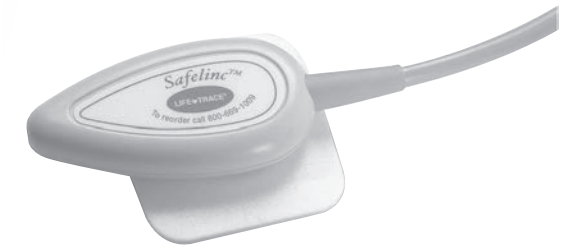

Figure 4.21. Example of leg plate that is attached to the patient's upper thigh. The scalp electrode wires connect directly to this plate. (Tyco Healthcare-Kendall LTP, Chicopee, MA.)

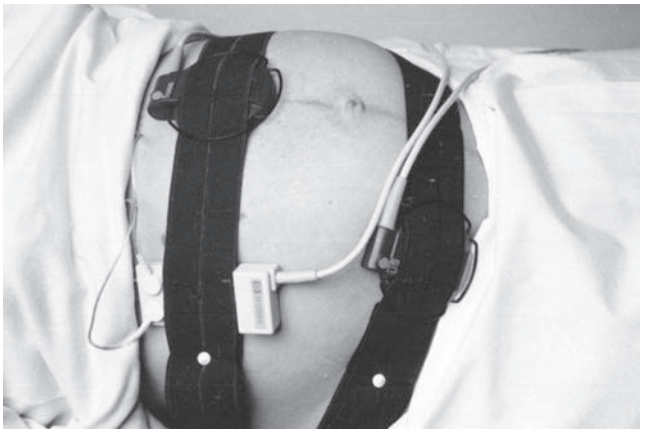

Figure 4.22. Maternal electrocardiogram, external tocodynamometer, and ultrasound fetal heart rate transducer on patient.

When removing the electrode, twist the wires counterclockwise simultaneously. This can be done just before or after delivery of the neonate. At cesarean section, the electrode should be removed through the vagina before delivery if possible.

Following delivery, the area of electrode placement in the baby's scalp should be cleaned in the nursery.

The External Tocodynamometer

If the membranes are not yet ruptured, the external tocodynamometer allows the patient's uterine activity to be monitored in a nonquantitative way. This is quite satisfactory for patients who are progressing well. Occasionally, FHR decelerations may be observed in a patient when contractions are not recorded with an external tocotransducer. Intrauterine pressure monitoring may clarify the timing of the decelerations with respect to uterine activity.

External tocotransducers come in many different sizes and shapes and are attached by means of an elastic belt around the patient's abdomen. When the uterus contracts, the change in shape and rigidity slightly depresses a plunger, causing a change in the voltage of a small electrical current. These voltage changes are proportional to the uterine activity and are represented qualitatively by the fetal monitor as contractions. Because this method does not reflect the true intrauterine pressure, avoid detecting only the peaks by adjusting the pen position to set the uterine activity channel at about 25 relative units between contractions. Positioning of the external tocotransducer is important because if not placed over the proper part of the abdomen, uterine activity may not be detected at all. Palpate to find where contractions are the most easily felt abdominally and place the transducer with careful attention to adjustment of the elastic belt to an appropriate tension (Fig. 4.22). In the presence of maternal obesity or a small uterus, the external tocotransducer is very poor at picking up uterine activity (Fig. 4.23). Palpate and pay attention to both the patient's complaints of contractions and any evidence of FHR decelerations suggestive of undetected uterine activity. While the external tocotransducer

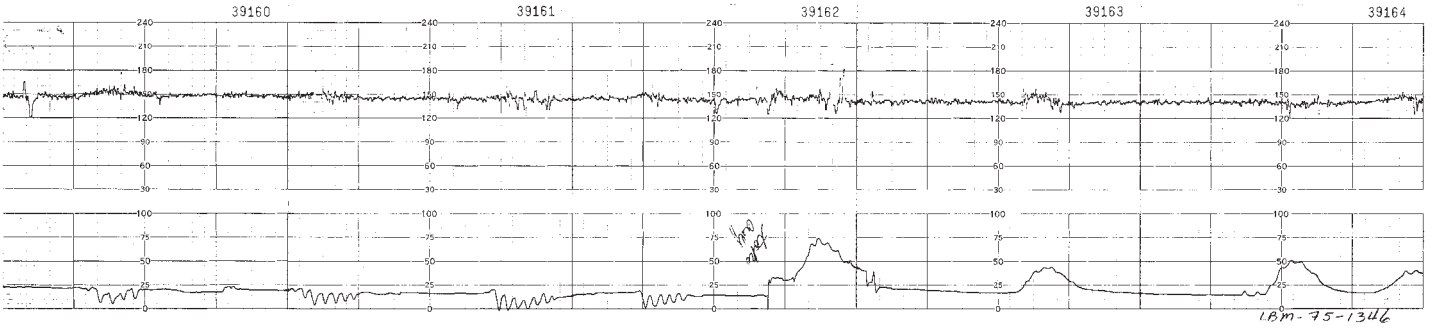

Figure 4.23. This tracing shows that contractions were evident only as irregular areas on the pressure tracing until the tocodynamometer was adjusted and repositioned. Subsequent contraction recording is satisfactory.

estimates the frequency and duration of contractions fairly well, the baseline tone and actual contraction amplitude cannot be measured with the external tocotransducer. Monitoring with a tocotransducer provides real-time pressure measurements of uterine activity compared to a baseline. Newer monitors have simplified the technology of establishing baseline, and this can be accomplished either manually or automatically. One should follow the specific instructions with the type of monitor used in order to obtain as accurate information as possible with external tocotransducers.

The Doppler Transducer

The Doppler transducer is usually secured to the patient with an adjustable elastic strap that encircles the maternal abdomen. Selection of the optimum location for the transducer should proceed in the following manner:

1. Place the retaining strap under the patient in the supine position.
2. Place an adequate amount of ultrasonic coupling gel over the transducer face and apply to the maternal abdomen.
3. Begin searching for the strongest signal by listening with the monitor audio turned on. In cephalic presentation, this is usually found to be below the umbilicus; with a breech, it may be higher on the maternal abdomen. It is often best to locate the fetal back with Leopold's maneuvers and place the transducer over this area. Use of real-time ultrasound can also assist in confirming fetal position and heart location.
4. When the optimal area is located, secure the transducer to the retaining strap and adjust for final placement at the location where the signal is clearest and the monitor is able to record well.

Because the Doppler transducer both transmits ultrasonic beams and receives the reflected signals, the angle of the transducer is important. By the laws of physics, the beam is reflected from a moving interface with the angle of incidence equaling the angle of reflection. Therefore, the beam will only return to the transducer when it is arranged perpendicular to the moving interface. Because of this, the transducer should be tilted (Fig. 4.24) to get the optimal signal, then moved to a point where the tangential placement allows reception of the reflected beam without transducer tilt. Once the transducer placement is optimal, it is necessary to constantly recheck the signal. If the fetus moves or the mother changes position, the signal may be lost and the transducer may have to be repositioned. With newer monitors using wide-beam ultrasound and autocorrelation, obtaining a continuous, reliable fetal cardiac tracing is less problematic, but the possibility of recording MHR is increased.

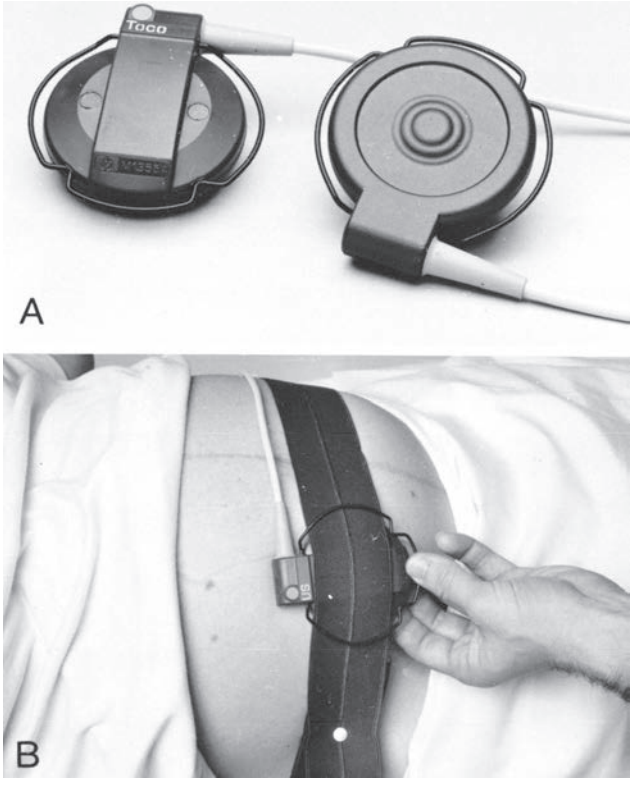

Figure 4.24. Newer monitors have dramatically improved the quality of the external mode. **A:** Both sides of the external tocodynamometer transducer. **B:** Demonstration of the placement of the ultrasound fetal heart rate (FHR) transducer over the area of the maternal abdomen that gives the best FHR signal.

Central Monitoring (Fig. 4.25)

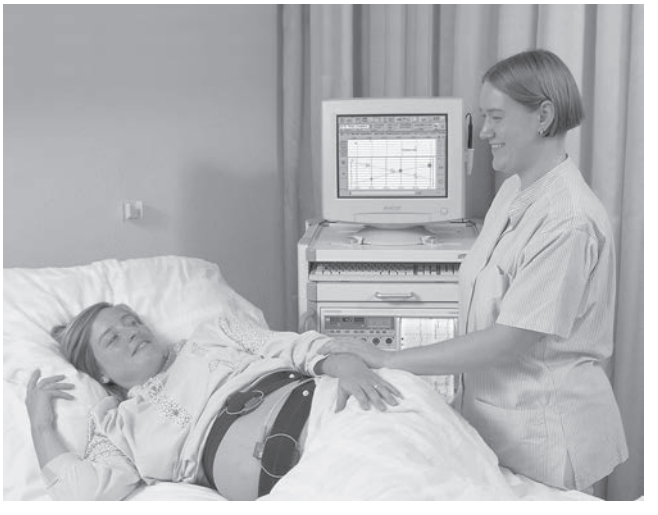

Figure 4.25. Example of centralized monitoring in a labor and delivery suite. This allows the physician or nurse to observe simultaneously the fetal heart rate (FHR) tracing of several patients. In addition, a terminal can be placed in the physician's office to allow for instant access to the FHR data. (Koninklijke Philips Electronics, the Netherlands.)

Figure 4.25. *(continued)*

Twin Monitoring

Monitoring twins has always presented a challenge. On fetal monitors with twin monitoring capability, one twin's heart rate is recorded in bold dark style and the other is recorded in thinner light style (Fig. 4.26). The physician/nurse who is monitoring the patient should assign the dark tracing to one twin and the light tracing to the other. Too often, this is not noted in the record, and later on, it is not possible to determine which tracing goes with which twin. When monitoring both twins with Doppler transducers, it is possible to be monitoring the same twin on each transducer. This is referred to as signal coincidence. The easiest way to monitor twins is to have a fetal electrode on the presenting twin and use a Doppler transducer on twin B.

Because of the difficulty in determining whether there is signal coincidence (having one twin on both transducers), it is necessary to detect this potentially problematic situation. Newer monitors with twin monitoring capability have built-in logic to detect signal coincidence. Logic in the monitor will compare the two recordings from the two Doppler transducers and, if they are the same, will alarm indicating signal coincidence that indicates the same twin recording on both transducers. On the GE monitor, overlapping hearts above the FHR tracing indicate coincidence. When the coincidence is resolved, the overlapping hearts become side-by-side hearts (Fig. 4.27). On the Philips monitors, question marks above the tracing indicate the same twin on both transducers (Fig. 4.28).

Another problem with twin monitoring occurs when the two twin baseline heart rates are nearly identical. A method that is now available allows the two tracings to be separated by 20 BPM making tracing interpretation less problematic (Fig. 4.29). This can also be done with triplets (Fig. 4.30).

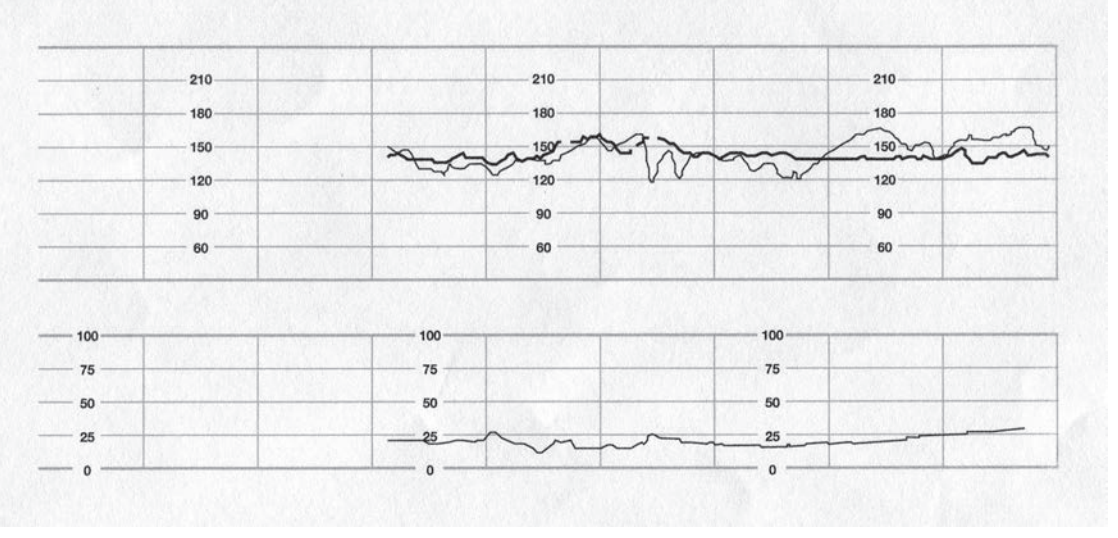

Figure 4.26. Example of twin monitor strip. Note the dark and the light tracing representing the twins. One should note twin A and B as assigned to dark and light tracing.

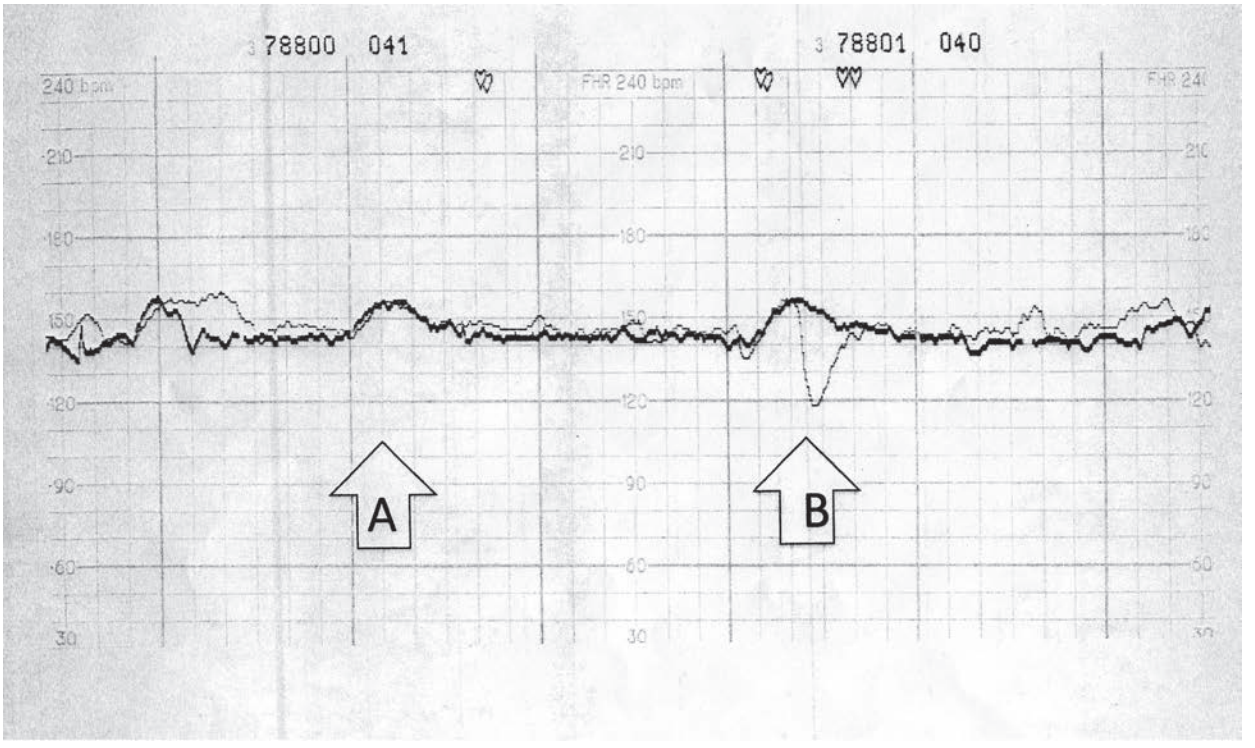

Figure 4.27. With the GE Corometrics monitor, when only one twin is being traced on both transducers, there are overlapping hearts displayed above the tracing (A). When the Doppler transducers are each recording different twins, the hearts are separated (B).

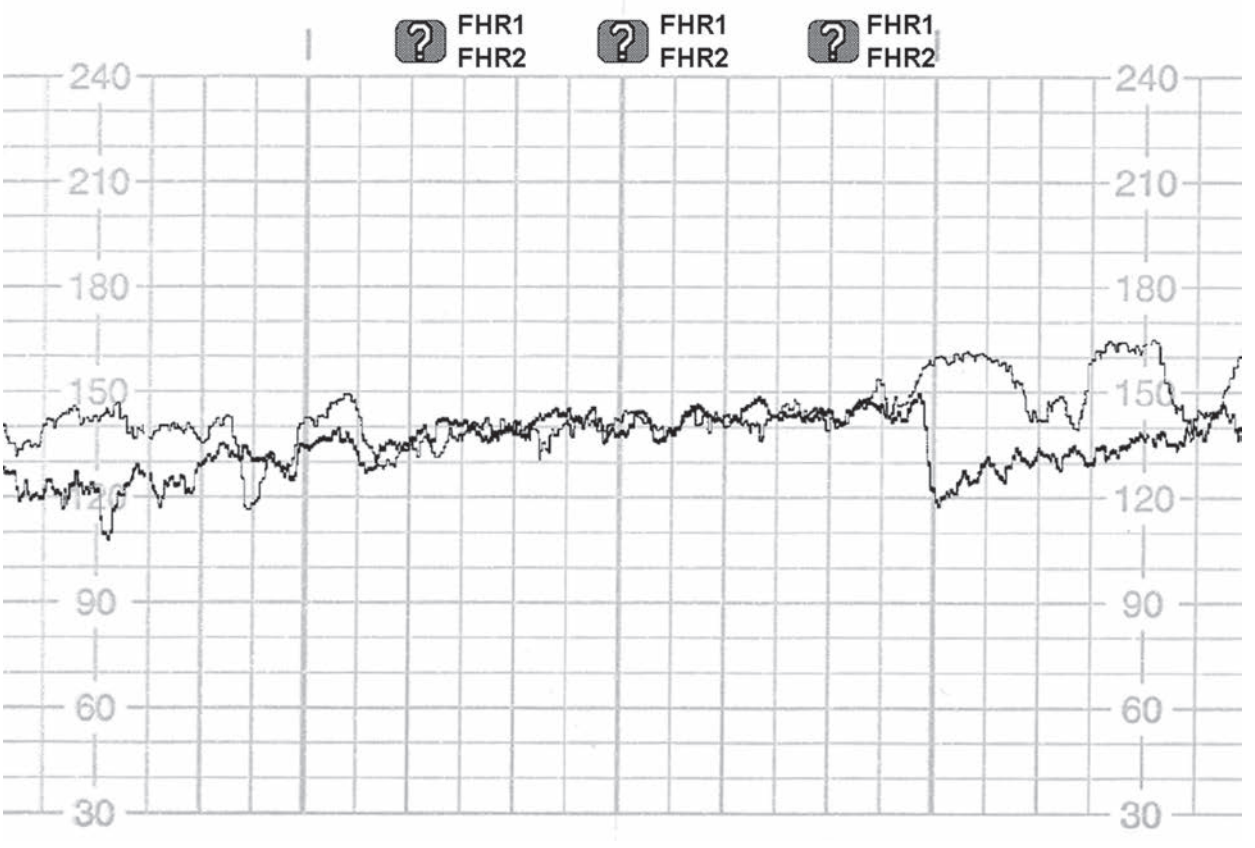

Figure 4.28. On the Philips monitor, when the same twin is being recorded on both transducers, a question mark is printed above the tracing, and when the twins are again separated, the question marks go away.

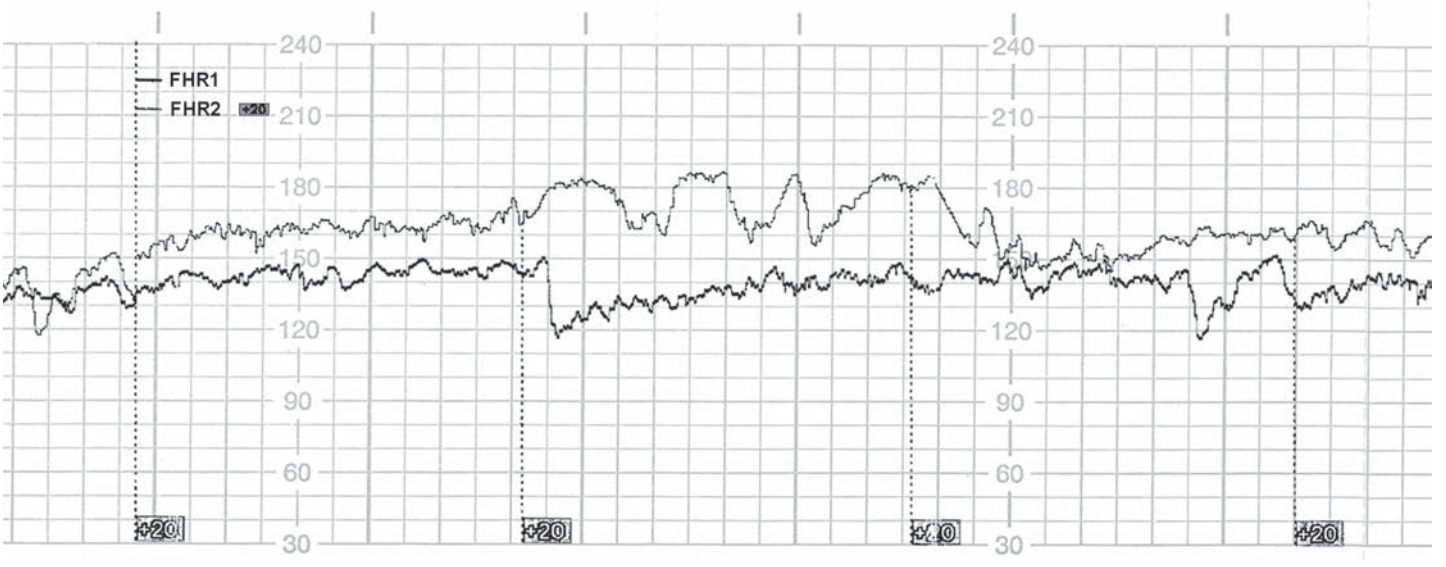

Figure 4.29. There is an option to separate the twins by 20 beats per minute (BPM) in order to get a better look at each tracing. On the Philips monitor, a +20 is printed when the separation begins and goes away when the separation is terminated.

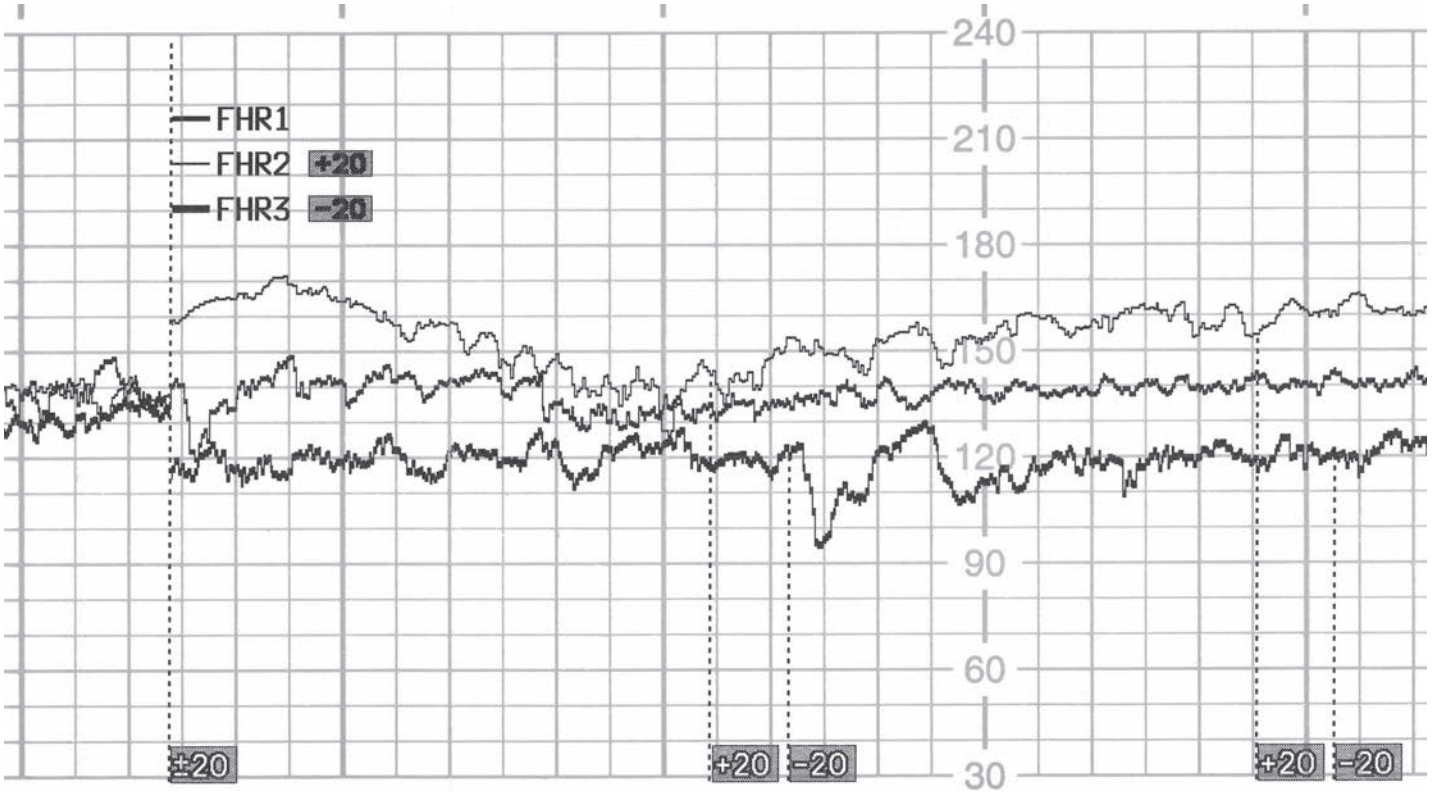

Figure 4.30. With triplets, one fetus may have a 20-BPM increase and another may have a 20-BPM decrease in order to separate the three tracings oaf the triplets.

Signal Ambiguity from MHR

Since the publication of the third edition of this book, a problem caused by newer instrumentation of FHR monitors has come to our attention (14). Figure 4.31 is an example of transition from fetal to maternal heart rate recording while positioning a patient for an epidural anesthetic with Doppler FHR recording. It is well known that during the second stage of labor, with maternal pushing efforts, the mother becomes tachycardic and accelerates her heart rate with contractions (15). This has incorrectly been interpreted by clinicians as FHR with accelerations. With the newer monitors, the transition from fetal to maternal signal does not always transition with discontinuity of recording (signal loss) as with older monitors (Fig. 4.32). There are now more than 20 documented cases of MHR rather than fetal recording from the abdominal Doppler transducer resulting in the birth of three stillborn fetuses and a dozen cases of severe fetal metabolic acidosis resulting in neonatal encephalopathy and later cerebral palsy.

The newer monitors have electronic logic that was originally used when monitoring twins (see twin monitoring) to alert if the two Doppler transducers were monitoring the same twin or if there was one twin and the mother's heart rate being recorded. This alerting system compares electronic signals from the two transducers and alerts if they are both recording the same twin or, in the case of MHR recording, comparing the Doppler transducer recording with an MHR source from either a maternal ECG or from maternal pulse oximeter recording. If an MHR source (ECG or pulse oximeter) is fed into the monitor, it will alert, indicating signal coincidence the same as with twins when signal coincidence occurs (Fig. 4.33).

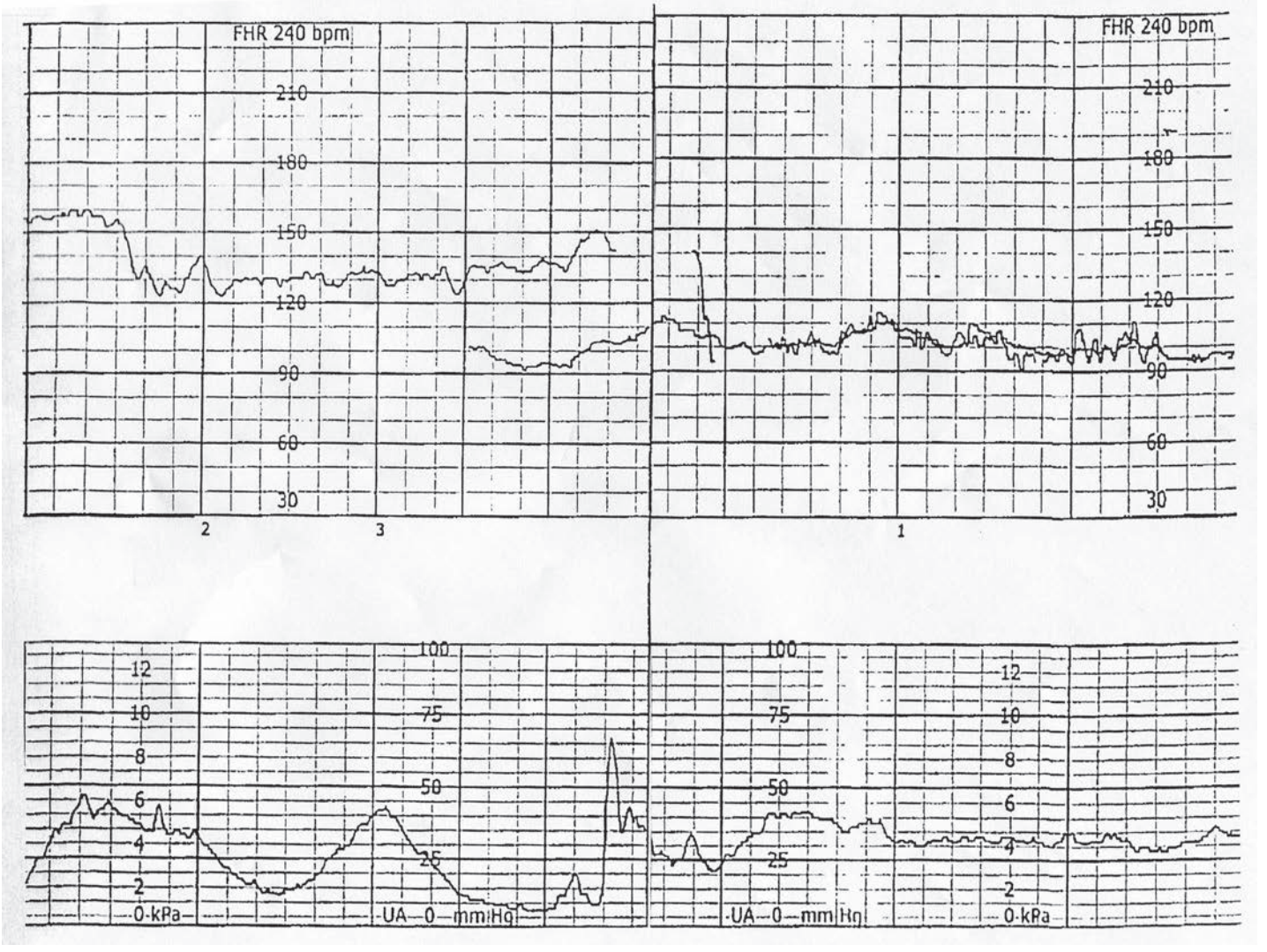

Figure 4.31. When this patient, who was being monitored with a Doppler external transducer, was moved to administer an epidural, the fetal signal was lost and the monitor began recording the maternal heart rate (MHR). The MHR was being recorded from a pulse oximeter just prior to the switch showing that the new heart rate was indeed maternal and not a fetal deceleration. This monitor did not have a signal coincidence warning system in place.

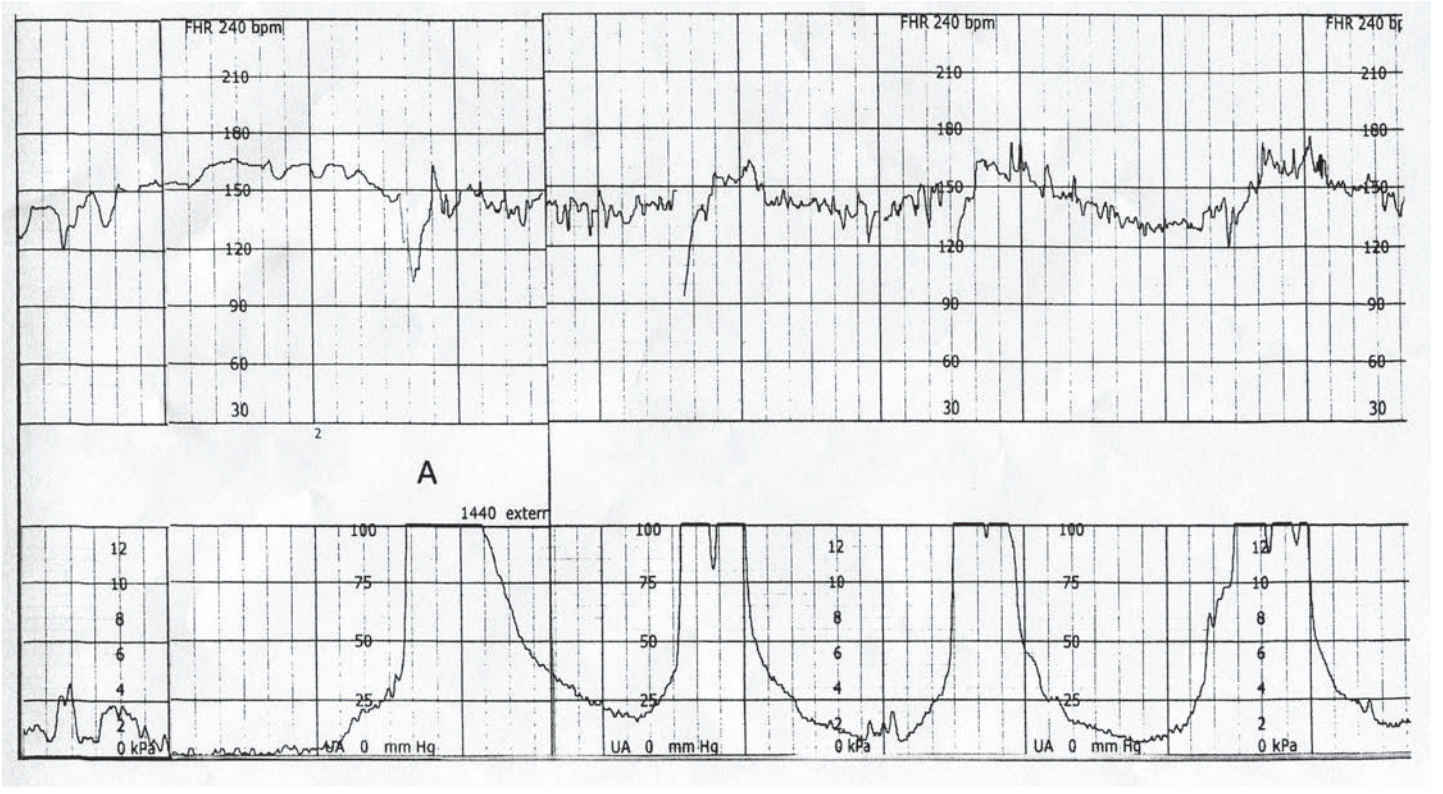

Figure 4.32. This fetus was being monitored with a Doppler external transducer, and at point A, it switched to begin monitoring the maternal heart rate. The switch occurred during a continuous tracing without signal loss as was usually seen on older monitors.

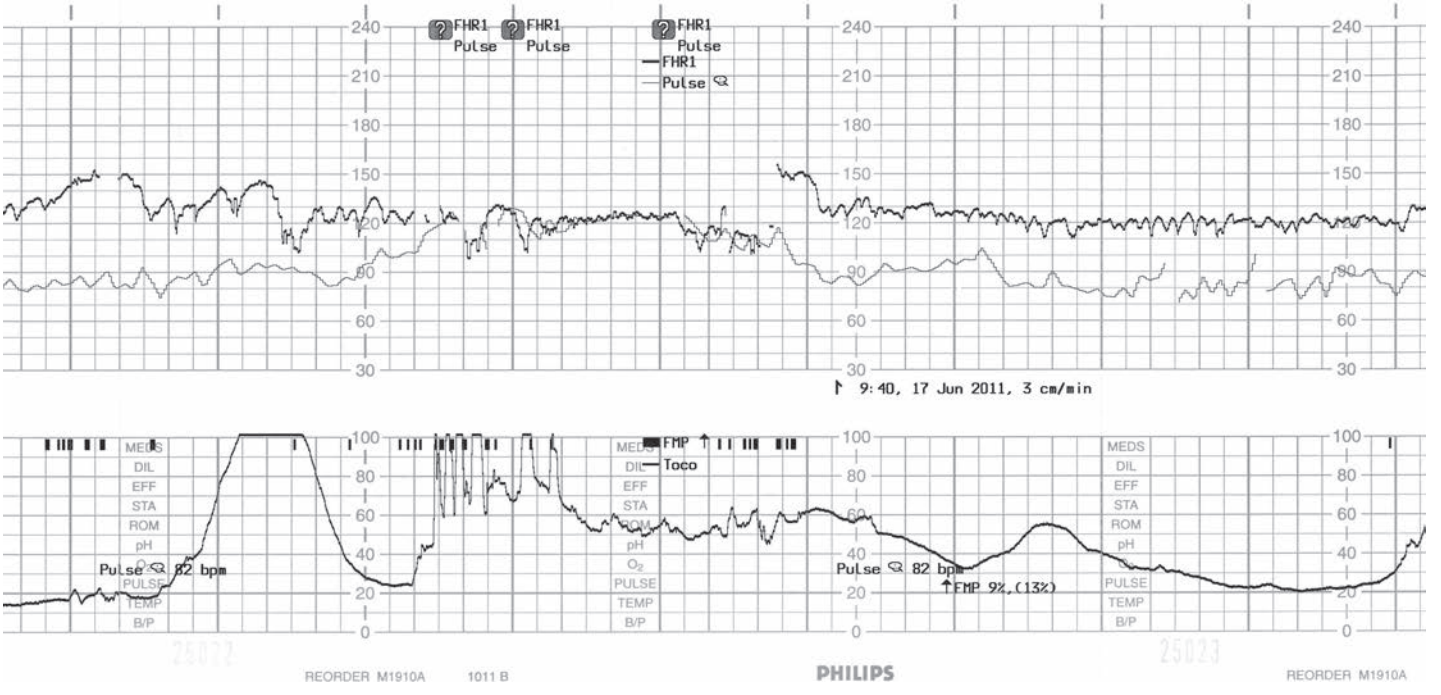

Figure 4.33. On this recording, it begins with the fetal heart rate baseline at about 120 BPM and maternal pulse rate from an optical sensor integrated in the Toco transducer with baseline about 80 BPM. When the ultrasound Doppler transducer from the maternal abdomen began to pick up the maternal pulse the characteristic question marks appear at the upper part of the tracing, indicating coincidence on a Philips monitor.

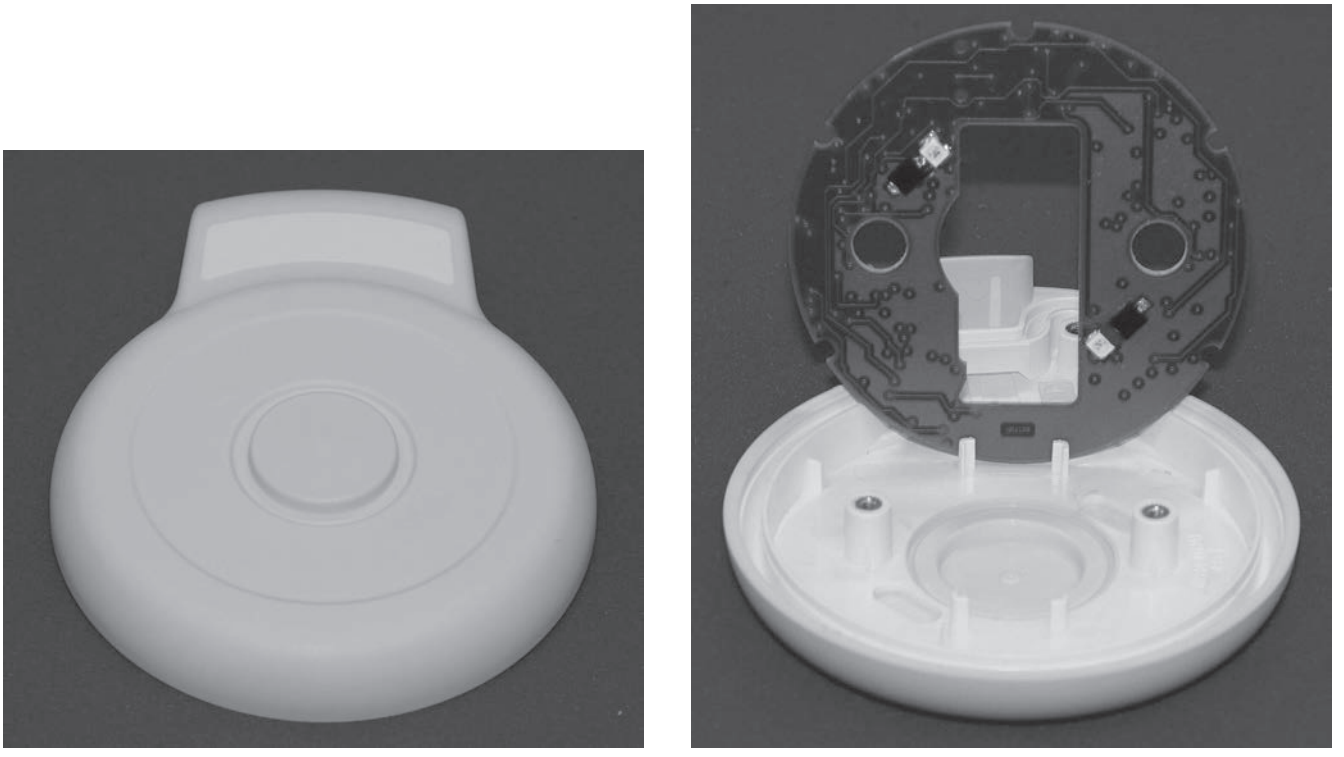

Figure 4.34. This figure shows the new Toco transducer developed by Philips: It has an integrated maternal optical reflection sensor system giving a continuous maternal pulse rate signal that is automatically used to detect fetal/maternal signal coincidence.

This problem has been reported to the FDA, and because of this, some monitors have been recalled. The Philips company has developed a solution that involves a optical reflection sensor being placed on the tocotransducer so that there is always an MHR source and presumably such coincidence will always be detected (Fig. 4.34). This new system of Philips is called Smart Pulse and was approved by the FDA in August of 2011.

NETWORKING OF FETAL HEART RATE MONITORING

Since publication of the previous edition, there have been many advances in our ability to record, store, and transmit fetal monitoring data electronically. Any practitioner who has access to a computer or "smart phone" can observe fetal monitoring data from remote locations, with the appropriate software. While this is a clear advance, there is some concern that it will enable decisions to be made remotely when it may be better if the provider were at bedside to manage urgent developments. At the time of this writing, there are rapid new techniques for remote monitoring developing, which make it difficult to be more specific with the inevitable future technologic developments in this area.

In summary, FHR monitoring instrumentation continues to evolve, and there have been recent problems that have come to our attention. Specifically the problem of signal ambiguity resulting in MHR recording that has led to poor fetal outcomes because the fetal signal was lost during these times. Twin monitoring, even with signal coincidence detection capability, similarly can result in MHR recording from one transducer, thus obscuring one twin's FHR recording. The development of continuous maternal signal acquisition should obviate this problem in the future.

Fetal Cardiac Arrhythmias

Fetal cardiac arrhythmias occur in one form or another in a significant number of pregnancies. The documentation of various fetal arrhythmias has increased subsequent to the more extensive application of antepartum and intrapartum electronic FHR monitoring. The importance of recognition of an abnormal cardiac rhythm, correct diagnosis of arrhythmia type, associated incidence of underlying heart disease, and the need for appropriate medical intervention depends on the specific type of arrhythmia present. Diagnosis can be established through the use of both M-mode and real-time ultrasound (16,17). In the absence of signs of failure

(i.e., cardiac enlargement or hydrops) or evidence of FHR abnormalities suggestive of hypoxia, most fetal cardiac arrhythmias are benign, do not require immediate delivery, and are not associated with structural fetal cardiac abnormalities. Many arrhythmias, particularly those diagnosed during the intrapartum period, such as PACs and PVCs, do not persist in the neonatal period and rarely require medical therapy. PACs and PVCs are, for the most part, entirely benign and do not require any special attention. However, certain types of arrhythmias are of clinical significance for both mother and fetus. Supraventricular tachycardias (SVTs) and fetal heart block may be associated with previously undiagnosed fetal compromise, maternal disease, or both and frequently require active management.

FETAL TACHYCARDIA

The three general types of fetal tachycardia are sinus tachycardia, atrial flutter/fibrillation, and SVT.

Sinus Tachycardia

Sinus tachycardia is defined as FHR above 160 BPM and is usually secondary to maternal fever, drugs (i.e., atropine, β-sympathomimetics), amnionitis, congenital infection, or hyperthyroidism (Fig. 4.35). Although benign, on occasion, sinus tachycardia accompanied by late or severe variable decelerations may be a sign of early fetal hypoxia (18). Recognition of the cause of tachycardia and differentiation from SVT are of obvious importance. Tachycardia is not a sign of fetal hypoxia unless it is associated with ominous decelerations.

Atrial Flutter/Fibrillation

The antepartum diagnosis of atrial flutter or fibrillation is rare. A monotonous atrial rate that varies between 400 and 500 BPM is seen with this form of supraventricular tachyarrhythmia in the fetus. The ventricular rate is much lower due to an accompanying AV block. If the AV block is fixed, a regular ventricular rate of 60 to 200 BPM is seen. If the block is intermittent, the ventricular rate will vary widely. Atrial flutter and fibrillation are serious arrhythmias; nonimmune hydrops can occur, and a concomitant severe congenital heart defect is found in as many as 20% of cases (19). These arrhythmias are most resistant to in utero therapy and are associated with a very high fetal and neonatal mortality rate. Atrial fibrillation is rarer in the fetus than atrial flutter. Treatment with digoxin and/or verapamil may increase the degree of AV block but rarely corrects the atrial arrhythmia. However, control of ventricular rate does not necessarily

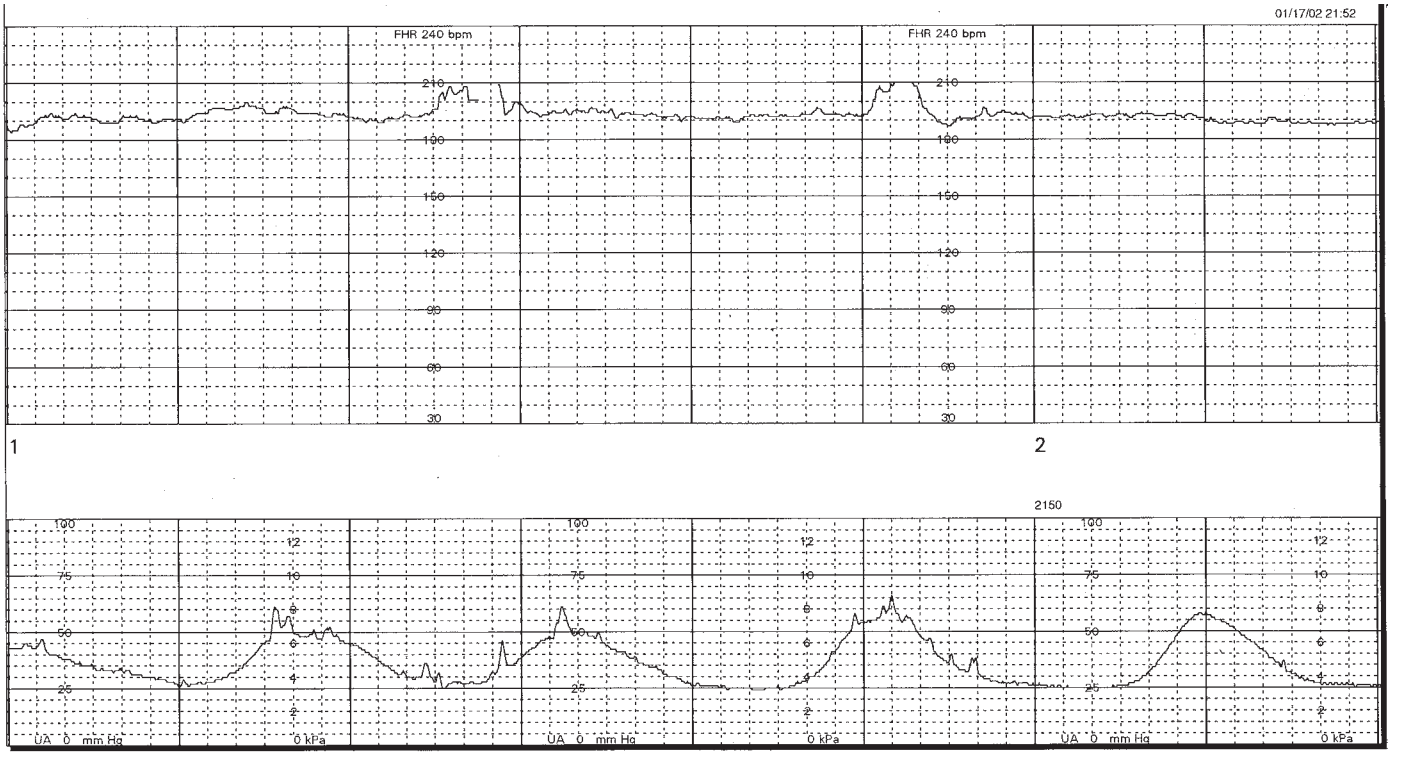

Figure 4.35. Sinus tachycardia. This example of fetal tachycardia is present in a patient with a fever and presumptive diagnosis of chorioamnionitis. Note the absence of decelerations, which is a reassuring finding regarding fetal oxygenation.

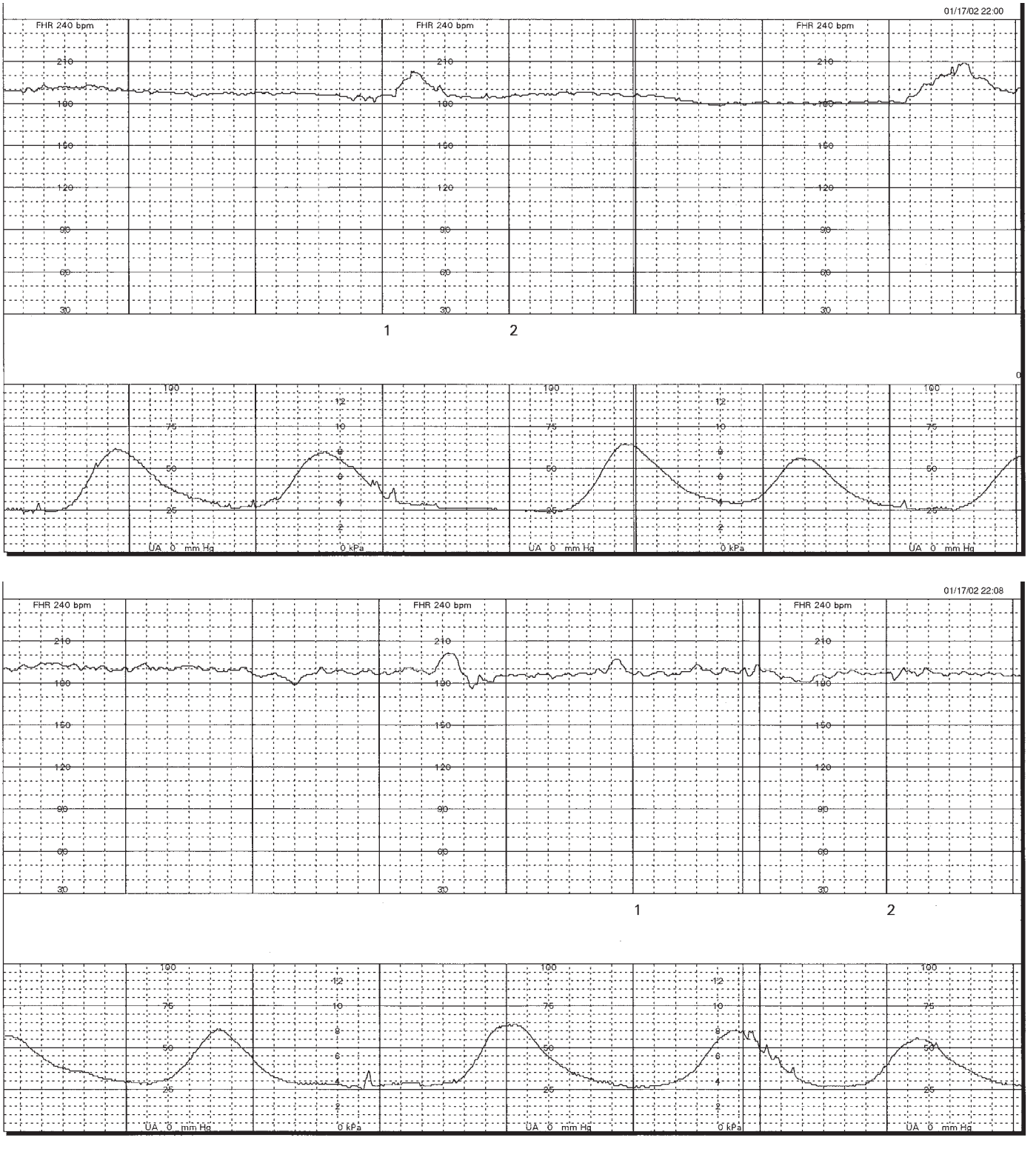

Figure 4.35. *(continued)*

improve fetal hydrops. This may require restoration of a 1:1 AV conduction. The use of type I antiarrhythmic agents (e.g., flecainide, quinidine, procainamide) should be considered as well in such patients.

Supraventricular Tachycardia

Thought to be the most frequent form of fetal tachyarrhythmia, SVT can occur for short periods and be of no clinical significance, or can persist for long periods and lead to high output failure, nonimmune hydrops fetalis, and fetal death (20,21). Defined as an FHR greater than 220 BPM with no variability or conduction abnormality, SVT can be suspected on cardiac auscultation and confirmed with M-mode echocardiography and pulsed-Doppler flow-velocity waveforms (22). Real-time ultrasound often will demonstrate varying degrees of cardiac failure. Although there may be no evidence of effusions or hydropic changes coexistent with SVT, rapid progression can be seen in as little as 36 hours of continued tachyarrhythmia. More commonly, this is not a continuous but rather intermittent arrhythmia for which the term (paroxysmal) atrial tachycardia was used in the past.

SVT may be a manifestation of atrial flutter or fibrillation and, in theory, results from one of two mechanisms. The first is increased automaticity of an ectopic pacemaker (other than the normal sinus nodal pacemaker) above the bundle of His. The second, and more likely, mechanism in fetal and childhood SVT is reentrant tachycardia resulting from a circular "circus movement" of electrical activity, most commonly within the AV node. Less commonly, but seen in the Wolff-Parkinson-White (WPW) syndrome, this movement is due to an accessory conduction pathway (bundle of Kent). Both types of reentrant tachycardia are thought to result from an atrial premature contraction being conducted through or around the AV node down a repolarized but relatively slow pathway. The fast pathway is thought to be in a refractory period following the previously normally timed atrial depolarization. If sufficiently slow conduction occurs, allowing recovery of the fast pathway, ventriculoatrial conduction up the fast pathway results in a sudden reentrant tachycardia. The initiation and maintenance of this tachycardia depends on the refractory periods and conduction velocities within the slow and fast conduction pathways in the AV node. The timing of the arrival of the premature contraction is the critical factor in the initiation of the arrhythmia.

TREATMENT OF FETAL TACHYCARDIA

Sinus tachycardia is often encountered in the intrapartum period and usually reflects a drug effect, sympathetic fetal response to maternal infection, occult amnionitis, or, rarely, fetal hypoxia. The importance of identifying sinus tachycardia is not to institute a specific therapy but to determine its cause and separate it from SVT (Fig. 4.35). Ventricular tachycardia (VT), as defined by three or more consecutive premature ventricular systoles, is extremely rare in the fetus, and its significance remains unknown. Ventricular fibrillation in the fetus has not been reported.

Treatment of fetal SVT has been extensively reported. In utero pharmacologic therapy utilizing single drugs or various combinations of digoxin, calcium-channel blockers, beta-blockers, procainamide, and quinidine have all been reported with varying degrees of success (23). Once the diagnosis of SVT is made, with or without the associated signs of fetal cardiac failure, delivery of the mature fetus or in utero therapy of the immature fetus must be initiated. Fetal SVT requires urgent management at any gestational age. Digoxin has been the most commonly used drug for the initial treatment, and the combination of rapid digitalization and maintenance of the maternal digoxin level in the middle-to-upper therapeutic range with the addition of a calcium-channel blocker, such as verapamil, or beta-blocker, such as propranolol or atenolol, has been our most successful approach (Figs. 4.36 and 4.37). Quinidine is an alternative drug that, following adequate digitalization, can be of value in controlling fetal SVT. Unfortunately, 30% of patients cannot tolerate this drug because of severe nausea and vomiting. Digoxin should be initiated as a single agent with an intravenous loading dose of 1.0 mg (0.5 mg followed by 0.25 mg at 6-hour intervals) while the patient is hospitalized and on continuous cardiac monitoring. The goal is to achieve a maternal blood level at the upper end of the therapeutic range. If there is no improvement in fetal rhythm after 2 days with a high therapeutic blood level of digoxin in the mother, a second agent or even third agent may be required. It is very important to consider that many of these additional antiarrhythmic agents increase both the serum level and bioavailability of digoxin. Consequently, the maternal digoxin dose should be reduced by at least 50% when using such agents as quinidine, verapamil, or amiodarone. When M-mode echocardiography reveals atrial flutter or fibrillation, one of the drugs of choice to control the associated rapid ventricular response due to AV node bypass is procainamide, following the administration of digoxin, verapamil, or propranolol. The different types of fetal SVT can be controlled most frequently with digoxin and either verapamil or propranolol. When a combination of agents is indicated, the best choice is to combine the use of an agent that affects the fast pathway along with one that affects the slow pathway (Tables 4.1 and 4.2). With the exception of digoxin, cardiac antiarrhythmic drugs used to treat reentrant SVT also decrease automaticity with consequent lowering of the incidence of premature beats.

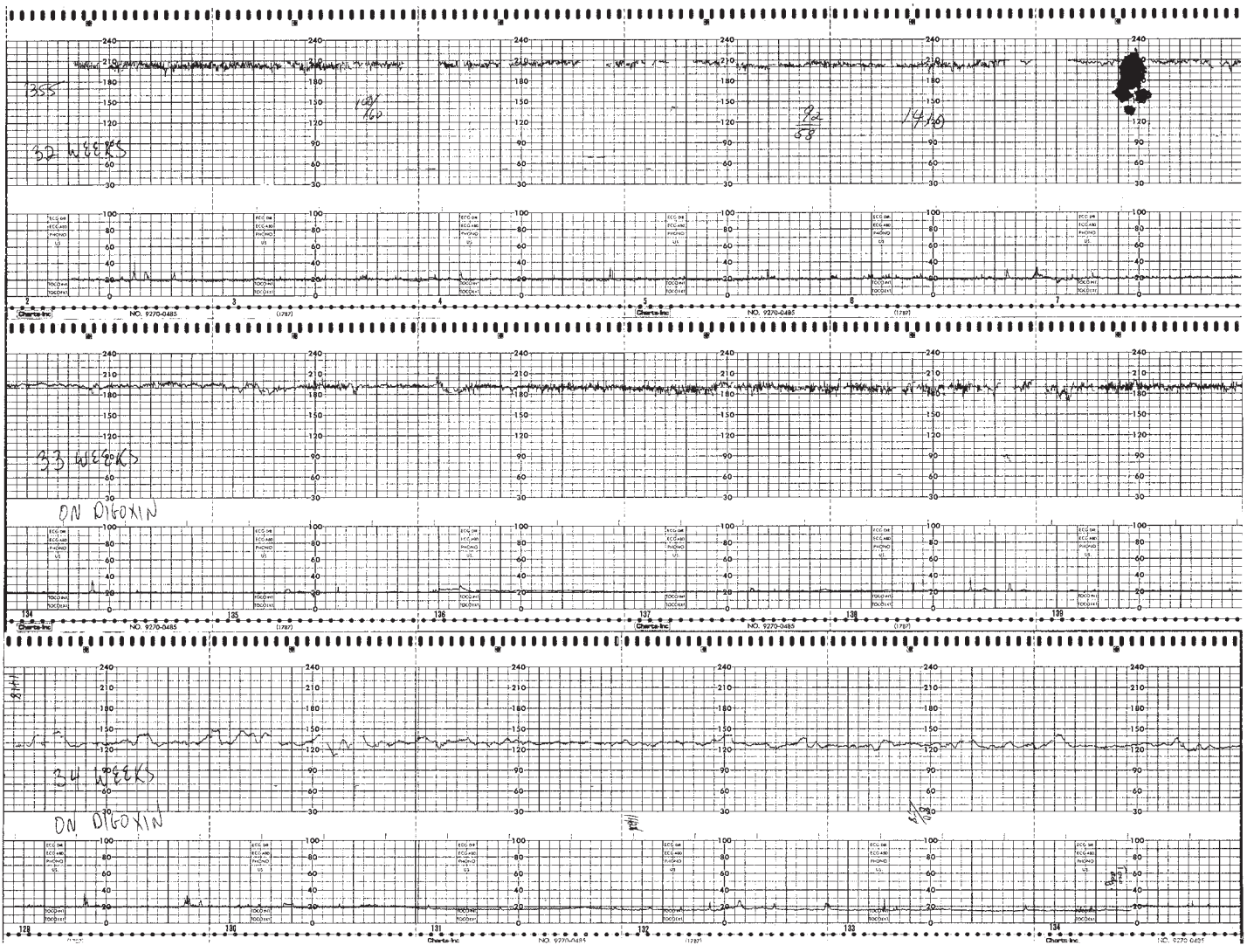

Figure 4.36. Sequential examples of fetal supraventricular tachycardia cardioverted in utero with digoxin treatment of the mother. The fetus was not hydropic during treatment.

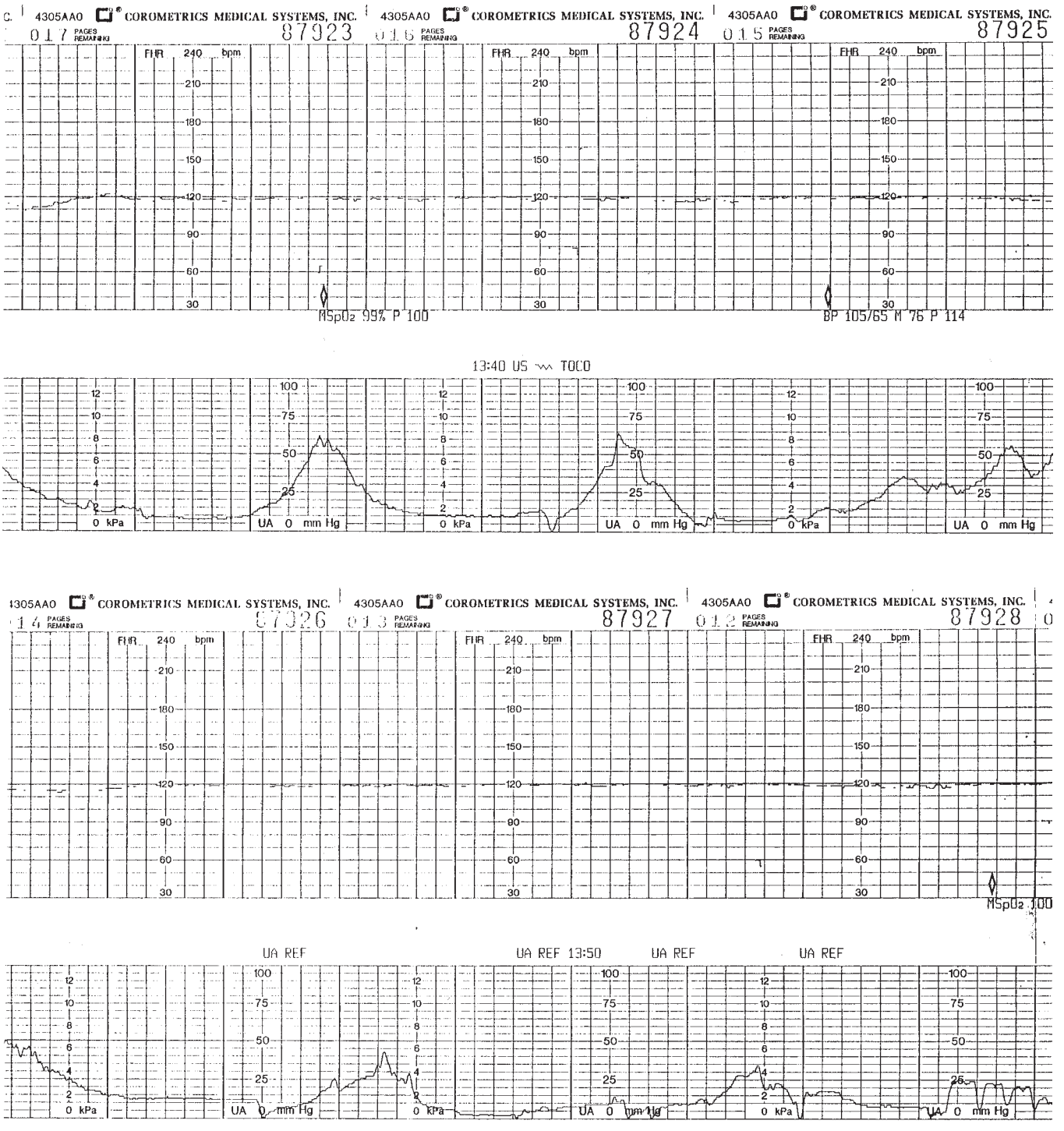

Figure 4.37. Example of supraventricular tachycardia. Note the complete absence of accelerations in the fetal heart rate (FHR). The actual FHR was 240 beats per minute with signal halving resulting from the monitor logic.

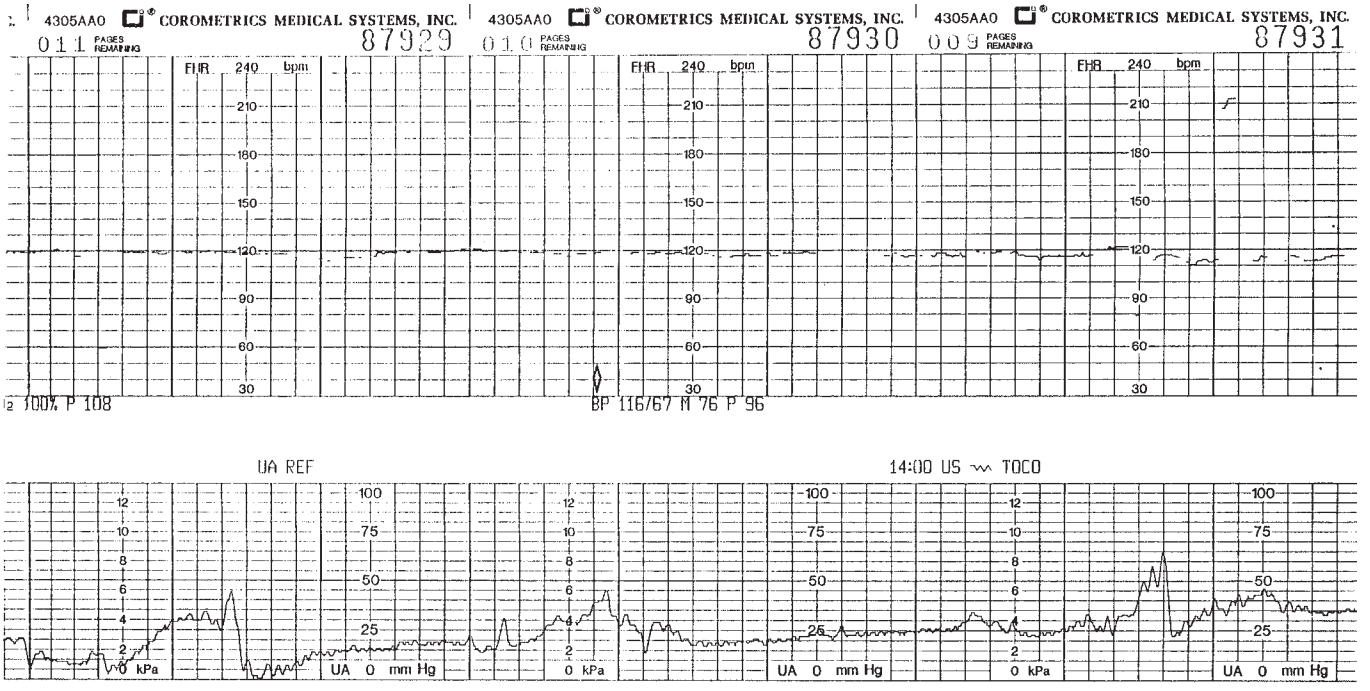

Figure 4.37. *(continued)*

TABLE 4.1 Antiarrhythmic drug classes

Class	Repolarization	Indications	Drug
IA	Prolongs	Atrial flutter Atrial fibrillation SVT VT	Quinidine Procainamide Disopyramide
IB	Shortens	VT	Lidocaine Mexiletine
IC	Unchanged	VT SVT	Flecainide Encainide
II	Unchanged	Atrial tachycardia VT	β-Blockers
III	Markedly prolongs	VT Atrial flutter Atrial fibrillation SVT	Amiodarone Sotalol
IV	Unchanged	Reciprocating SVT	Verapamil Diltiazem Adenosine

SVT, supraventricular tachycardia; VT, ventricular tachycardia. From Kleinman CS, Copel JA: Electrophysiological principles and fetal antiarrhythmic therapy. *Ultrasound Obstet Gynecol* 1:286–297, 1991, with permission.

CONGENITAL HEART BLOCK

Although most fetal arrhythmias are either extrasystoles or tachyarrhythmias, bradyarrhythmias due to complete heart block comprise approximately 10% of cases (24). The forms of heart block are first, second, and third degree (complete). Prolongation of the interval between the P and R waves (first-degree AV block), usually secondary to impaired conduction in the AV junction proximal to the bundle of His, requires no treatment and has not been reported in the fetus. Second-degree or partial AV block, in which some but not all atrial impulses are conducted to the ventricles, is present in most fetuses with SVT. Second-degree AV block presents in two forms, Mobitz types I and II. In Mobitz type I block, the P-R interval increases progressively until complete blockage of an atrial impulse results in a dropped ventricular beat (Wenckebach type).

TABLE 4.2 Depression of AV nodal conduction—fast and slow pathways

Antegrade (slow) limb	Retrograde (fast) limb
Digitalis	Class IA—quinidine, procainamide
β-Blockers	Class IC—flecainide
Calcium-channel blockers	

This cycle is repeated, following anywhere from two to eight consecutive impulses with progressive lengthening of the P-R interval. In the fetus, Mobitz type I second-degree block is rarely either persistent or of any significance (25). Mobitz type II block occurs infrequently and is more serious. The delay is at a level below the AV junction, and the P-R interval is normal or increased but fixed. This arrhythmia has dropped beats in a regular or irregular frequency and usually results from blockage in the bundle of His or trifascicular block. Mobitz type II second-degree block has been well described in the fetus (26). This form may precede development of complete heart block.

Complete, or third-degree, AV block has been the focus of most reports of fetal or neonatal heart block. Thought to be due to a failure of union of the AV node and His bundle in early fetal development, complete congenital heart block may also result from damage to the conducting system after it has been normally formed. It results in the fetus from blockage at the AV junction with, by definition, complete dissociation of the atria and the ventricles. The ventricular rate is usually between 40 and 60 BPM, and fetal hemodynamic compensation, presumably secondary to an increase in stroke volume, is frequently observed (Fig. 4.38). In the absence of significant underlying congenital heart disease, the neonate often does well, although a pacemaker may need to be placed very soon after delivery (27). However, this clinical condition offers a guarded prognosis for the newborn, whatever the actual cause may be. As many as 50% of infants with complete heart block have associated congenital cardiac malformations (25). Mothers of infants with congenital heart block in the absence of congenital heart disease are at increased risk for connective tissue disease, particularly systemic lupus erythematosus (SLE), either subclinical or overt (28). The maternal disease may be nonexistent, heralded by a serologic abnormality, or associated with current or subsequent development of severe connective tissue disease. Evaluation of the mother once fetal congenital heart block is diagnosed is clearly indicated as only 50% of fetuses with bradycardia are born to women with a history of connective tissue disease (29).

The presence of specific antibodies to the soluble tissue ribonucleoprotein antigen Ro (SS-A) in the serum of mothers giving birth to infants with complete heart block has been described (30). There is immunofluorescent evidence of maternal anti-Ro immunoglobulin in the cardiac tissue of infants with congenital heart block. This finding suggests a transplacental passage of this immunoglobulin with direct effects in the fetal cardiac conduction system (31). This anti-Ro immunoglobulin can be measured in the serum of pregnant women with SLE, and if present, close fetal cardiac evaluation should be performed prospectively during pregnancy.

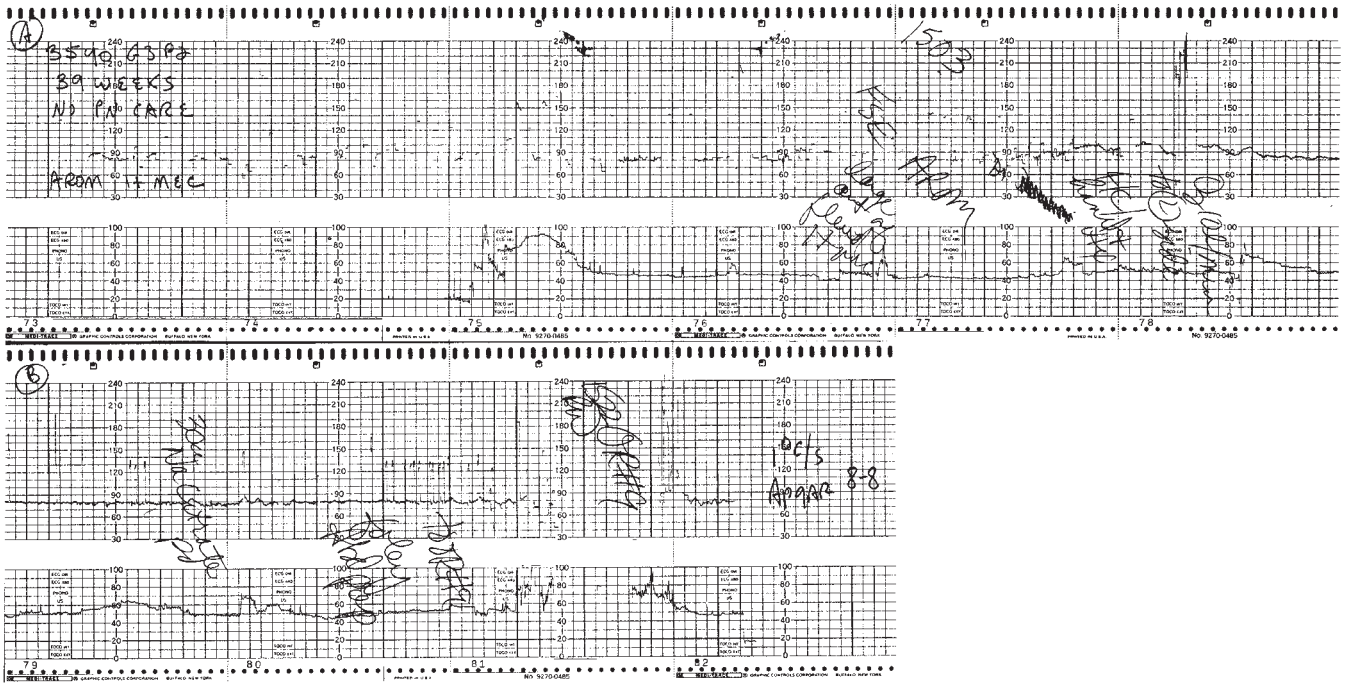

Figure 4.38. Example of emergency delivery for a patient presenting with a fetal heart rate of 80 beats per minute and no prenatal care. At delivery, the infant was discovered to have congenital heart block. The mother was antinuclear antigen negative and asymptomatic for any connective tissue disease.

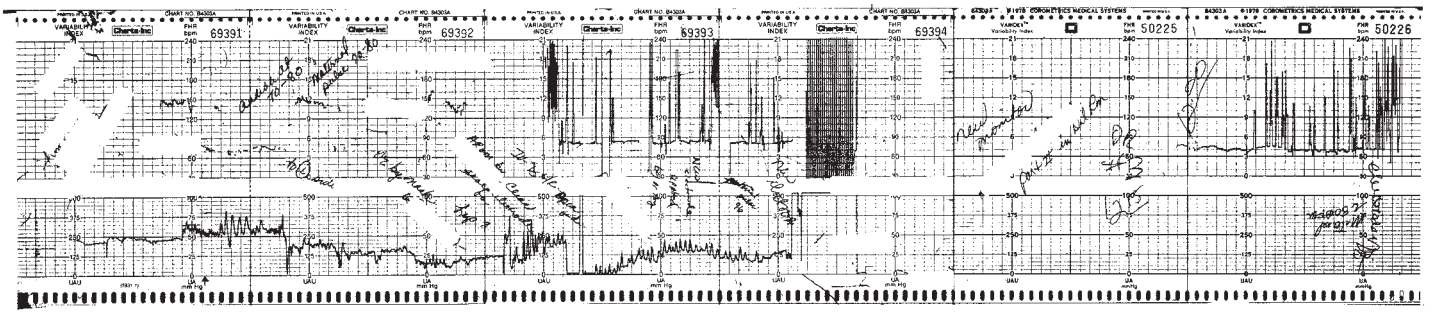

Figure 4.39. When complete heart block in the fetus is undiagnosed prior to entry into labor and delivery, it is not possible to determine the exact etiology of the abnormally slow fetal heart rate. The assumption that the heart rate was a prolonged fetal deceleration led to emergent cesarean section in this patient. At delivery, the infant was in no distress but maintained a heart rate of between 65 and 80 beats per minute. At discharge, he did not need a pacemaker, and the child has continued to do well. Workup of the mother diagnosed serologic evidence for systemic lupus erythematosus, although she has remained asymptomatic.

Treatment of congenital heart block depends on the coexistence of major cardiac abnormalities and fetal and neonatal tolerance of the fixed low heart rate. As mentioned, temporary or permanent pacemakers may need to be placed following delivery. Complete fetal heart block resulting in nonimmune hydrops from heart failure in utero occurs in approximately 25% of cases and is associated with high mortality. The mother may be exposed to unnecessary surgical risks when the arrhythmia is unknown and confused with a preterminal bradycardia in the intrapartum period (Fig. 4.39). In the fetus with complete heart block diagnosed before labor, the use of frequent intrapartum fetal pH determinations or continuous pulse oximetry of the fetus can result in a successful vaginal delivery. Our experience, however, has been that patients with fetal congenital heart block without congenital heart abnormalities incompatible with life are usually delivered via cesarean section because of the difficulty in monitoring these patients intrapartum. When intermittent, it is possible to allow these patients a trial of labor (Fig. 4.40).

Many forms of fetal therapy have been suggested in cases of complete fetal heart block (32). The treatment most often recommended is maternal administration of steroids, such as dexamethasone, in an effort to limit fetal inflammatory response to the transplacentally acquired maternal autoantibodies. Such treatment is associated with frequent and potentially severe maternal effects, and there is no evidence that the antibody-mediated damage of the fetal conduction system is reversible. Other therapies include intravenous gamma globulin and maternal plasmapheresis, but these therapies are not proven in the prevention or reversal of fetal heart block. Various medications to increase the FHR have also been reported (i.e., ritodrine, terbutaline, and isoproterenol) with variable responses and no proven benefit.

Direct pacing of the fetal heart in cases of complete heart block with nonimmune hydrops has also been reported. Carpenter reported the first case of percutaneous transthoracic fetal heart pacing with successful capture but fetal death within 3 hours (33). More recently, Harrison performed open fetal surgery with placement of an epicardial pacing wire and pulse generator (34). Once again, fetal death occurred quickly despite successful pacing.

Close monitoring of the fetus diagnosed with complete heart block is recommended. This includes both maternal perceptions of fetal movement as well the use of real-time ultrasound to identify early development of cardiac decompensation. Such assessment includes observing early hydropic changes, ventricular size and function, and AV valve insufficiency. The exact role of either medical or surgical therapies for treatment of fetal heart block remains unclear. The mortality rate for newborns with complete heart block is approximately 25%. After the neonatal period, survival is close to 90% with most deaths related to pacemaker failure (35). The newborn with complete heart block in the absence of congenital heart disease frequently has neonatal lupus erythematosus often manifested by a distinctive skin rash due to antibody deposition on basal keratinocytes (29). Additional manifestations include anemia, thrombocytopenia, hepatosplenomegaly, hepatitis, myasthenia, or myopathy. This constellation of findings may appear to a greater or lesser degree and will disappear by 6 months of age.

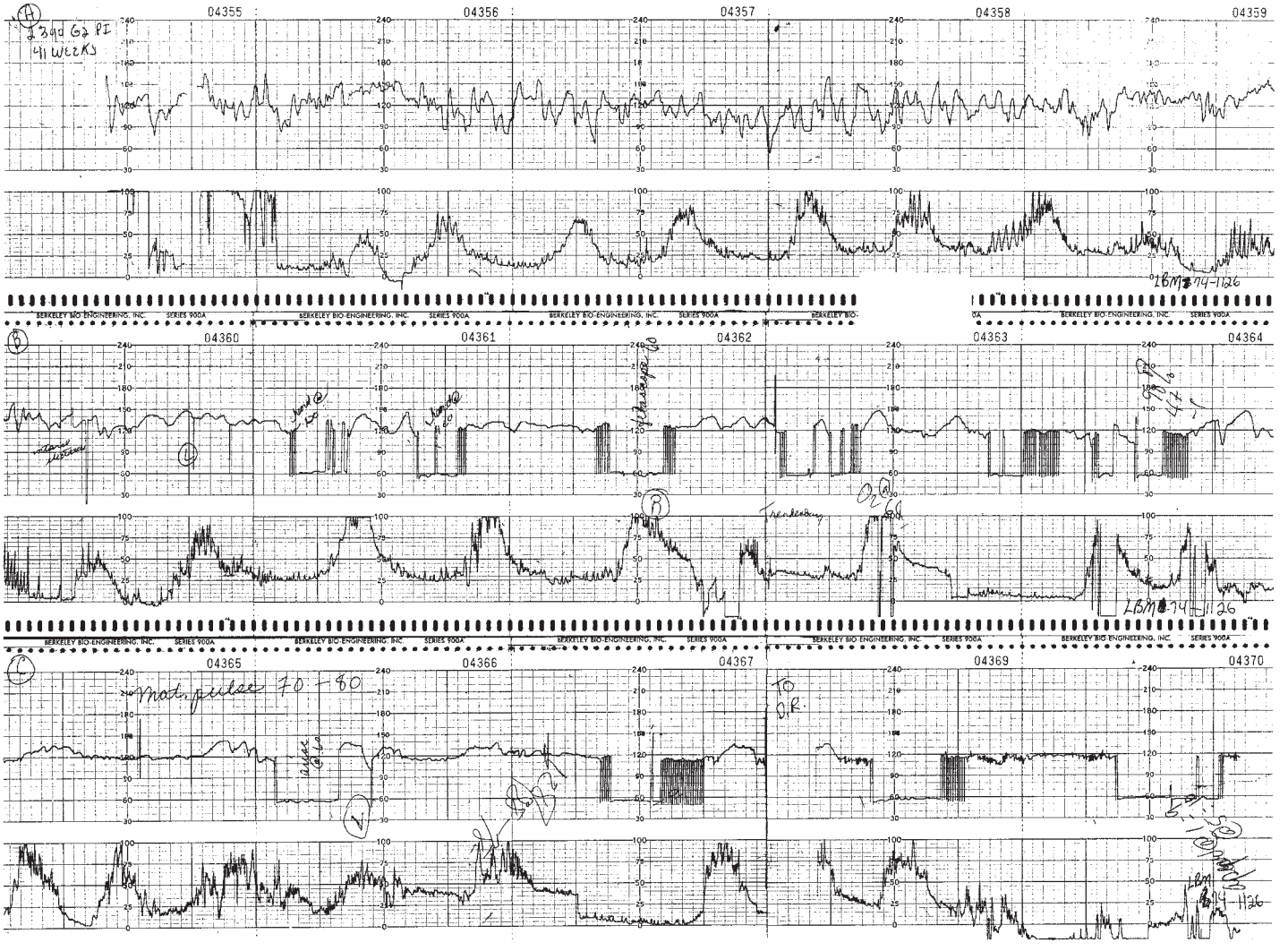

Figure 4.40. An interesting case of fetal heart block. Note that the **first panel**, recorded with a Doppler system containing excessive logic, shows an erratic pattern. The **middle and lower panels** are from a direct fetal scalp electrocardiograph (ECG). There are intermittent abrupt drops to 60 beats per minute (BPM) from the 120-BPM baseline. Where it says "heard at 120 BPM," the nurse was using a Doppler listening device and counting the atrial rate. With the third episode of 60 BPM, the nurse listened with a fetoscope (phone) and the rate of 60 BPM agreed with the ECG of 60 BPM, indicating the ventricular rate. Thus, a diagnosis of an intermittent 2-to-1 heart block was made.

MONITORING FETAL ARRHYTHMIAS

Accurate diagnosis of fetal arrhythmias is very difficult, if not impossible, using the FHR monitor alone. Monitors may have the logic system in operation at all times on the external mode and, therefore, heart rates above 200 BPM will often not even record. Figure 4.41 shows an expanded view of the recording of a fetal arrhythmia when the premature beat prompts a sudden rise and the following pause prompts a sudden drop in the rate, resulting in the characteristic vertical lines associated with the instantaneous FHR recording of fetal arrhythmias. Because electrical noise or maternal electrocardiograph (ECG) artifact can precipitate the same pattern, it is important to examine the raw fetal ECG tracing on the scope to differentiate between arrhythmia and artifact. Other examples of fetal arrhythmias and premature ventricle contractions are displayed in Figures 4.42 through 4.44. Congenital heart block is shown

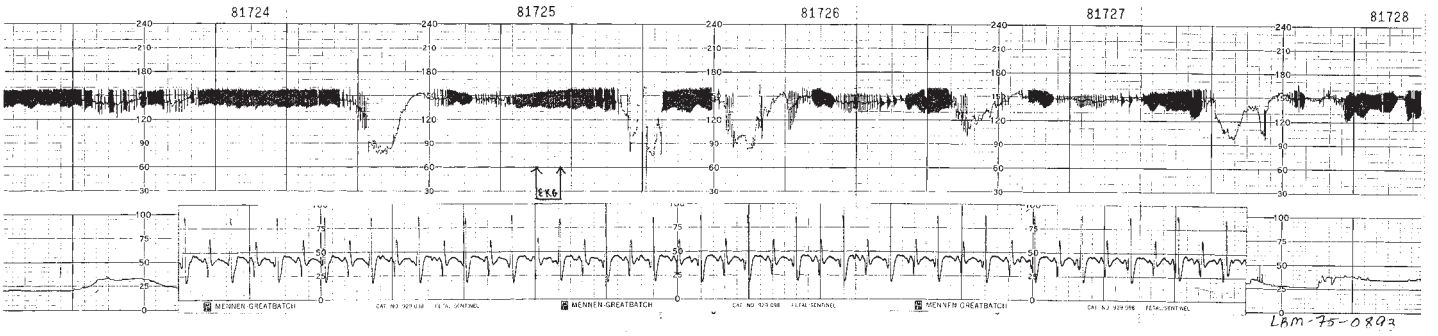

Figure 4.41. This tracing shows the fetal heart rate (FHR) pattern **above** and the simultaneous fetal electrocardiograph (ECG) **below**. The large vertical excursions on the FHR scale are caused by the fetal arrhythmia, which is shown on the fetal ECG tracing to be due to premature multifocal atrial contractions. Note the biphasic P waves.

in Figure 4.45 in a patient with antepartum diagnosis early in the third trimester.

Although when very frequent, continuous monitoring of FHR may be difficult, the clinical significance of these arrhythmias should be appreciated. Premature atrial and ventricular contractions usually have no clinical significance. Although there has been some association with other congenital abnormalities in the cardiac and other organ systems, this occurs infrequently. PACs and PVCs are not to be considered signs of fetal hypoxia and do not carry any significance as far as intervention. We do not recommend a fetal echocardiograph for patients with an antepartum finding of FHR irregularity consistent with PACs or PVCs. However, frequent assessment of FHR to rule out evolution to SVT is recommended. Sometimes, a terminal FHR pattern will show some PACs or PVCs (36), but the significance of the pattern comes from the periodic and baseline FHR characteristics. Transient fetal cardiac standstill should be similarly managed. Position change, examination to rule out cord prolapse or rapid descent, and elevation of the presenting part are suggested maneuvers (37). Figure 4.46 displays an example of transient fetal cardiac asystole mediated by vagal stimulations during a variable deceleration.

One will occasionally see a tracing with all downward deflections. This may be due to dropped beats (Figs. 4.47 and 4.48), a very low amplitude signal, or premature beats where the interval is too short to be counted (<250 milliseconds) and causes only the compensatory pause to be shown. In rare cases, PVCs occur every other beat, creating the bigeminal rhythm noted in Figure 4.44.

The key to interpreting artifacts and arrhythmias is an understanding of fetal monitor instrumentation. The correct diagnosis and appropriate management can only be achieved with careful application of the technologic advances in assessment of the fetal heart.

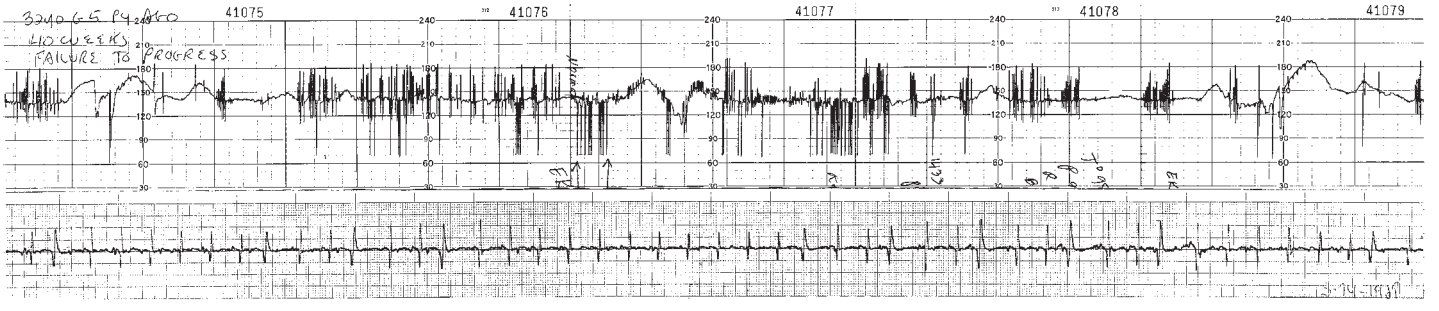

Figure 4.42. This tracing shows the fetal heart rate (FHR) patterns **above** and the simultaneous fetal echocardiograph (ECG) **below**. The large vertical excursions on the FHR scale are caused by the fetal arrhythmia, which is shown on the fetal ECG tracing to be due to premature ventricular contractions. Note the changing configuration of the fetal QRS complexes with each premature beat.

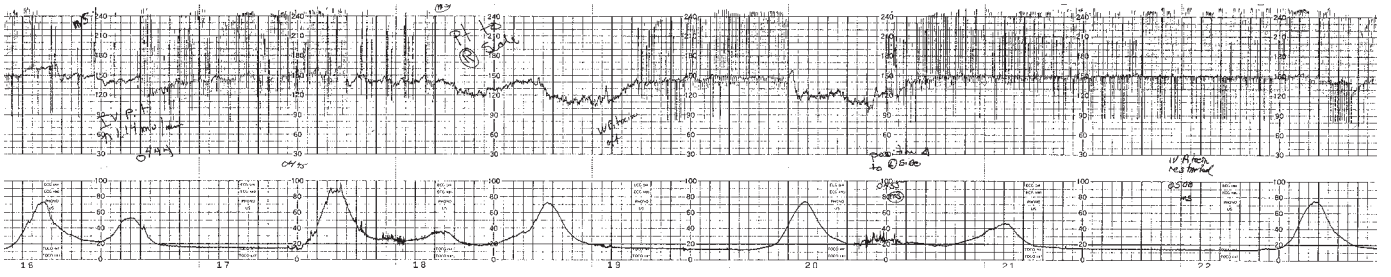

Figure 4.43. This tracing is an example of frequent multifocal premature ventricular contractions. Note the brief appearance of normal sinus rhythm following contractions.

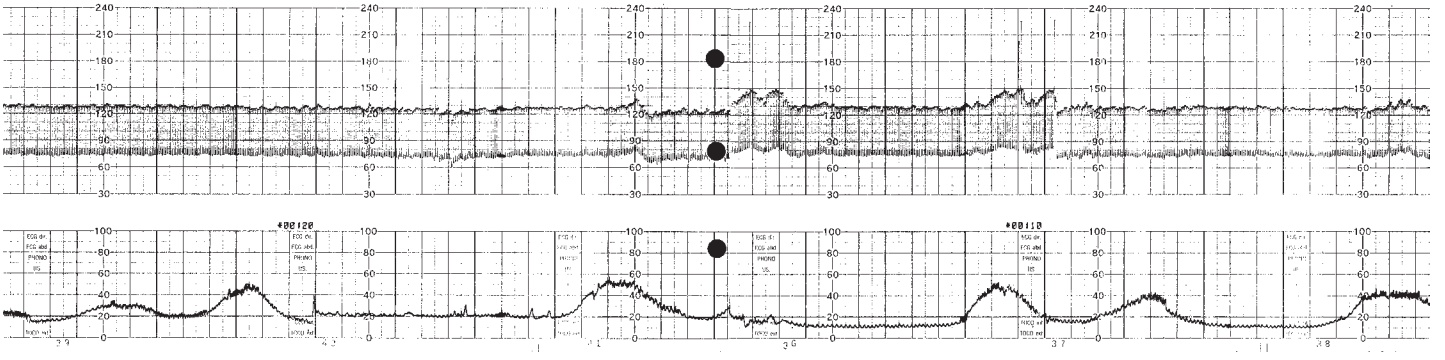

Figure 4.44. This is an example of frequent unifocal premature ventricular contractions. This was noted to be bigeminy on neonatal electrocardiography.

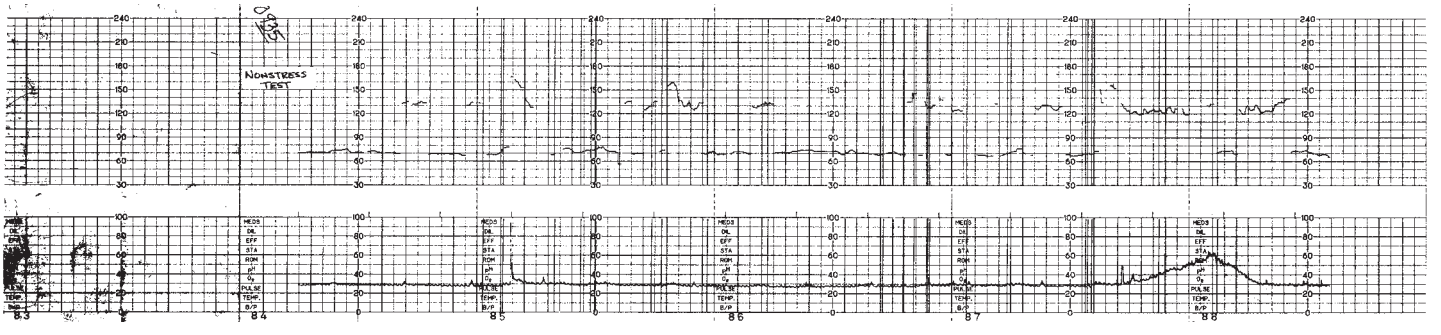

Figure 4.45. This is an example of intermittent congenital heart block detected during routine auscultation at the time of a prenatal visit. A nonstress test revealed areas of reactivity as well as a prolonged period of heart block. Ultrasound failed to reveal any structural cardiac defects or signs of cardiac failure. The patient was observed closely with no evidence of fetal deterioration. She was admitted in active labor at 35 weeks and delivered vaginally a normally grown, apparently healthy newborn. On postpartum day 3, because of frequent prolonged episodes of bradycardia and hypoxia, a pacemaker was placed in the neonate with excellent response. Workup of the mother was negative for any evidence of connective tissue disease.

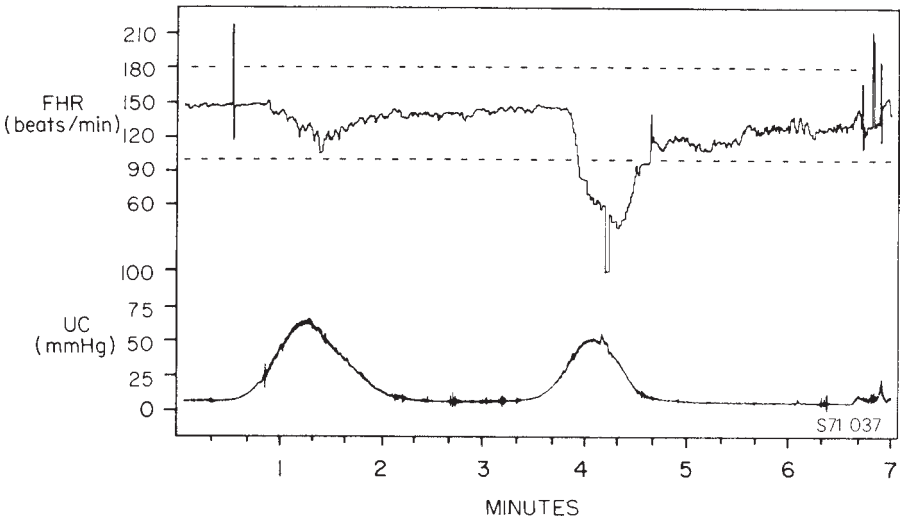

Figure 4.46. An example of a fetal heart rate (FHR)/uterine contraction tracing containing an episode of transient fetal cardiac arrest. At the 4-minute mark, during the course of severe variable deceleration, there was a transient fall of FHR to the zero level. Concomitant examination of the fetal electrocardiograph tracing proved the episode of transient fetal cardiac arrest. (From Yeh S, Zanini B, Petrie RH, et al.: Intrapartum fetal cardiac arrest: a preliminary observation. *Obstet Gynecol* 50:571, 1977, with permission.)

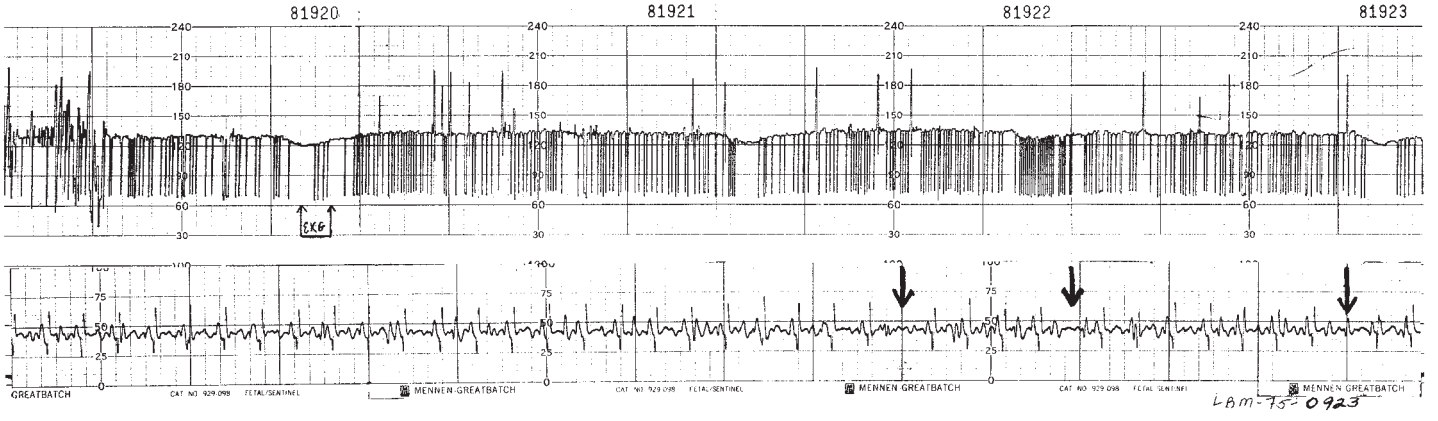

Figure 4.47. This tracing shows a series of downward deflections of the fetal heart rate in the **upper tracing**; the **lower tracing** of the simultaneous fetal electrocardiograph shows absent QRS complexes or dropped beats.

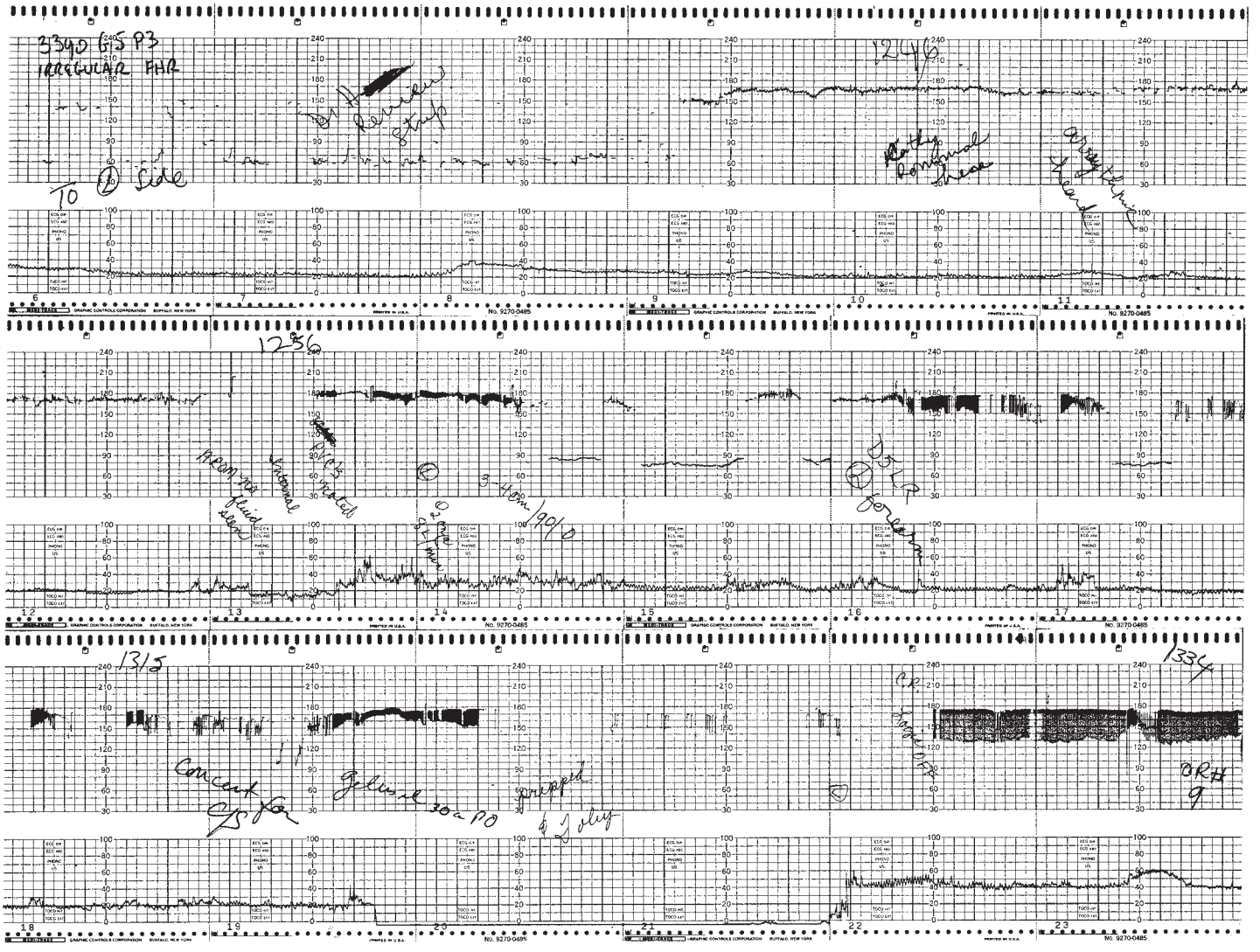

Figure 4.48. Intrapartum fetal arrhythmia evaluation. In the **middle panel**, an external monitor is recording a fetal heart rate of 80 to 90 BPM, most likely representing compensatory pauses of PVCs. In the **third panel**, when the logic is on, the monitor does not record because of the frequent premature beats with "abnormal" R-R intervals.

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CHAPTER 5

Uterine Contraction Monitoring

In discussions on the benefits of fetal monitoring, uterine contraction monitoring is most often ignored. The initial development of fetal heart rate (FHR) monitoring was concerned with the assessment of fetal status. Contraction patterns were included so that the various decelerative patterns could be timed in relation to contractions. With the use of monitors in labor, however, it became apparent that one principal benefit of such routine monitoring was the data it provided relative to uterine activity.

Uterine activity may be assumed to be adequate if progress in labor, as defined by progressive cervical dilatation and descent, is occurring. Failure to progress in labor may be due to inadequate uterine contractions. On the other hand, excessive uterine activity, as in abruptio placentae, may cause inadequate placental perfusion, and thus give rise to fetal hypoxia and acidosis. When it is necessary to induce or augment labor, the clinician must be aware of uterine activity, as overstimulation could lead to fetal compromise or even uterine rupture.

Manual palpation has been the traditional method of monitoring uterine contractions in labor. This method can be used to identify contraction frequency and duration, but it can measure intensity only relatively. It is time-consuming, requires constant evaluation, and provides no permanent record. The effort can be tedious at best, and what occurs in most cases is intermittent manual palpation for short intervals. The contraction monitor provides a tool that significantly improves the accurate monitoring of uterine contractions.

PHYSIOLOGY OF UTERINE CONTRACTIONS

Uterine contractions have as a primary function the expulsion of the intrauterine contents. Uterine activity before the onset of active labor may prepare the uterus and cervix for labor. The uterus is a smooth muscle organ that, during pregnancy, is progressively stretched. Contractions may be a physiologic response to this stretch, perhaps dampened by mechanisms that normally inhibit the premature onset of labor. Two types of preparatory contractions have been described by Caldeyro-Barcia and Poseiro (1). The first are small, weak contractions of short duration, localized to isolated areas of the uterus occurring about once a minute. These high-frequency, low-intensity contractions begin in early pregnancy and seem to disappear near term. They may be related to or may be the same as the localized periodic thickening of the uterine wall frequently seen on routine ultrasound during early gestation (2,3). The second are the better-known Braxton Hicks contractions. These have a higher magnitude of strength (10 to 20 mm Hg), are more generalized, and have a frequency that increases from one contraction per hour at 30 weeks to as often as every 5 to 10 minutes at term. This background frequency is greater with multiple gestations and the vast majority of the time is not perceived by the patient (4). The transition into the regular rhythmical contractions of labor may be insidious or abrupt, and the exact control of this transition remains unknown.

Contractions can be described by frequency, duration, strength (amplitude), uniformity, and shape. During normal labor, the amplitude of contractions increases from an average of 30 mm Hg in early labor to 50 mm Hg in later first stage and 50 to 80 mm Hg during the second stage. The uterus is not a flaccid sac but has baseline tone. At and near term, this baseline tone is generally 8 to 12 mm Hg, with values in excess of 25 mm Hg defined as hypertonus (or, redundantly, baseline hypertonus). The smaller nonpregnant uterus will generate very strong uterine contractions because, according to the law of Laplace, at a given amount of uterine wall tension, the amount of intracavitary pressure is inversely proportional to the radius of the cavity. This is true for the uterus only to a point, however. With excessively large volumes, as in polyhydramnios, uterine tone may begin to rise because of excessive stretching of the muscle fibers (5).

Once actual labor begins, contractions generally become more frequent, coordinated, and stronger. The propagation of uterine contractions is the result of pacemaker-like activity originating usually from the area of the uterotubal junction. For the contraction to fulfill its purpose most efficiently (i.e., expulsion of the fetus via cervical dilation and fetal descent), the contraction must start in the fundus and progressively propagate toward the cervix. Reynolds et al. (6) described this as "fundal dominance," which, simply stated, means that, because of the lesser curvature at the fundus and the greater muscle mass, the strength of contractions is greatest at the fundus and least at the cervix. Caldeyro-Barcia and Poseiro (1) further refined this description. They described a triple descending gradient of wave propagation, intensity, and duration, such that the origin of the contraction is in the fundus and the direction of the contractile wave is toward the cervix. Not only is the contraction more intense in the fundus, but the duration of the contraction is progressively shorter from the fundus to midcorpus to cervix.

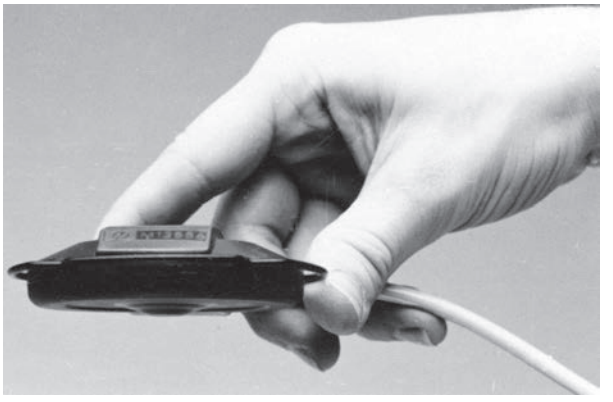

Figure 5.1. Tocodynamometer.

MONITORING OF UTERINE ACTIVITY

External Monitoring

Contractions are most conveniently monitored externally with a tocodynamometer. Mechanical devices for monitoring contractions externally were introduced as early as 1861. Murphy (7) described a ring tocodynamometer and, subsequently, Reynolds et al. (8) used three such instruments on various portions of the uterus to describe normal contraction physiology. The tocodynamometer or "toco" is essentially a weight with a centrally placed pressure-sensitive surface secured to the abdominal wall with a strap (Fig. 5.1). The toco is positioned near the fundus and adjusted to a position that results in the best contraction recording. A sensitivity calibration device, present either on the toco or, more commonly, on the monitor itself, is adjusted to place the resting pressure at 15 to 20 mm Hg to obtain the best tracing. It must always be remembered that the contraction strength is only relatively accurate and varies greatly with maternal position, body habitus, and the tightness of the belt. These factors also affect the sensitivity of the recording. The duration of the contraction will appear to vary: The more sensitive the toco, the longer the apparent duration (Fig. 5.2). With external contraction monitoring, frequency is most accurately, duration less accurately, and intensity least accurately recorded.

The external technique has the advantage of being noninvasive, thus being applicable for patients with intact membranes, that is, the antepartum patient, the patient in premature labor, or the patient during the intrapartum period. There are, however, disadvantages. Patient mobility is limited. Often, the best tracings are obtained with the patient supine. Generally, external monitors are more

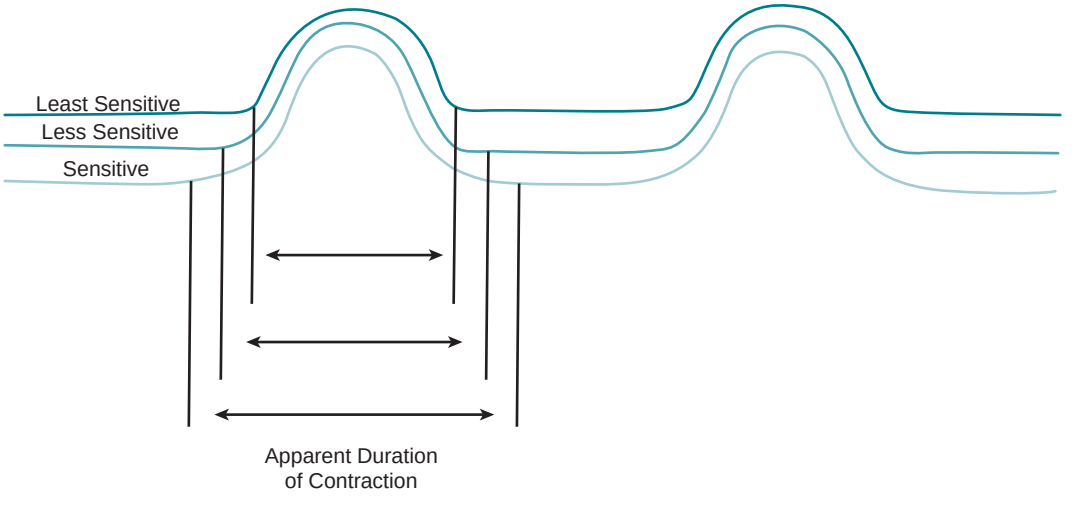

Figure 5.2. This drawing illustrates the correlation between the sensitivity of the contraction-monitoring device and the apparent duration of the contraction. The less sensitive the monitoring technique, the shorter the apparent duration of the contraction.

uncomfortable for the patient. Some patients generate poor to nonexistent tracings of uterine activity. The limitations with regard to intensity and duration have been discussed. At times, when FHR patterns of concern occur and maternal position is changed, loss of a previously good contraction tracing results, thus making FHR decelerations difficult to time or even record due to loss of signal.

Internal Monitoring

According to the law of Pascal, pressure within a fluid-filled closed spheroid is equal at all points. This describes the uterus quite well; therefore, the pregnant uterus is ideal for contraction pressure monitoring. As reported by Williams and Stallworthy (9), as early as 1872 Schatz had used a hydrostatic bag in the lower uterine segment for pressure recording after the membranes had ruptured. In 1927, Bourne and Burn (10) used the hydrostatic bag extraovularly (between the membranes and the lower uterine segment). Alvarez and Caldeyro-Barcia (11) described a transabdominal technique for inserting open fluid-filled catheters in the amniotic cavity to record contractions. Other techniques, including electrohysterography and intramyometrial pressure recording, have been reported. In 1952, Williams and Stallworthy (9) suggested the use of a Drew-Smythe metal cannula (originally designed for high amniotomy) as a guide to introduce a polyethylene tubing transcervically into the amniotic cavity. This was the forerunner for the technique now commonly used for internal contraction monitoring. Currently, sterile disposable kits are available with flexible plastic catheters containing a pressure-sensing device located in the catheter tip (Figs. 5.3 and 5.4). The details of insertion, instrumentation,

Figure 5.3. Internal pressure catheter with pressure-sensing device in tip. There is no need to fill the catheter with fluid when using this technology (Tyco Healthcare-Kendall LTP, Chicopee, MA).

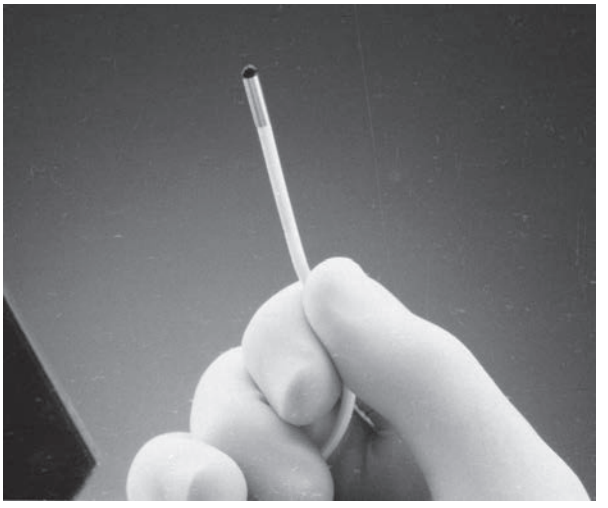

Figure 5.4. Close-up of catheter tip showing pressure sensor (Viggo-Spectramed, Oxnard, CA).

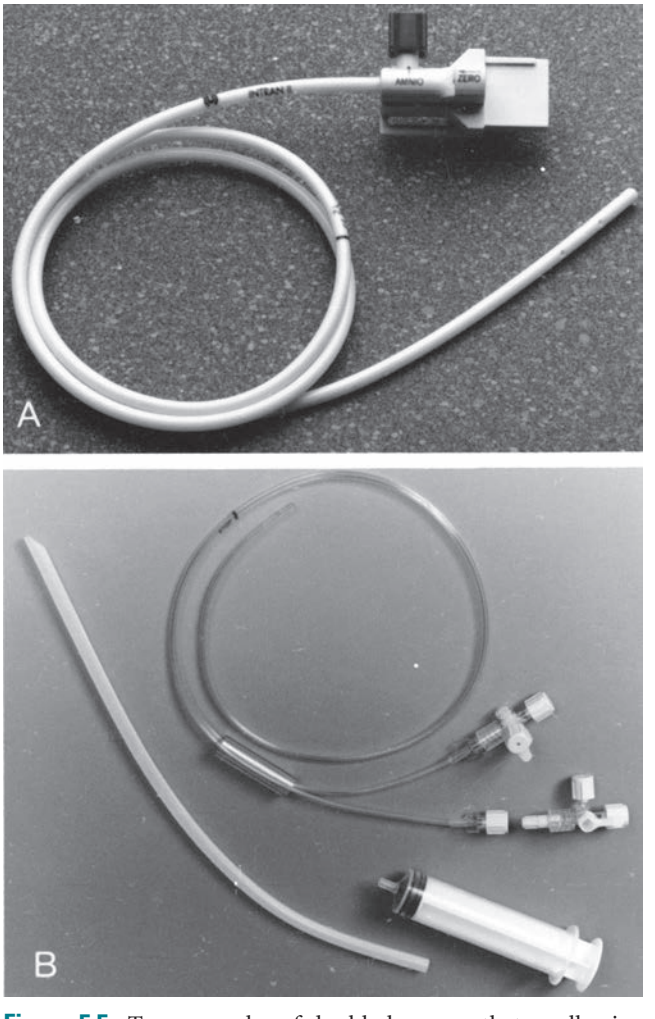

Figure 5.5. Two examples of double lumen catheters allowing simultaneous amnioinfusion and uterine contraction monitoring. A: Catheter with pressure sensor tip (Utah Medical Products, Inc., Midvale, UT). B: Double lumen with fluid column for measuring uterine contractions (Gish Biomedical, Inc., Santa Ana, CA).

and calibration are described in Chapter 4. This method can be used to define and record accurately the frequency, duration, strength, and tonus of the uterus and its contraction. It is less confining and more comfortable for the patient and is generally unaffected by maternal position.

Regarding the value of internal pressure monitoring, the literature is somewhat controversial. The clinician must wait for membrane rupture or perform amniotomy to use internal techniques. There is some evidence that internal monitoring is associated with an increased risk of infection, although these data are often confounded by the fact that many patients with internal pressure monitoring have protracted labors, prolonged rupture of membranes, and frequent pelvic examinations. Uterine perforation has also been described. To avoid this complication, the guide should never be inserted much beyond the edge of the cervix. The alternative to electronic contraction monitoring is manual palpation or reliance on the patient's sensation. Caldeyro-Barcia and Poseiro (1) have said that intrapartum contractions are palpable to the examiner at a minimum pressure of 10 mm Hg and that the patient senses the pain of contractions at a minimum of 15 mm Hg. As with external monitoring, palpation and patient sensation will be reliable with regard to frequency but will be less so for duration and intensity (Figs. 5.5 and 5.6).

QUANTITATION OF UTERINE ACTIVITY

For many reasons, it may be important to quantitate the amount of uterine activity per unit of time. The most practical reason would be to determine, in evaluating poor progress in labor, whether uterine contractions are sufficient. Because failure to progress with a clinically adequate pelvis and inadequate contractions is an indication for labor augmentation, adequate uterine activity must be defined. In 1957, Caldeyro-Barcia et al. (5) defined the Montevideo unit as the product of the average contraction peak in millimeters of mercury multiplied by the number of contractions in 10 minutes. Schifrin (12) defined adequate uterine activity in labor to be greater than 200 Montevideo units. This has been the

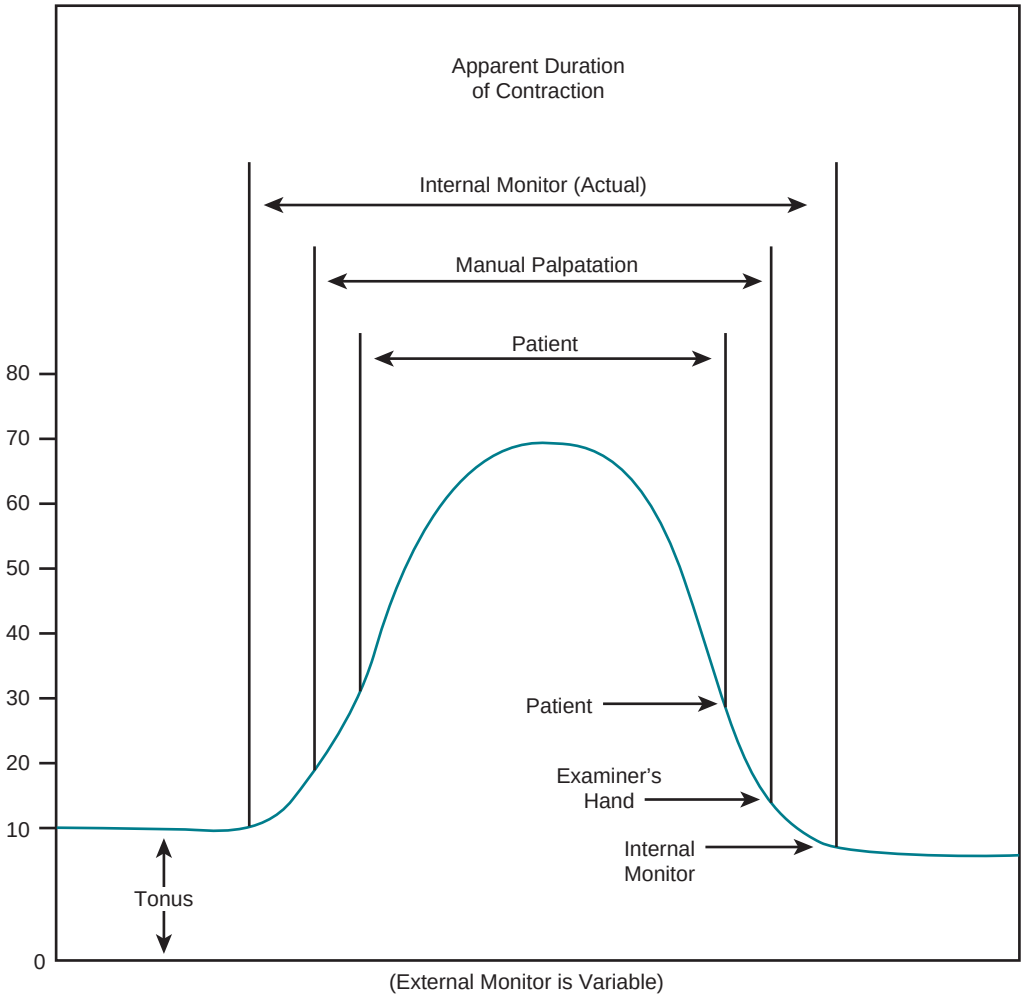

Figure 5.6. Relative sensitivity of various methods available for detection of uterine contractions.

quantitation measure most extensively used by investigators, although it does have the limitation of not including contraction duration in its calculation. To overcome this problem, El-Sahwi et al. (13) defined the Alexandria unit as the average contraction peak amplitude (in millimeters of mercury), multiplied by the average duration (in minutes), multiplied by the average number of contractions in 10 minutes. Both methods are very time-consuming. Miller et al. (14) described a computerized method of quantitating uterine activity by integrating the entire area under the curve (Fig. 5.7). Besides inadequate uterine activity, abnormal rhythmicity or excessive uterine activity may cause problems. These can occur at term in spontaneous or augmented labors or in pregnancies complicated by premature labor, polyhydramnios, or placental abruption. During antepartum contraction stress testing, excessive uterine activity can at times result from breast stimulation or oxytocin administration (Fig. 5.8).

There are two characteristics of these nonsynchronous abnormal contraction patterns. The first is their effect on the progress in labor, and this will depend on cause. Some abnormal patterns may be noted with protracted active phases of labor or secondary arrest of labor (15). In these cases, the patterns may be the result, rather than the cause, of abnormal labor. Other causes may include injudicious oxytocin administration, polyhydramnios, and placental abruption, in which case the effect of these contraction patterns on labor is to shorten it. The other consideration is the effect that these contraction patterns may have on intervillous blood flow, fetal oxygenation, and FHR. Intramyometrial pressure is usually approximately two to three times that of intraamniotic pressure. Mean arterial blood pressure is about 85 to 90 mm Hg in labor. Therefore, the duration that intraamniotic pressure exceeds 30 to 40 mm Hg (corresponding to myometrial pressures in excess of mean arterial pressure) determines how long the maternal spiral arteries are compressed. Another important aspect is how much relaxation time is available for recovery. The effects that these contraction patterns may have on the fetus are most immediately reflected in heart rate and may be manifested by increased variability, delayed (late) decelerations, or prolonged decelerations (Fig. 5.9). Shenker (16) suggested that the most frequent cause of late decelerations is excessive uterine activity. If these excessive contractions and their resultant fetal hypoxia are prolonged, fetal acidemia may result.

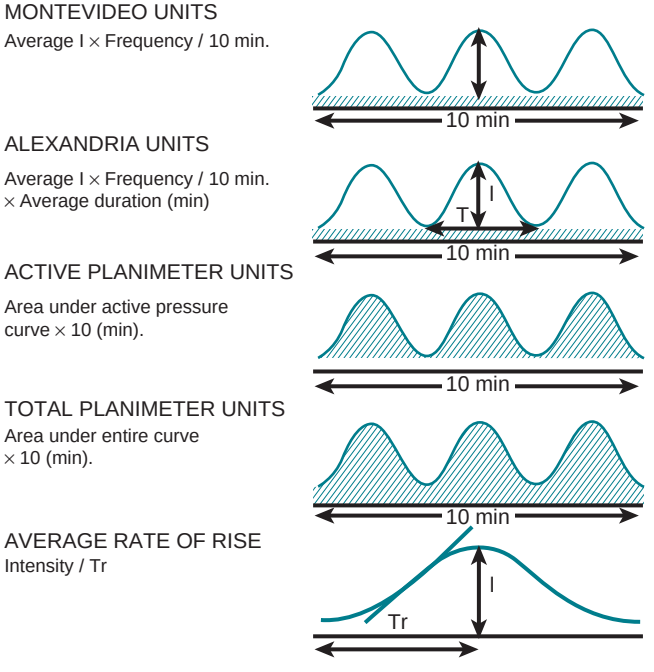

Figure 5.7. Available methods for quantitation of uterine activity. (From Miller FC, Yeh SY, Schifrin BS, et al.: Quantitation of uterine activity in 100 primiparous patients. *Am J Obstet Gynecol* 124:398, 1976.)

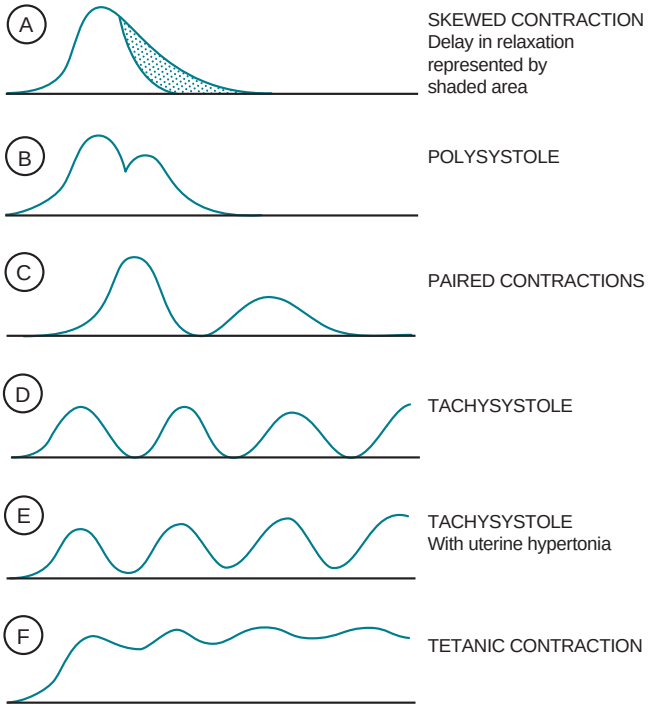

Figure 5.8. Abnormal contraction patterns. (From Stookey RA, Sokol RJ, Rosen MJ: Abnormal contraction patterns in patients monitored during labor. *Obstet Gynecol* 42:359, 1973.)

OTHER FACTORS AFFECTING UTERINE CONTRACTION

To understand and correct abnormal contraction patterns and heart rate reactions to them, it is important to be aware of the intrinsic and extrinsic factors that affect uterine contractility. These factors may manifest themselves by

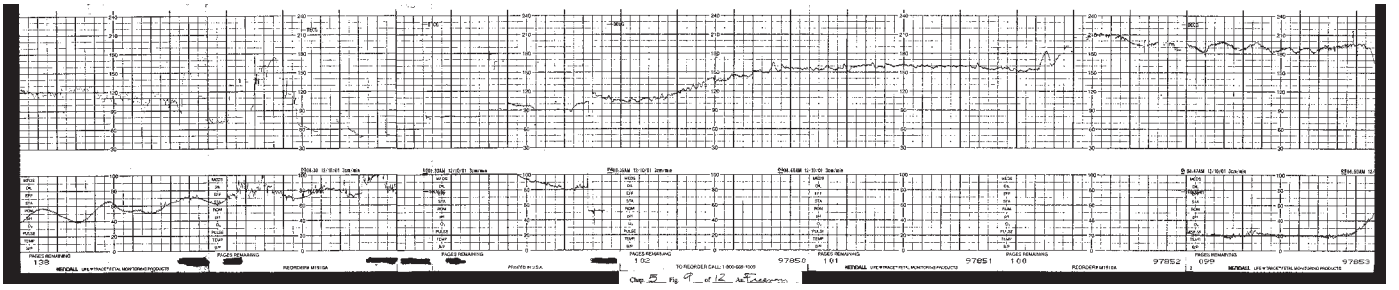

Figure 5.9. Note the changes of the fetal heart rate following a tetanic uterine contraction. In this example, there is a temporary rise in the baseline heart rate with subsequent return to a normal baseline and evidence of late decelerations.

decreasing or increasing contraction strength, frequency, or both. Intrinsic factors include pathologic state and maternal position. The most common diseases that alter uterine contractions include polyhydramnios, preeclampsia, abruption, and chorioamnionitis. Abruption usually causes the greatest degree of hyperactivity (Fig. 5.10). Polysystole, tachysystole, hypertonus, or any form of increased uterine activity may be seen. With abruptio placentae, it may be the uterine hyperactivity, loss of placental surface area, or both that result in changes in FHR that are often in evidence. Even in patients without complaints of severe pain or evidence of vaginal bleeding, the finding of frequent spontaneous contractions, often without return to baseline, in the presence of abnormal FHR patterns makes the diagnosis of abruption a strong consideration. With pregnancies complicated by preeclampsia or eclampsia, Alvarez et al. (17) have pointed out that uterine tonus is unaffected but that frequency and intensity of contractions are often increased. Polyhydramnios has a variable effect on tonus until the uterus is severely stretched and baseline tone is often low to low normal. However, if the hydramnios worsens, a hypertonus may develop. Uterine contractions may be quite prolonged with hydramnios (Fig. 5.11).

Position has a relatively consistent effect on uterine activity. It is clear that uteroplacental perfusion is poorer in the supine position than in either lateral position. Caldeyro-Barcia et al. (18) have shown that, generally, when the patient is turned from her back to her side, contractions become stronger and less frequent (Fig. 5.12). Finally, maternal pushing efforts during labor can add 20 mm Hg or more to the recorded intensity of contractions. They are usually seen as rapid, brief elevations of intrauterine pressure superimposed on the uterine contraction (Fig. 5.13).

There has always been conjecture and debate as to whether oxytocin reproduces a physiologic contraction pattern. Much of this debate stems from the fact that many clinicians use dose rates far in excess of physiologic values. Caldeyro-Barcia et al. (19), Caldeyro-Barcia and Poseiro (20), and Poseiro and Noriega-Guerra (21) have shown that oxytocin both accelerates and coordinates uterine contractions, and that at physiologic doses of 1 to 8 mU/minute, this occurs without elevation of tonus. These studies have shown no difference between contractions, with or without oxytocin, with data from intraamniotic pressure, intramyometrial pressure, and electrohysterography. Alvarez and Cibils (22) have shown that both types of contractions have

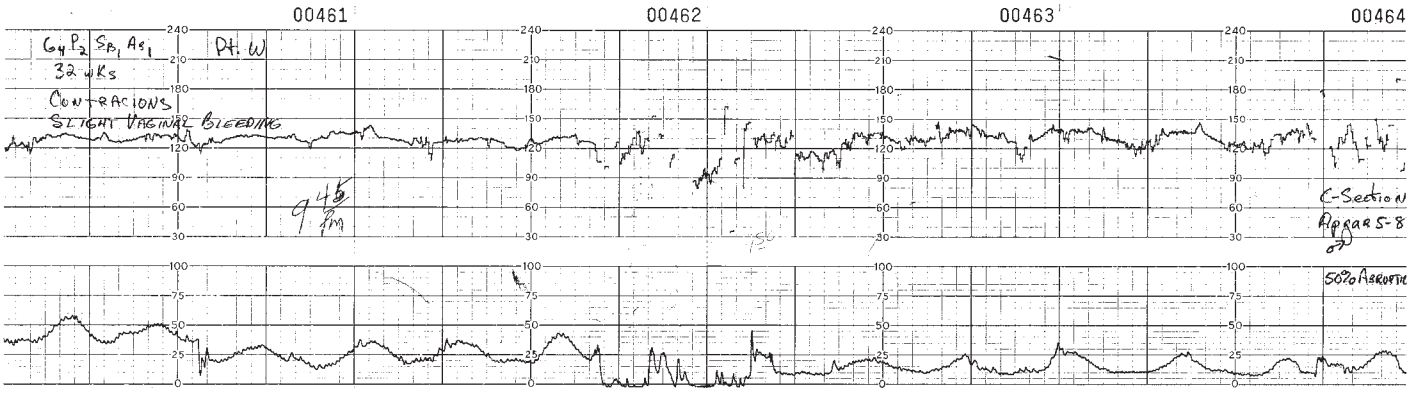

Figure 5.10. This example of uterine hyperactivity in the form of tachysystole was seen in a patient in premature labor with vaginal bleeding. In this case, it is very difficult to interpret the occurrence of late decelerations. Both fetal heart rate and contractions are being monitored externally. The cause of uterine hyperactivity and late decelerations was a 50% abruptio placentae.

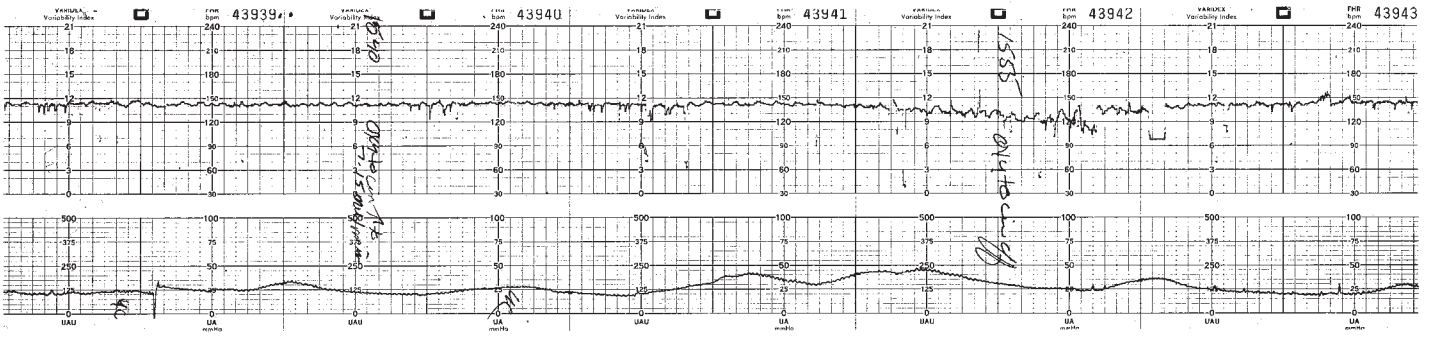

Figure 5.11. Prolonged uterine contraction seen in an externally monitored diabetic with polyhydramnios. Labor is being augmented with oxytocin. Such prolonged contractions in response to oxytocin are commonly seen with polyhydramnios.

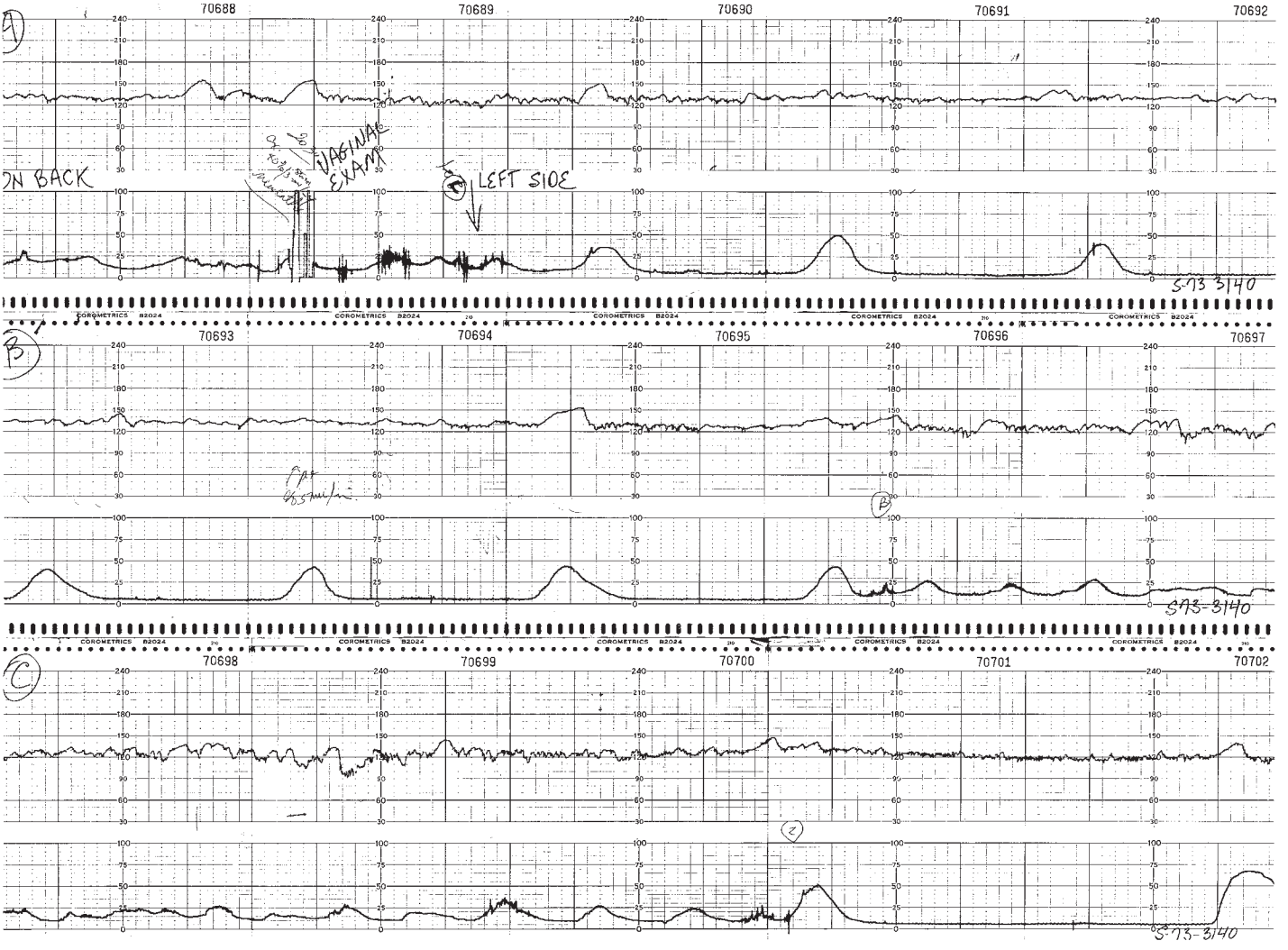

Figure 5.12. In **panel A**, frequent uterine contractions are occurring. When the patient is turned to her left side, the contractions become less frequent and increase in strength. In **panel B**, the patient has turned and is lying on her back. The contractions have become more frequent and smaller. Finally, again with the patient on her left side in **panel C**, the contractions are spaced out as before in **panel A**.

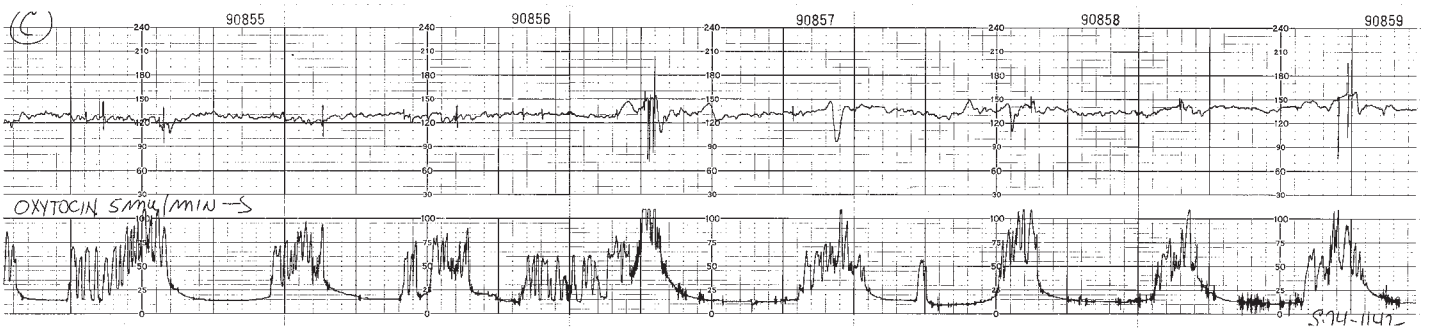

Figure 5.13. Small spikes (intermittent short elevations of pressure) seen on top of uterine contractions represent maternal pushing.

equivalent efficiency in producing cervical dilatation. When doses of oxytocin exceed physiologic requirements, however, all forms of hyperactivity as well as hypertonus can be seen. Seitchik and Castillo (23) report that the majority of uterine hypercontractility episodes can be avoided by a slow increase (intervals not less than 30 minutes) in oxytocin of 1.0 mU/minute from an initial rate of 1.0 mU/minute. Steady progress in labor, as defined by progressive cervical dilatation, occurs in 95% of patients with an infusion rate less than or equal to 6 mU/minute. Other protocols using a more aggressive use of oxytocin have been proposed for labor augmentation (24–26). Termed "active management of labor," these approaches use oxytocin in a more rapidly increasing dosage and achieve much higher total amounts of drug being administered than with the protocol suggested by Seitchik and Castillo. The success in labor augmentation with oxytocin may be more related to the timing of initiation and achieving of an adequate labor pattern than the actual amount used. Many other drugs affect uterine activity. The more common ones are listed in Table 5.1.

TABLE 5.1 Drugs affecting uterine contractions

Stimulating drugs
Acetylcholine
Ergonovine
Estrogen
Norepinephrine
Oxytocin
Propranolol
Prostaglandins
Quinine
Sparteine sulfate
Vasopressin
Inhibiting drugs
β-Sympathomimetics
Ritodrine, Orciprenaline, Isoxsuprine, Salbutamol, Fenoterol, etc.
Calcium channel blockers
Nitroglycerin
Atosiban
Halothane
Magnesium sulfate
Progesterone
Prostaglandin synthetase inhibitors

SUMMARY

The study of fetal monitoring is incomplete without detailed knowledge of uterine activity and its effects on fetal oxygenation and heart rate. Most significant periodic changes in the FHR occur at or after the time of the uterine contraction. The intent of this chapter has been to describe the details of contraction monitoring and the physiologic mechanisms of labor so that the reader might better be able to integrate this knowledge and understand the physiologic and pathophysiologic basis of fetal monitoring.

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CHAPTER 6

Basic Pattern Recognition

The evaluation of the fetus during labor by electronic fetal heart rate (FHR) monitoring is a complex process. Patterns that are known to have an excellent correlation with normal oxygenation and a vigorous baby at birth have been identified and are highly reliable. These must be distinguished from patterns that suggest suboptimal oxygenation so that action may be taken to reverse the problem, find alternatives to correct the abnormal pattern, or intervene operatively before damage or death can occur. Many factors must be weighed to determine the significance of the pattern. The FHR is evaluated for baseline rate, variability, and presence of accelerations or decelerations, as well as the progression of each component over time. Contraction frequency and strength must be considered. The patient's parity, her rate of progress in labor, the estimated time of delivery, and maternal and obstetric complications are all factored into this rather complex equation.

Quantifying various parameters of fetal well-being by means of mathematical and statistical computations of the FHR is difficult. This is because the interpretation of FHR tracings is both a study of pattern recognition and a process of evaluating multiple clinical and heart rate variables in determining the status of fetal oxygenation. Previous chapters have dealt with the basic understanding of the physiology and technology of electronic FHR monitoring. This chapter is devoted to pattern recognition.

Efforts to arrive at universal nomenclature and criteria for various FHR patterns have not been uniformly successful. In 1997, a workshop convened by the National Institute of Child Health and Human Development (NICHD) published a document in an attempt to create more consistent and uniform terminology and guidelines for interpreting FHR patterns (1). At that meeting, the participants found there were only two situations where there was universal agreement with respect to the significance of FHR patterns. The first was the pattern with normal baseline rate and variability and no periodic decelerations. It was agreed that such patterns were consistent with normal oxygenation and there were no requirements for intervention with respect to fetal well-being. The second patterns that were agreed to by the group as being consistent with hypoxia sufficient to cause death or damage were those patterns with repetitive late or variable decelerations or bradycardia all of which were accompanied by absent FHR variability. Any patterns between these two extremes could not gain general agreement from the group with respect to significance or management (1).

In 2008, the Eunice Kennedy Shriver National Institute of Child Health and Human Development partnered with the American College of Obstetricians and Gynecologists and the Society for Maternal–Fetal Medicine to sponsor a workshop focused on electronic FHR monitoring (2). This workshop gathered a diverse group of investigators with expertise and interest in the field to accomplish three goals: (a) to review and update the definitions for FHR pattern categorization from the prior workshop, (b) to assess existing classification systems for interpreting specific FHR patterns and make recommendations about a system for use in the United States, and (c) to make recommendations for research priorities for electronic fetal monitoring (EFM). A complete clinical understanding of EFM necessitates discussion of uterine contractions, baseline FHR rate and variability, presence of accelerations, periodic or episodic decelerations, and the changes in these characteristics over time. A number of assumptions and factors common to FHR interpretation in the United States are central to the proposed system of nomenclature and interpretation. Two such assumptions are of particular importance. First, the definitions are primarily developed for visual interpretation of FHR patterns but should be adaptable to computerized systems of interpretation. Second, the definitions should be applied to intrapartum patterns but also are applicable to antepartum observations.

Uterine contractions are quantified as the number of contractions present in a 10-minute window, averaged over

a 30-minute period. Contraction frequency alone is a partial assessment of uterine activity. Other factors such as duration, intensity, and relaxation time between contractions are equally important in clinical practice.

Listed as follows is terminology used to describe uterine activity:

- *Normal*: five contractions or less in 10 minutes, averaged over a 30-minute window
- *Tachysystole*: more than five contractions in 10 minutes, averaged over a 30-minute window

Characteristics of uterine contractions

- The terms hyperstimulation and hypercontractility are not defined and should be abandoned.
- Tachysystole should always be qualified as to the presence or absence of associated FHR decelerations.
- The term tachysystole applies to both spontaneous and stimulated labor. The clinical response to tachysystole may differ depending on whether contractions are spontaneous or stimulated.

The following are EFM category definitions and descriptions based on the 2008 NICHD Working Group findings. Decelerations are defined as recurrent if they occur with at least one-half of the contractions.

CLASSIFICATION OF FETAL HEART RATE TRACINGS

A variety of systems for EFM interpretation have been used in the United States and worldwide. Based on careful review of the available options, a three-tiered system for the categorization of FHR patterns is recommended (see box). It is important to recognize that FHR tracing patterns provide information only on the current acid-base status of the fetus. Categorization of the FHR tracing evaluates the fetus at that point in time; tracing patterns can and will change. An FHR tracing may move back and forth between the categories depending on the clinical situation and management strategies used.

NICHD 2008 Terminology
Three Tier FHR Interpretation System

Category I: Normal FHR Pattern
Baseline rate 110–160 BPM
Baseline FHR variability: moderate (5–25 BPM)
Late and variable decelerations absent
Early decelerations present or absent
Accelerations present or absent
Category II: Equivocal FHR Patterns
Baseline Rate and Variability
Baseline rate: Bradycardia (<110 BPM) not accompanied by absent variability
Tachycardia (>160 BPM)
Variability minimal (=or<5 BPM but present)
Variability absent without recurrent decelerations
Marked baseline variability (>25 BPM)
Periodic Changes
Absence of induced accelerations after fetal stimulation
Recurrent variable decelerations accompanied by minimal or moderate baseline variability
Prolonged deceleration >2 min but <10 min
Recurrent late decelerations with moderate baseline variability
Variable decelerations with other characteristics such as slow return to baseline or overshoot
Category III: Abnormal FHR Patterns
Absent FHR variability *and* of the following:
Recurrent late decelerations
Recurrent variable decelerations
Bradycardia
Sinusoidal pattern

Category I FHR Tracings are Normal

Category I FHR tracings are strongly predictive of normal fetal acid-base status at the time of observation. Category I FHR tracings may be monitored in a routine manner, and no specific action is required.

Category II FHR Tracings are Indeterminate

Category II FHR tracings are not predictive of abnormal fetal acid-base status, yet presently there is not adequate evidence to classify these as Category I or Category III. Category II FHR tracings require evaluation and continued surveillance and reevaluation, taking into account the entire associated clinical circumstances. In some circumstances, either ancillary tests to ensure fetal well-being or intrauterine resuscitative measures may be used with Category II tracings.

Category III FHR Tracings are Abnormal

Category III tracings are associated with abnormal fetal acid-base status at the time of observation. Category III FHR tracings require prompt evaluation. Depending on the clinical situation, efforts to expeditiously resolve the abnormal FHR pattern may include but are not limited to provision of

maternal oxygen, change in maternal position, discontinuation of labor stimulation, treatment of maternal hypotension, and treatment of tachysystole with FHR changes. If a Category III tracing does not resolve with these measures, delivery should be undertaken. We attempt to follow these new guidelines for nomenclature in this edition of this book.

PATIENT IDENTIFICATION

Many modern monitoring systems have electronic storage of patient information and EFM tracings. These systems require patient data entry before starting the monitor on a new patient. However, many hospitals continue to store paper records for backup medical records, for teaching purposes, or because some information (e.g., continuous fetal pulse oximetry data) may not be stored on the digital record. Therefore, it is important to appropriately label the paper record as well. Monitors should be identified numerically and tracings labeled accordingly. Should a technical problem occur, it can be traced to the correct monitor. Also, different monitors may have different logic and other technical characteristics, and when a facility has various brands, the clinician can better interpret the tracing by taking the make and model into consideration.

BASELINE FETAL HEART RATE

The normal baseline FHR ranges from 110 to 160 beats per minute (BPM). The NICHD defined baseline FHR as the mean FHR during a 10 minute window. The mean is expressed in increments of 5 BPM. Changes of shorter duration are called "periodic changes." While it is appropriate to attempt to create a defined duration of a periodic change, for which a more sustained rate becomes a baseline change, this is often inconsistent with what is the obvious physiologic explanation for a prolonged periodic change. For example, a profound hypotensive episode that results in a prolonged deceleration will ultimately return to the original baseline when the insult is reversed. Thus the deceleration may have lasted longer than 10 minutes and, according to the previous NICHD definition, would now be called a bradycardia, but it was truly not a baseline change. Similarly, a fetus that is unusually active may have an acceleration that lasts considerably longer than 10 minutes but returns to the original baseline when the fetus returns to a more quiet state.

TABLE 6.1 Causes of fetal tachycardia

Fetal hypoxia
Maternal fever
Parasympatholytic drugs
Atropine
Hydroxyzine hydrochloride (Atarax or Vistaril)
Phenothiazines
Maternal hyperthyroidism
Fetal anemia
Fetal sepsis
Fetal heart failure
Chorioamnionitis
Fetal cardiac tachyarrhythmia
β-Sympathomimetic drugs

Fetal Tachycardia

Fetal tachycardia is defined as a baseline heart rate in excess of 160 BPM. Factors associated with or causing tachycardia are listed in Table 6.1. Because tachycardias represent increased sympathetic and/or decreased parasympathetic autonomic tone, they are generally associated with a loss of variability (Fig. 6.1). Most fetal tachycardias do not reflect a hypoxic fetus, particularly when present in a term gestation. In a preterm

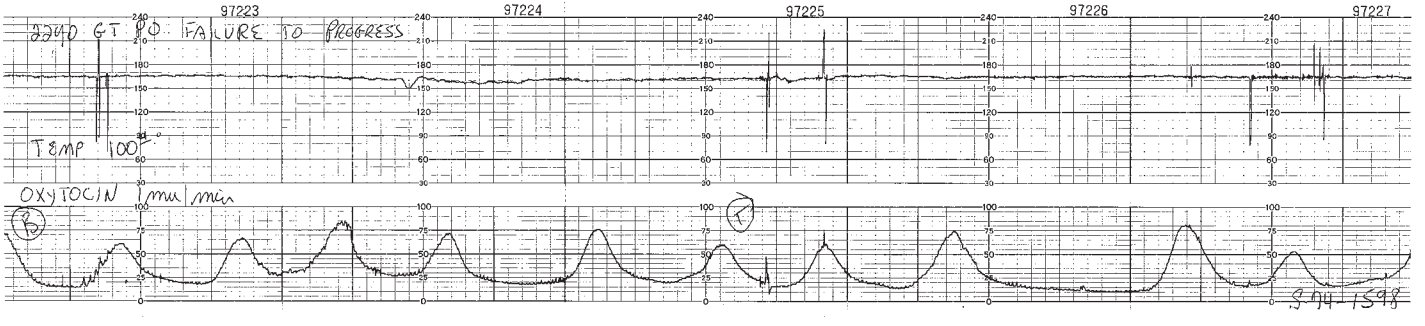


Figure 6.1. Fetal tachycardia, fetal heart rate 165 beats per minute. This tachycardia is associated with maternal fever (note temperature [100.4°F]). Also note the associated loss of variability. The absence of associated decelerations and presence of an explanation (fever) makes hypoxia an unlikely cause.

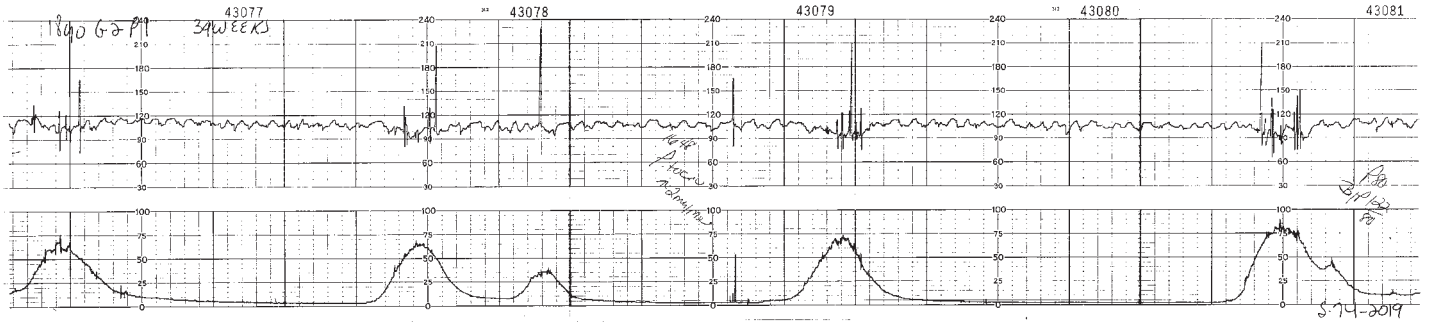


Figure 6.2. Fetal bradycardia. The fetal heart rate is 110 beats per minute. There is normal variability present by direct internal scalp electrode monitoring. Four hours later, the patient delivered a 3,025 g baby with Apgar scores of 9 at 1 minute and 10 at 5 minutes. Mother and baby did well.

fetus or when seen in a term pregnancy without an obvious explanation (e.g., maternal fever), close evaluation is critical.

Fetal Bradycardia

Bradycardia is a baseline FHR of <110 BPM Bradycardia, within the range of 80 to 110 BPM with moderate variability generally indicates good oxygenation without acidemia (Fig. 6.2). Slowing of the baseline heart rate is most likely in response to an increased vagal tone (3). Fetal bradycardia that is first noted at the initiation of monitoring may be difficult to distinguish from a prolonged deceleration. Generally, prolonged decelerations are associated with loss of variability and their rate fluctuates up and down rather than remaining consistent, unless rates below 70 BPM are seen.

Actual baseline FHRs of <70 BPM are generally seen without variability and may represent congenital heart block (Fig. 6.3). Persistent bradycardia from complete atrioventricular dissociation should alert the clinician to the possible diagnosis of maternal connective tissue disease, most often systemic lupus erythematosus, which results in fetal heart block and bradycardia consequent to transplacental passage of maternal antibodies that have an affinity for fetal cardiac conduction fibers (4) (see Chapter 4). Structural cardiac defects and cytomegalovirus infections are also potential etiologies of congenital heart block. To reliably establish that fetal cardiac activity is present and that the persistent bradycardia is due to fetal heart block, confirmation with real-time ultrasound is necessary. Ultrasound allows for a further advantage in that cardiac structure can be carefully evaluated as well (see Chapter 7). However, ultrasound is really only an option in the antepartum period because it may not be possible to take the time required to make this evaluation in labor where either a bradycadia or prolonged deceleration may require urgent operative intervention. When the diagnosis of complete fetal heart block is made antenatally, the problem then becomes how to monitor the fetus in labor because the normal alterations in FHR in response to hypoxrmia and other central nervous system (CNS) input cannot be transmitted to the ventricles. Thus, in this situation, the FHR cannot be used as a way to monitor for fetal hypoxemia and to rule out acidemia. Three options are reasonable in this situation. Intermittent scalp pH is theoretically an option, but the need for repeating the test every 30 minutes makes this option unrealistic except in very rapid labors. In the past, an elective cesarean section was often undertaken, simply because there had been no other way to monitor the fetus. Although no longer available in the United States, fetal pulse oximetry has

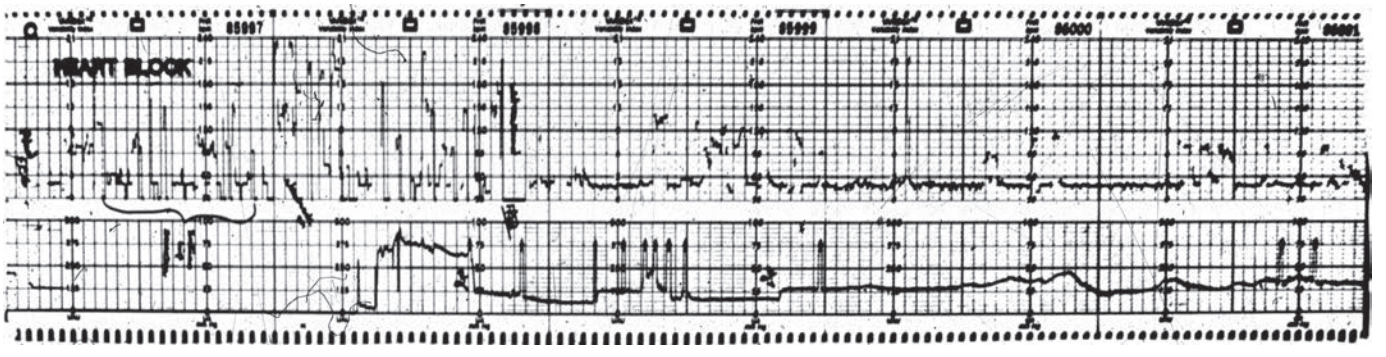


Figure 6.3. Fetal bradycardia due to complete heart block. Note the rate of 50 beats per minute with lack of variability.

provided a logical option, and limited data suggest that this is a safe and effective way of monitoring patients whose fetal arrhythmia prevents adequate FHR monitoring (5).

Bradycardia with moderate variability may occur in the second stage and is not indicative of abnormal fetal acid-base status. Other, more rare causes of fetal bradycardia are maternal hypothermia, prolonged hypoglycemia, beta-blocker therapy, and fetal panhypopituitarism with brainstem injury (6). Persistent bradycardia requires careful evaluation to ensure that it is not the maternal heart rate being recorded in the presence of a fetal demise or secondary to signal ambiguity. Real-time ultrasound can establish the correct diagnosis and thus avoid unnecessary and potentially dangerous therapy in the presence of an already dead fetus. Maternal heart rate can also be recorded from the Doppler transducer and in the rare situation where the fetus is dead the MHR may be recorded from the scalp electrode (see Chapter 4).

Variability

In the 2008 NICHD update on nomenclature, FHR variability was classified as follows:

Absent
Minimal (1 to 5 BPM)
Moderate (5 to 25 BPM)
Increased (>25 BPM)

In determining the immediate fetal status, the most important single FHR characteristic is variability. Moderate variability (6 to 25 BPM peak to trough) is a reflection of intact neurologic modulation of the FHR and of intact or normal cardiac responsiveness. The two components are short-term and long-term variability (LTV) (Fig. 6.4). Short-term variability (STV) is the beat-to-beat irregularity caused by the normal variance in intervals between consecutive cardiac cycles. It is a consequence of the constant "push-pull" effect of sympathetic and parasympathetic nervous system input. LTV is the waviness of the FHR tracing, generally at a frequency of 3 to 5 cycles per minute. With older EFM systems, using external Doppler, FHR variability could not be accurately determined because Doppler monitoring can artifactually increase FHR variability due to the imprecision of the signal. With newer Doppler signal processing systems, however, variability can be accurately assessed. Although there is utility in distinguishing between LTV and STV in research settings because mathematical quantification of these two types are different, there is no current evidence that the distinction between the two has any clinical relevance. Perhaps the single exception to this is the sinusoidal tracing that is absent short term but uniformly increased LTV. Increased variability has been shown in animals to be a sign of mild hypoxia (7). However, when FHR variability is moderate or increased, fetal pH is usually normal.

As gestation advances and the fetal autonomic nervous system matures, the baseline FHR decreases and both STV and LTV increases. This is thought to reflect an increase in vagal control of cardiovascular reflexes. Decreased variability may be seen with prematurity, fetal sleep cycles or anything that causes fetal CNS depression. Fetal hypoxemia with acidemia is the most worrisome cause; however, any condition that depresses the CNS can also decrease variability (Table 6.2). In labor it is critical to carefully review the strip for the presence or absence of late, variable, or prolonged decelerations when decreased variability is seen. It is highly unlikely that minimal or absent variability reflects CNS depression due to metabolic acidemia unless there are decelerations indicating ongoing hypoxemia. The exception to this rule may occur in the fetus who presents in labor with a absent variability without decelerations, as one cannot rule out an hypoxic insult prior to the initiation of electronic monitoring. Especially in the absence of significant decelerations, other causes of CNS depression should be considered. Certain drugs may be responsible (Table 6.3), particularly drugs that depress the CNS (Fig. 6.5) and drugs with autonomic blocking effects. Parasympathetic blocking drugs

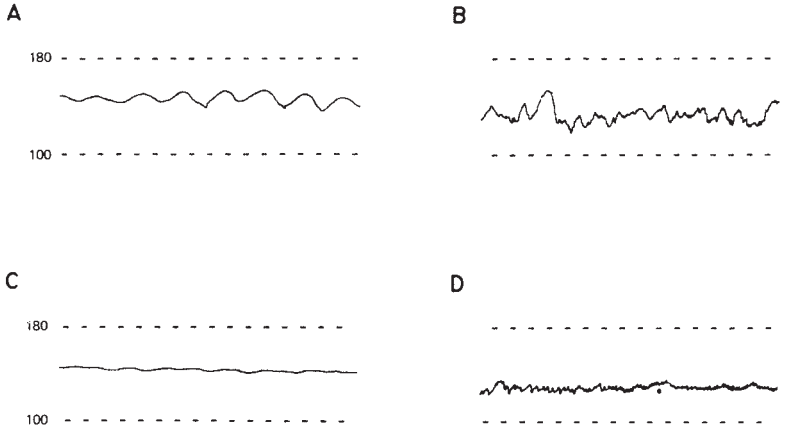


Figure 6.4. Long-term variability (LTV) is demonstrated in **A** and **B** and is absent in **C** and **D.** Short-term variability (STV) alone is shown in **D** and its concurrent presence with LTV is shown in **B.** Absence of both LTV and STV is seen in **C.** (From Zanini B, Paul RA, Huey JR: *Intrapartum fetal heart rate: correlation with scalp pH in the preterm fetus.* Am J Obstet Gynecol 136:43, 1980, with permission.)

TABLE 6.2 Causes of decreased FHR variability

Hypoxia/acidosis
Drugs CNS depressants Parasympatholytics
Fetal sleep cycles
Congenital anomalies
Extreme prematurity
Fetal tachycardia
Preexisting neurologic abnormality

CNS, central nervous system, FHR, fetal heart rate.

decrease variability while increasing baseline heart rate. Sympatholytic drugs (e.g., beta-adrenergic blockers) also decrease variability but decrease baseline heart rate.

Baseline heart rate variability is also associated with fetal wakefulness. When the fetus is sleeping, there may be decreased variability (Fig. 6.6). Usually the variability will spontaneously return to its previous level after a reasonable time, although the duration of fetal sleep states in labor is not as consistent as in the antepartum period. Stimulation of the fetus by manipulation of the uterus or noise may arouse the fetus and cause the variability to return or result in a FHR acceleration, both of which reliably predict the absence of abnormal fetal academia. The new onset of loss of variability in the absence of periodic FHR decelerations with contractions is not a sign of fetal hypoxia.

From an intrapartum monitoring perspective, the most ominous cause of decreased variability is fetal hypoxemia where FHR decelerations precede the loss of variability. In the presence of heart rate patterns, such as persistent late decelerations, loss of variability is associated with a high incidence of fetal acidosis and low Apgar scores (8) (Fig. 6.7), particularly in the case of preterm infants (9). A most difficult heart rate pattern to interpret is absent variability seen in the fetus with a normal baseline heart rate and no decelerations. This may represent a previous insult to the fetus that has since been corrected but has resulted in persistent neurologic damage. Also, this pattern may be seen in fetuses with significant congenital anomalies, especially of the central nervous and cardiac systems. Extreme prematurity is associated with decreased variability and nonreactive FHR patterns. Finally, these patterns can be idiopathic and occur in a subsequently vigorous and healthy neonate (10). Table 6.3 lists the causes of decreased variability.

TABLE 6.3 Examples of drugs causing decreased fetal heart rate variability

Analgesics/narcotics Demerol Heroin Nisentil Morphine
Barbiturates Phenobarbital Secobarbital
Tranquilizers Diazepam
Phenothiazines Largon Phenergan
Parasympatholytics Atropine
General anesthetics

PERIODIC CHANGES

Tachycardia, bradycardia, and alterations in variability involve baseline FHR changes. Periodic changes are transient heart rate accelerations or decelerations that ultimately return to the original or a newly established baseline heart rate. Generally, these periodic changes occur in response to contractions and may also be seen with fetal movement.

Decelerations

There are four principal types of decelerations named according to shape and temporal relationship to contractions. These are early, late, variable and prolonged decelerations.

Early Decelerations

Early decelerations are uniformly shaped decelerations of gradual onset and gradual return to baseline (Fig. 6.8). They begin early in the uterine contraction cycle, have their nadir at the peak of the contraction, and return to baseline before completion of the contraction. Acceleration of the heart rate generally does not precede or follow early deceleration. An important characteristic of early deceleration is the minimal amplitude. The degree of FHR slowing is generally proportional to the strength of the contraction but rarely falls below 100 to 110, or 20 to 30 BPM below baseline. Early decelerations are thought to be caused by fetal head compression, with altered cerebral blood flow precipitating cardiac slowing through a vagal reflex. Early deceleration is generally seen

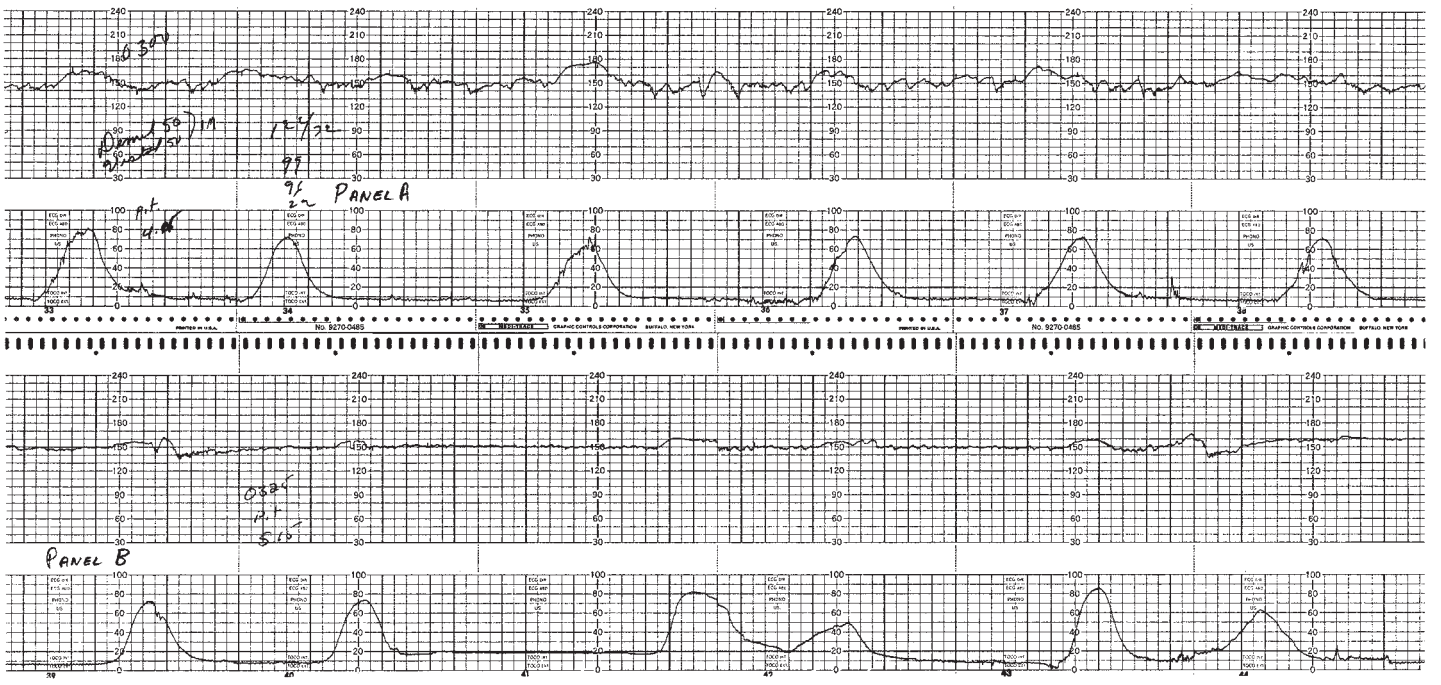


Figure 6.5. Narcotics are among the most frequent causes of decreased fetal heart rate variability in labor. In **A,** Demerol and Vistaril are given intramuscularly. At the beginning of **B,** about 20 minutes later, there is a noticeable decrease in variability without a change in baseline heart rate.

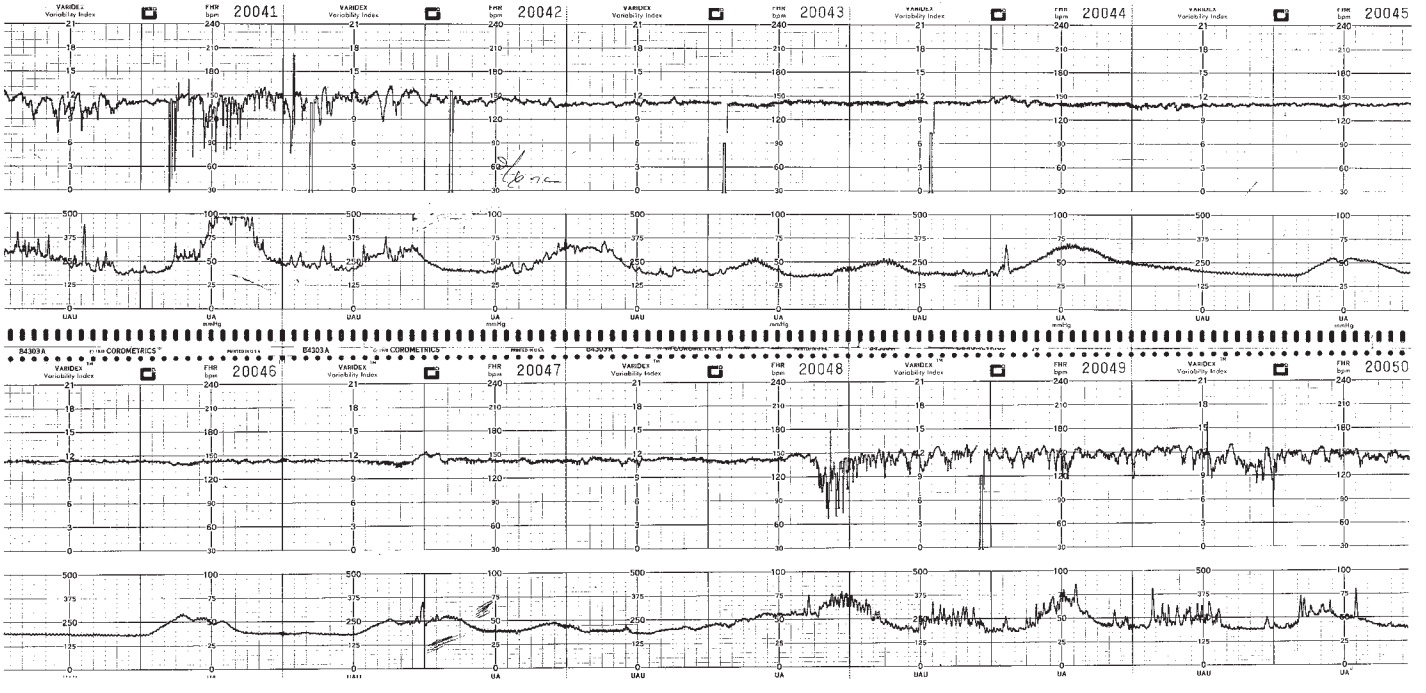


Figure 6.6. Spontaneous changes in variability occur normally in labor. Note the abrupt decrease in variability at **panel 20042**, which again abruptly returns to normal at **panel 20048**. This decreased variability lasted 20 to 30 minutes. There were no medications used. A vigorous normal baby was subsequently delivered.

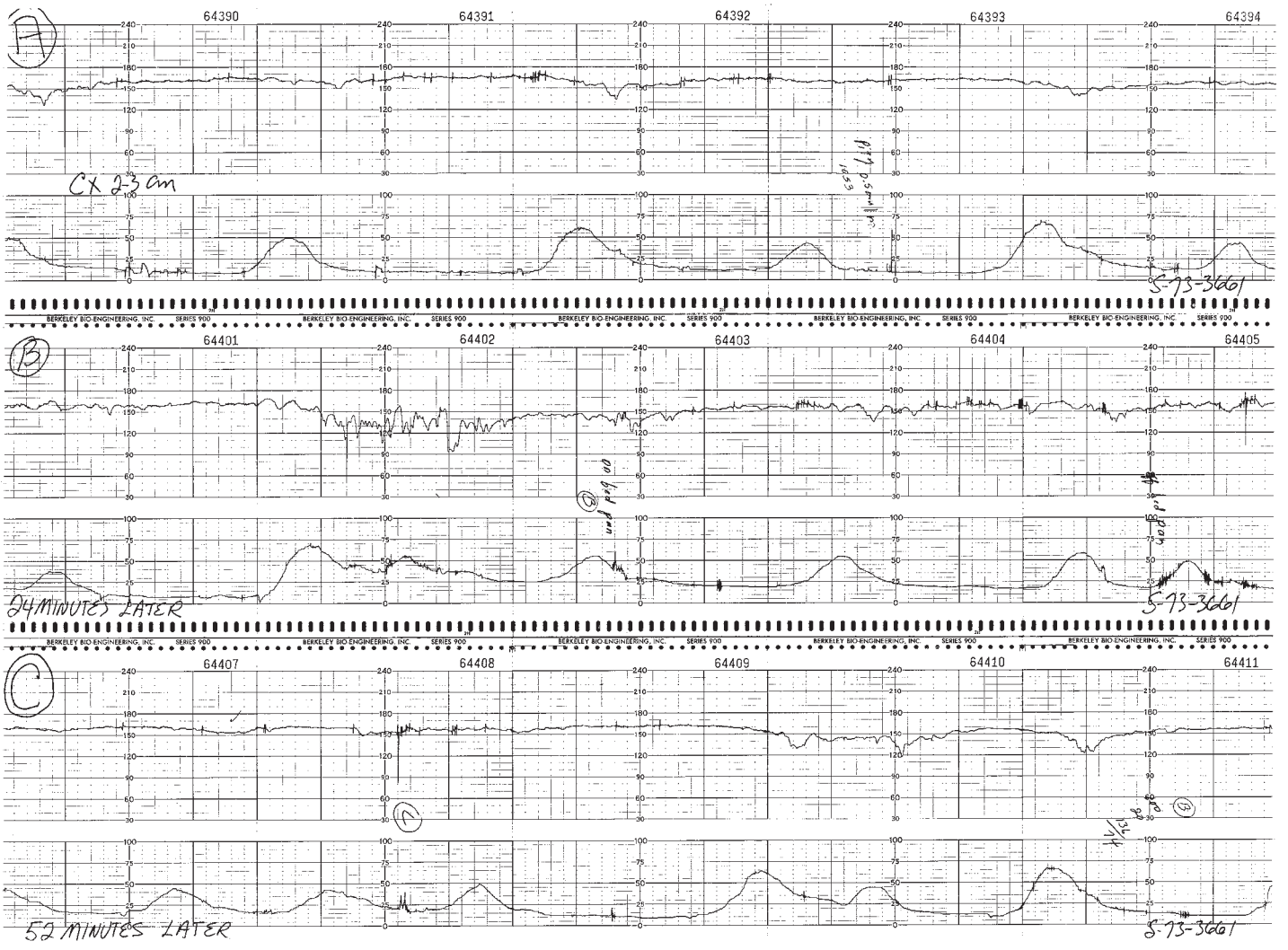


Figure 6.7. Persistent late decelerations are seen after most contractions. At **panel 64407** and after, variability is notably decreased. There is an associated fetal tachycardia.

in the active phase of labor, between 4 and 7 cm of cervical dilation. It is not associated with tachycardia, loss of variability, or other heart rate changes. Early deceleration is a FHR pattern and that is not associated with fetal hypoxia, acidosis, or low Apgar scores.

Late Decelerations

In shape and uniformity, late decelerations are similar to early decelerations, but the timing is delayed relative to the uterine contraction (Fig. 6.9). The onset of the deceleration is often seen 30 seconds or more after the onset of the contraction.

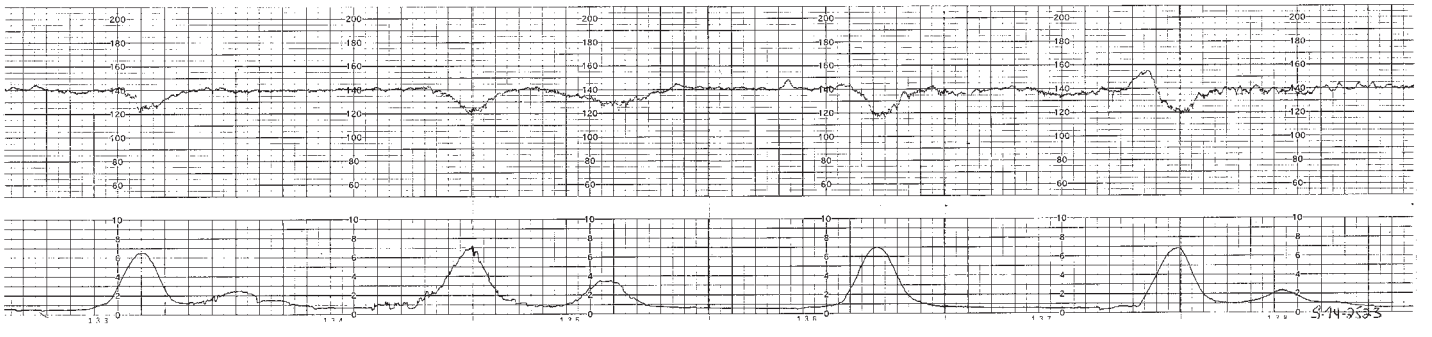


Figure 6.8. Early decelerations are seen with each contraction on this panel. They are uniform, mirror the contractions, and decelerate only 10 to 20 beats per minute.

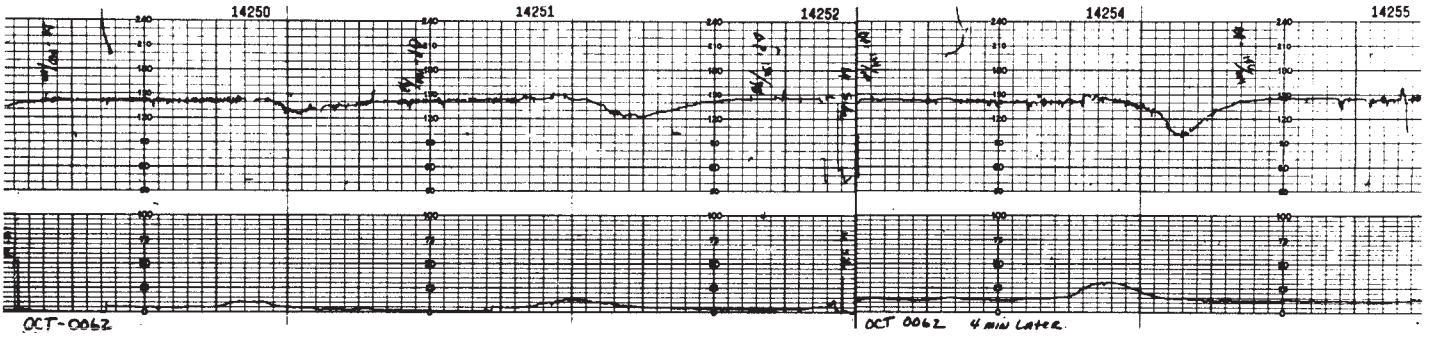


Figure 6.9. Late decelerations are seen after each of the three contractions. They are uniform, smooth, and drop only 20 to 30 beats per minute below baseline. There are no associated accelerations.

The nadir of the deceleration occurs after the contraction peak, and usually the return to baseline occurs after the contraction is over. In recognizing late decelerations, several important characteristics in addition to the timing are important. The descent and return are gradual and smooth. There are usually no accelerations seen preceding or following the deceleration. The FHR rarely falls more than 30 to 40 BPM below baseline and usually not by more than 10 to 20 BPM. Late decelerations are significant and concerning when they are persistent and not correctable. Variability is often increased during the deceleration. Late decelerations may occur before fetal metabolic acidemia develops and in this case they are caused by vagal stimulation. After fetal metabolic acidosis develops the late decelerations are due to fetal myocardial depression (see Chapter 2). Although late decelerations may be associated with hypoxia, acidemia, and hypotension, only a decrease in fetal oxygen tension is essential for late decelerations to occur. The degree of this hypoxia and the appearance of late decelerations are related to many factors, including, on occasion, the strength and duration of the uterine contraction (Fig. 6.10). There may be a correlation between the magnitude of late decelerations (amount of slowing) and the degree of hypoxia, but this is not always the case, as the most depressed fetuses may have only shallow late decelerations. Persistent late decelerations are significant and potentially ominous. The association of late decelerations with loss of variability and/or elevation of baseline FHR is of more significance than the decelerations alone and reflects fetal intolerance to the hypoxic stress of uterine contractions.

The cause of late decelerations is uteroplacental insufficiency (UPI) elicited by intervillous stasis occurring during uterine contractions, and the factors which lead to this hypoxia may be intrinsic or extrinsic to the placenta. Decreased uterine blood flow is a much more common cause of late deceleration than poor exchange from other causes. Causes of decreased flow include supine hypotension and decreased uterine artery perfusion secondary to epidural or spinal anesthesia. The most common cause of late deceleration is uterine hyperactivity or hypertonus, often as a result of excessive oxytocin stimulation. Late decelerations seen in association with vaginal bleeding and/or spontaneous uterine hyperactivity may be the result of premature placental separation (abruptio placentae) (Fig. 6.11). Several disease states, including chronic hypertension, postmaturity, intrauterine growth restriction, diabetes mellitus, pregnancy-induced hypertension/preeclampsia, and collagen vascular disease, may compromise placental exchange. The decreased intervillous blood flow associated with contractions (labor) may further aggravate this exchange and produce late decelerations. Because each contraction can produce hypoxic stress, the persistence of late deceleration may precipitate metabolic acidosis. The most important two variables to watch for at this point are loss of accelerations and the loss of variability (Fig. 6.12). Tachycardia may also occur with the

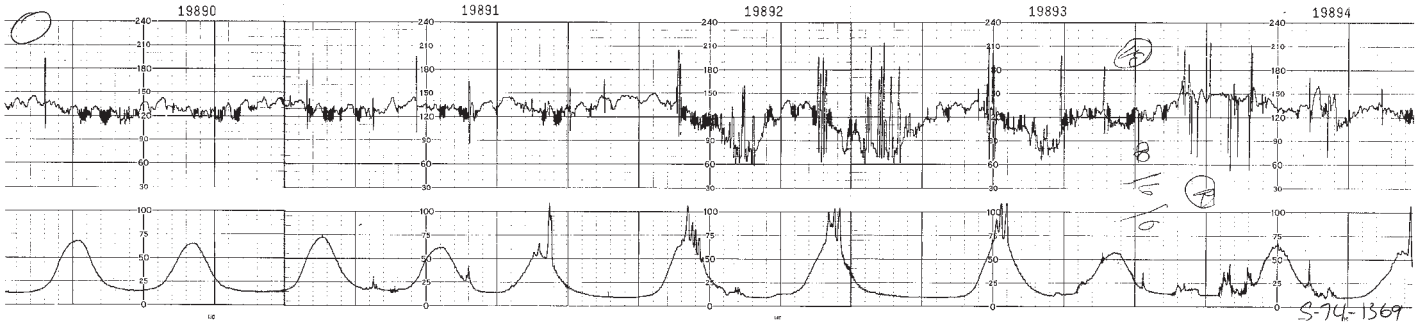


Figure 6.10. Late decelerations are seen occasionally only with the stronger contractions. In this panel, late decelerations are seen only with those contractions that exceed 70 mm Hg (internal pressure catheter and electrode).

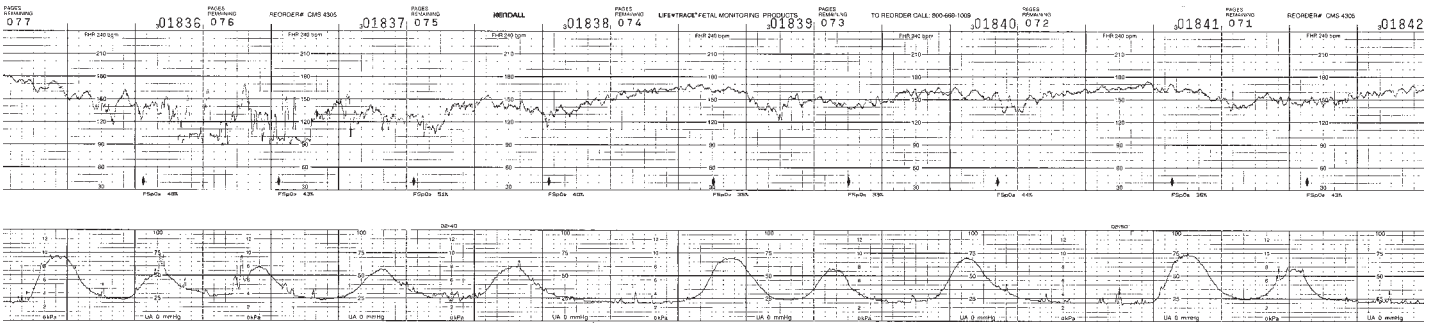


Figure 6.11. Late decelerations are seen after each contraction in this externally monitored patient, a term gestation admitted with vaginal bleeding. Note also the frequent/tachysystolic contraction pattern seen with the first five contractions. At cesarean section, a large abruption was found subsequent to delivering a 3,500 g female with Apgar scores of 7 at 1 minute and 8 at 5 minutes.

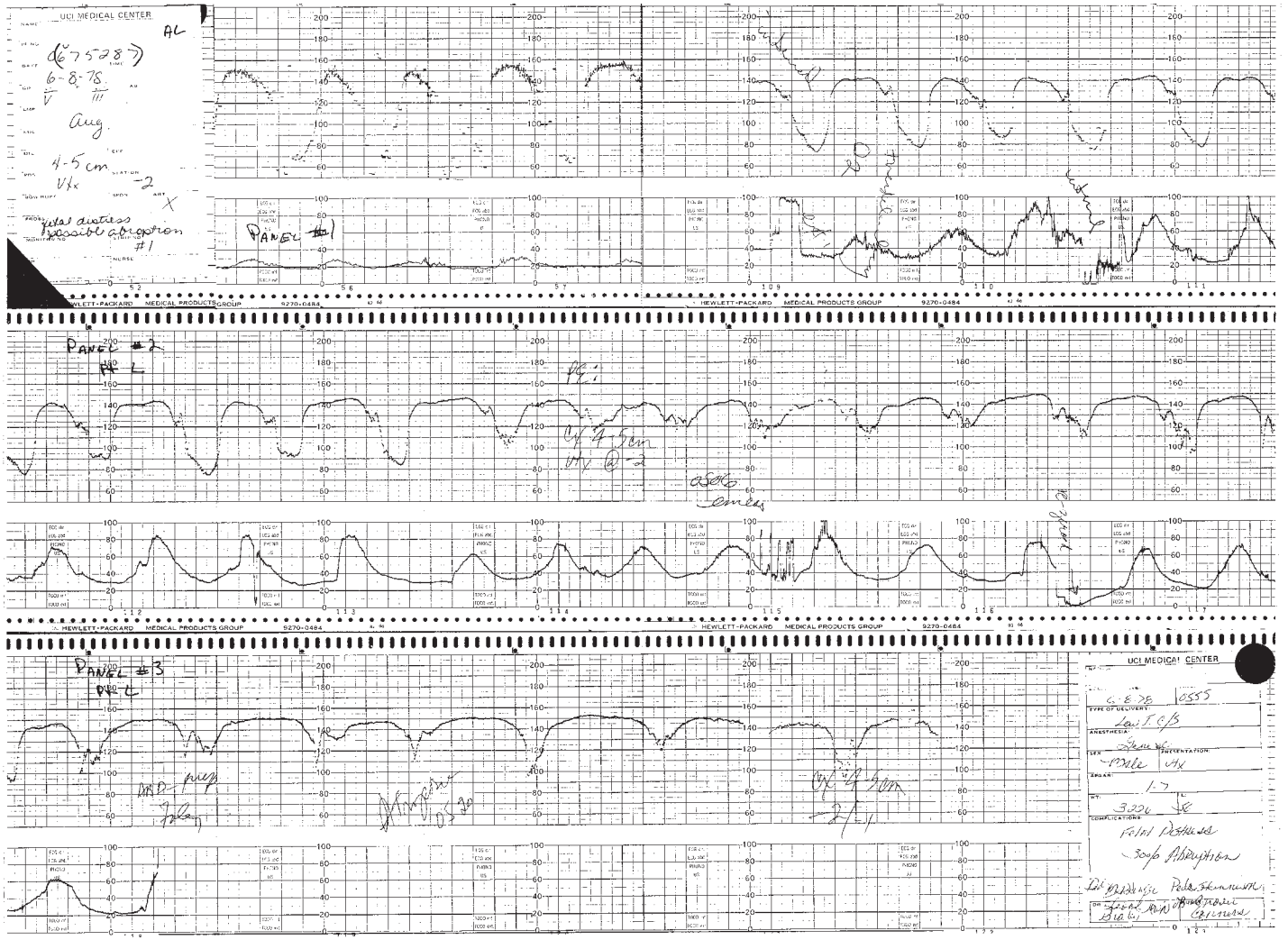


Figure 6.12. This is a gravida 5, para 3 admitted at term with contractions and minimal vaginal bleeding. On internal monitor (second half of **panel 1**), persistent late decelerations are noted that fail to respond to oxygen and position change. Variability is poor throughout and the baseline heart rate gradually increased from 140 to 155 beats per minute. In the presence of late decelerations, poor variability and rising baseline rate are signs of fetal intolerance to hypoxia and developing acidosis. In this case, a female with Apgar scores of 1 at 1 minute and 7 at 5 minutes was delivered. The 30% abruption is the cause of the late decelerations.

development of fetal acidosis, although for unknown reasons, this is not as consistently seen as it is with progressively severe variable decelerations. Decreased variability and fetal tachycardia are important signs of developing acidosis, and their presence with persistent late decelerations correlates highly with neonatal depression.

Variable Decelerations

The most frequently seen FHR deceleration pattern in labor is variable decelerations. This aptly named pattern is variable in nearly all respects: shape, duration, intensity, and timing relative to uterine contractions. It is commonly the result of umbilical cord compression but can result from any interruption of umbilical blood flow that is acute and intermittent. Other causes of interruption of cord flow include cord stretch and cold (e.g., rapid infusion of room-temperature amnioinfusion). In addition, head compression may also produce or alter the shape, depth, and duration of variable decelerations. Because cord compression during labor occurs most often during uterine contractions, variable decelerations usually coincide with uterine contractions (Fig. 6.13). This is, however, an inconsistent occurrence, and such decelerations may be seen with one but not the subsequent contraction. Characteristically, these decelerations are very abrupt in both onset and return to baseline. Small abrupt accelerations of the FHR usually precede and/or follow these decelerations. Variable decelerations are also occasionally observed during antepartum monitoring with fetal movement. There appears to be an association between the presence of variable decelerations on antepartum monitoring and both oligohydramnios and subsequent abnormal FHR tracings in labor. The degree of oligohydramnios correlates with the frequency of severe variable decelerations in labor (11). Assessment of amniotic fluid volume should be

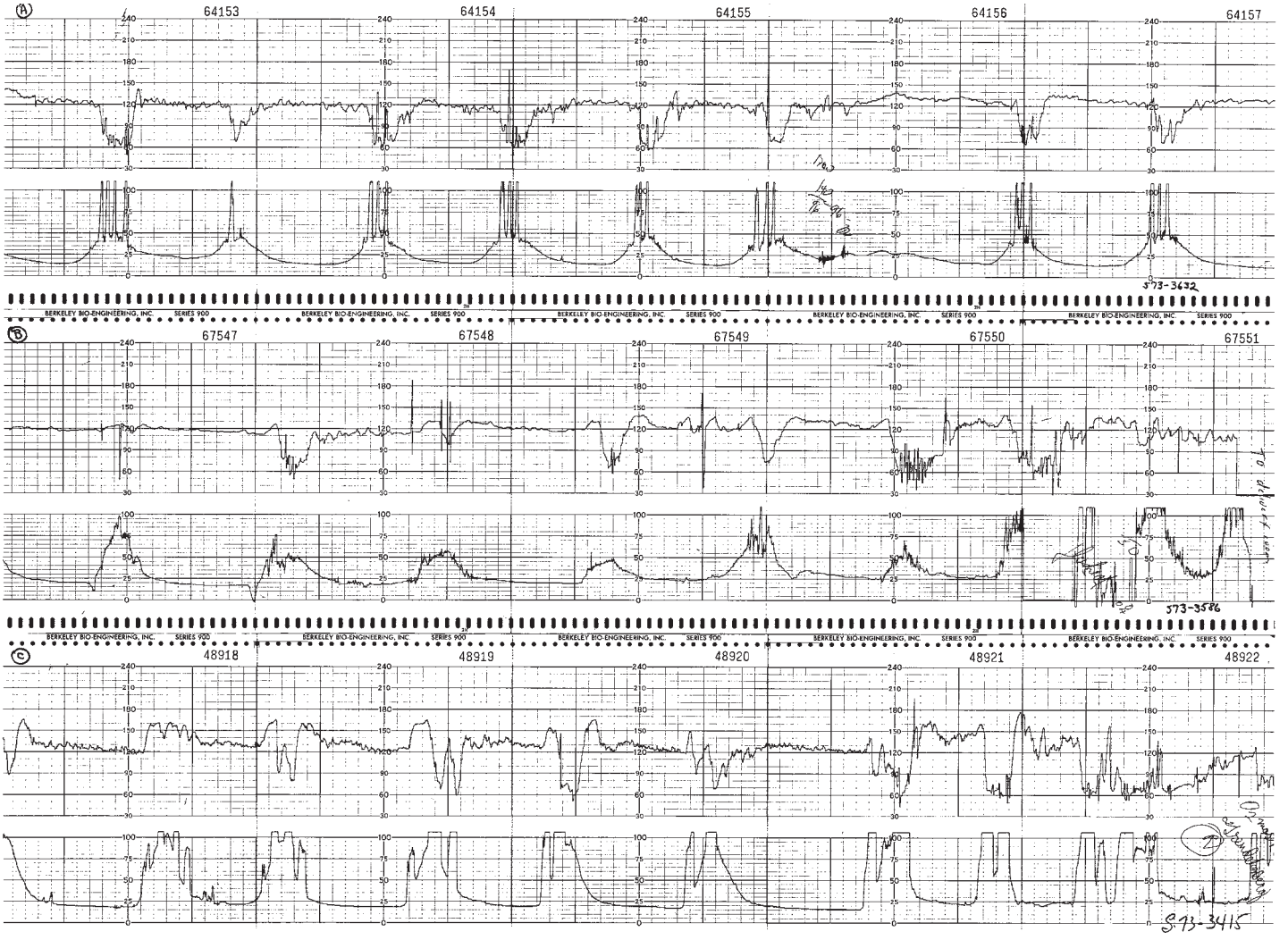


Figure 6.13. Typical variable decelerations are seen in this patient in the second stage of labor. Although all are occurring with contractions, they are variable in depth, duration, and shape. They are abrupt in onset and return to baseline. There are accelerations seen preceding and following most of these decelerations.

TABLE 6.4 Causes of variable decelerations and their relationships to labor

Oligohydramnios
Onset usually in the early active phase of labor or after membrane rupture
Descent
Onset usually at 8–10 cm of dilation
Often associated with nuchal cords
Aggravated by pushing efforts
Cord prolapse
Sometime after rupture of membranes
Associated with unengaged station or abnormal presentation
Unusual causes
True knots, short cords, limb entanglement occult prolapse, etc.
Onset and progression variably related to labor

considered in antepartum patients with frequent variable decelerations during FHR testing and in patients with variable decelerations occurring relatively early in labor.

It is useful to think of four potential groups of causes of variable decelerations, because these groups aid in understanding the pathophysiology, determining the best method(s) for correction, and predicting the most likely progression (Table 6.4). Variable decelerations that begin early in the active phase of labor are often associated with and caused by oligohydramnios (Fig. 6.14). These will not likely respond to position change but may well be reversed or ameliorated by amnioinfusion if they progress to require intervention. Those developing during or just before the onset of the second stage of labor are most likely due to umbilical cord stretch or compression. This time in labor coincides with an acceleration of descent of the fetal presenting part, and these decelerations are most commonly seen in association with cord encirclement about the fetal neck (nuchal cords). It is presumed that in such circumstances the variable decelerations are due to cord stretch as the fetus descends. Such events are so common that experienced labor and delivery nurses know it is time to examine the patient because the new appearance of variable deceleration often heralds the onset of the second stage (Fig. 6.15). Rarely the appearance of variable decelerations can announce the presence of umbilical cord prolapse, and this is another important reason to examine the patient. The final category of causes of cord compression can be thought of as "unusual cords" including such things as short cords, true knots, cord entanglement about fetal small parts, occult cord prolapse, etc. The course of cord compression is most unpredictable in such circumstances.

The vast majority of variable decelerations are not associated with significant hypoxia or, acidosis. Thus the challenge of evaluating and managing these patterns depends on the ability to distinguish between patterns that do and do not require further evaluation and management. In terms of fetal compromise, any insult should vary directly with the duration and degree of cord compression. With persistent mild degrees of cord compression, a mild respiratory acidosis may develop from carbon dioxide (CO_2) retention. However, if placental function is adequate and contractions are not too frequent, this CO_2 retention should clear rapidly with reversal of the respiratory acidosis. Should cord compression be prolonged and/or repetitive, progressive fetal hypoxia and resultant metabolic acidosis may also develop. For these reasons, variable decelerations have been graded as mild, moderate, or severe. The more severe the variable deceleration pattern, and the more prolonged and sustained it becomes, the more likely the result will be the delivery of a depressed newborn. Kubli et al. (12) graded variable decelerations on the basis of the level and duration of decelerations, without considering other parameters. Mild variable decelerations have a duration of <30

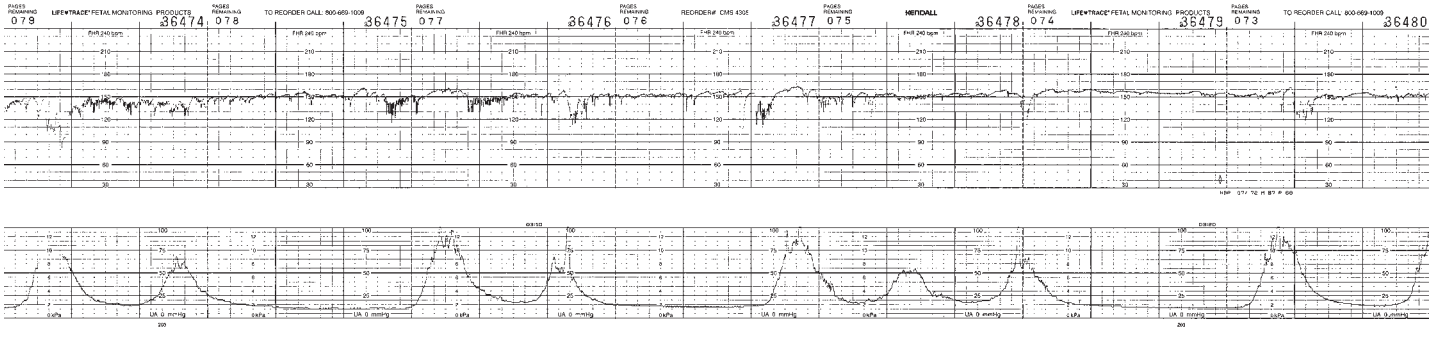


Figure 6.14. This nulliparous patient is at 41½ weeks in early labor. Note the occasional mild variable decelerations. The amniotic fluid volume on ultrasound was noted to be markedly reduced. Subsequently, at the time of amniotomy, thick meconium was noted and amnioinfusion was started. Variable decelerations appearing in early labor are unusual, and oligohydramnios should always be considered as an etiology of the cord compression.

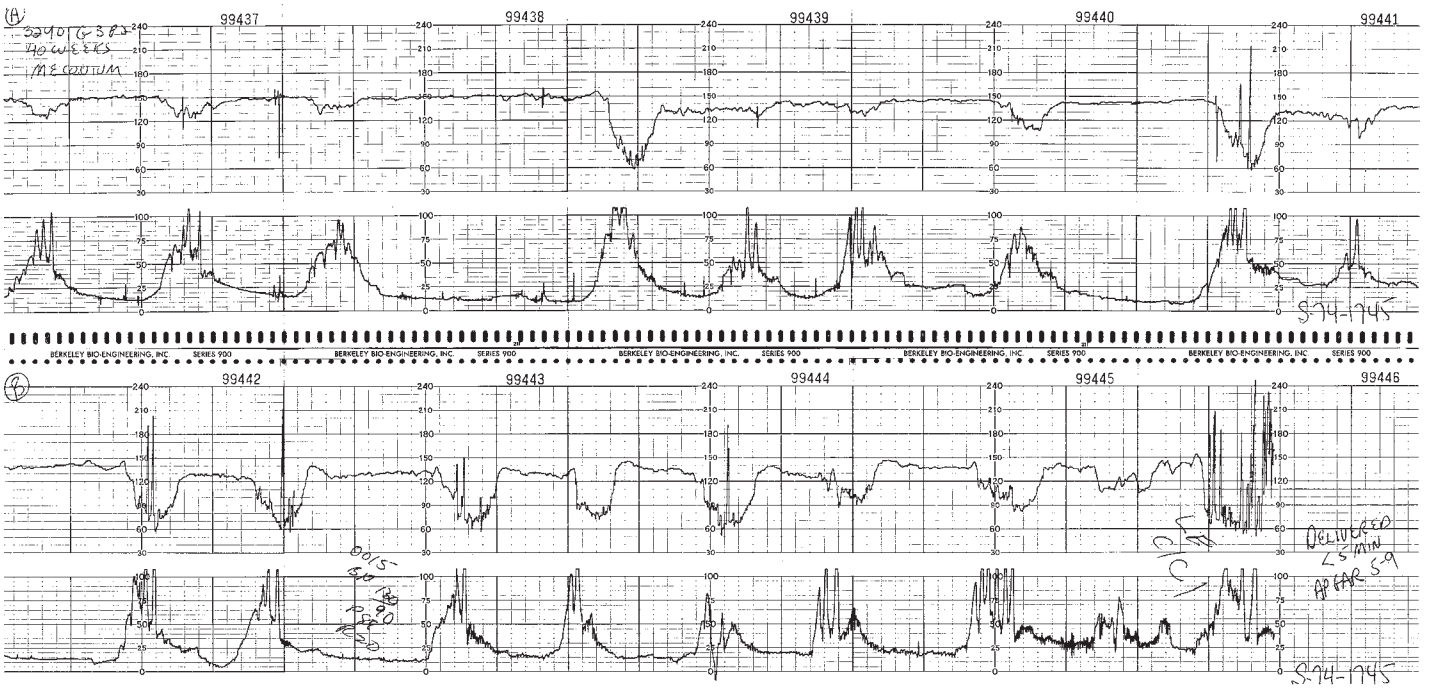


Figure 6.15. Variable decelerations here, as they often do, herald the onset of the second stage of labor in **panel A**. Maternal pushing can be detected by the short spikes on top of the contractions. The reason for this is probably a nuchal cord that is stretched during descent of the fetal vertex. Although the decelerations become larger and more regular, baseline heart rate and variability remain unchanged and do not signify hypoxia. A term-size infant with Apgar scores of 5 at 1 minute and 9 at 5 minutes was delivered <5 minutes after the monitor was removed and there was indeed a tight nuchal cord.

seconds, regardless of level, or a deceleration not below 70 to 80 BPM, regardless of duration (Fig. 6.16). Moderate variables have a level <80 BPM regardless of duration (Fig. 6.17). Severe variables are <70 BPM for >60 seconds (Fig. 6.18). While not included in the Kubli definitions, it should also be pointed out that variable decelerations may be categorized as severe in the setting of a tachycardia wherein the deceleration drop from a level, for example of 180 down to 80 or 90 BPM. While such a deceleration would not strictly fall into the severe category according to the original Kubli description, it would have the same or even worse significance given the additional concerning nature of the tachycardia in this setting. With variable decelerations, unlike late decelerations where hypoxia is the actual cause, the depth and duration of the decelerations correlate to the degree of hypoxia, but are not always indicative of hypoxia, because they are initially caused by a baroreceptor reflex (Fig. 6.19). To evaluate how a given fetus is responding to or tolerating these variable decelerations, other parameters of the FHR tracing require evaluation. Loss of variability and baseline tachycardia suggest possible progressive neurologic depression from hypoxia and acidosis (Fig. 6.20). In the presence of variable decelerations that are

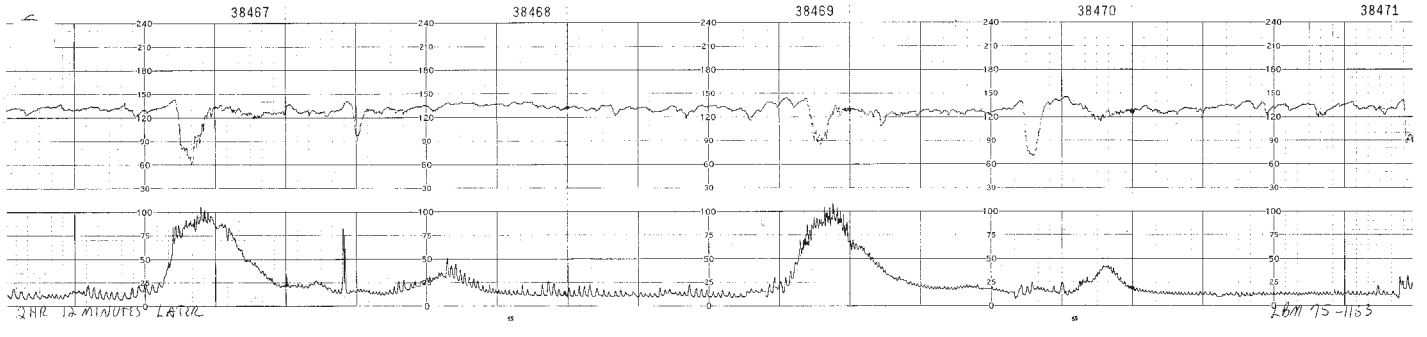


Figure 6.16. Mild variable decelerations are seen in this patient in early labor. They are occurring with contractions and probably with fetal movement. Baseline heart rate and variability are normal.

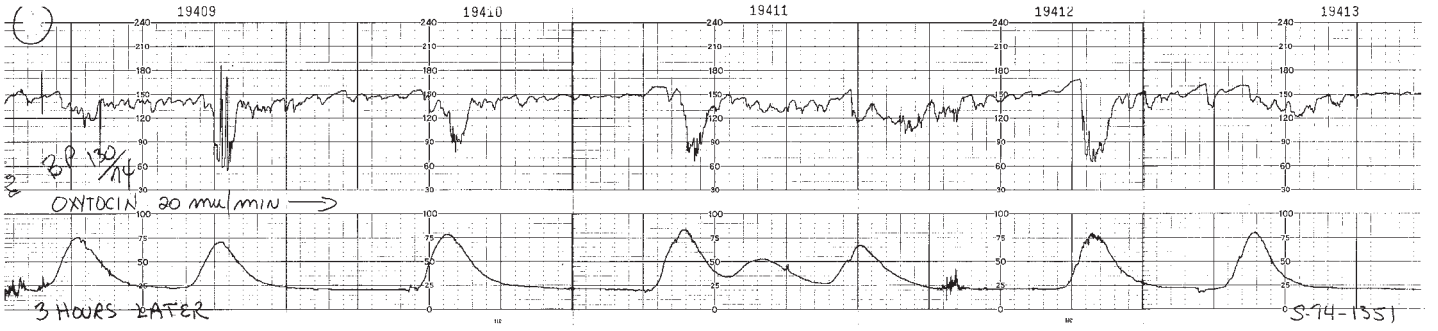


Figure 6.17. Moderate variable decelerations are seen in this panel. Baseline heart rate and variability are normal.

persistent, deeper, and of greater duration, the development of these additional warning signs, in the absence of other causes such as drugs, is an important sign that the fetus is not tolerating the intermittent cord compression. The other sign of fetal intolerance to cord compression is a slow return of the variable deceleration pattern to baseline heart rate (Fig. 6.21). Usually, variable decelerations are very abrupt in both their descent and return to baseline. Should the return to baseline persistently become more gradual, the indication is that progressive hypoxia is developing. It is probable that this slow return represents a component of late deceleration that would be consistent with a developing fetal hypoxia. Sometimes, distinct variable decelerations are followed by distinct late decelerations when simultaneous cord compression and primary UPI are occurring (Fig. 6.22). With mild variable decelerations, especially without tachycardia or loss of variability, it is unlikely that the cord compression has caused the hypoxia and late decelerations. Therefore, a placental perfusion or exchange problem probably coexists with cord compression. Variable decelerations with slow return to baseline is one of the most confusing and difficult patterns to interpret and manage. These patterns may or may not be associated with fetal hypoxia. Recent human data, using fetal pulse oximetry, have shown that when this pattern is preceded in its development by late or severe variable decelerations, then it is a sign of progressive hypoxia; but when slow return to baseline is associated with neither, then it is no more often associated with hypoxia than if the variable decelerations with which it occurs did not have a slow return to baseline (13).

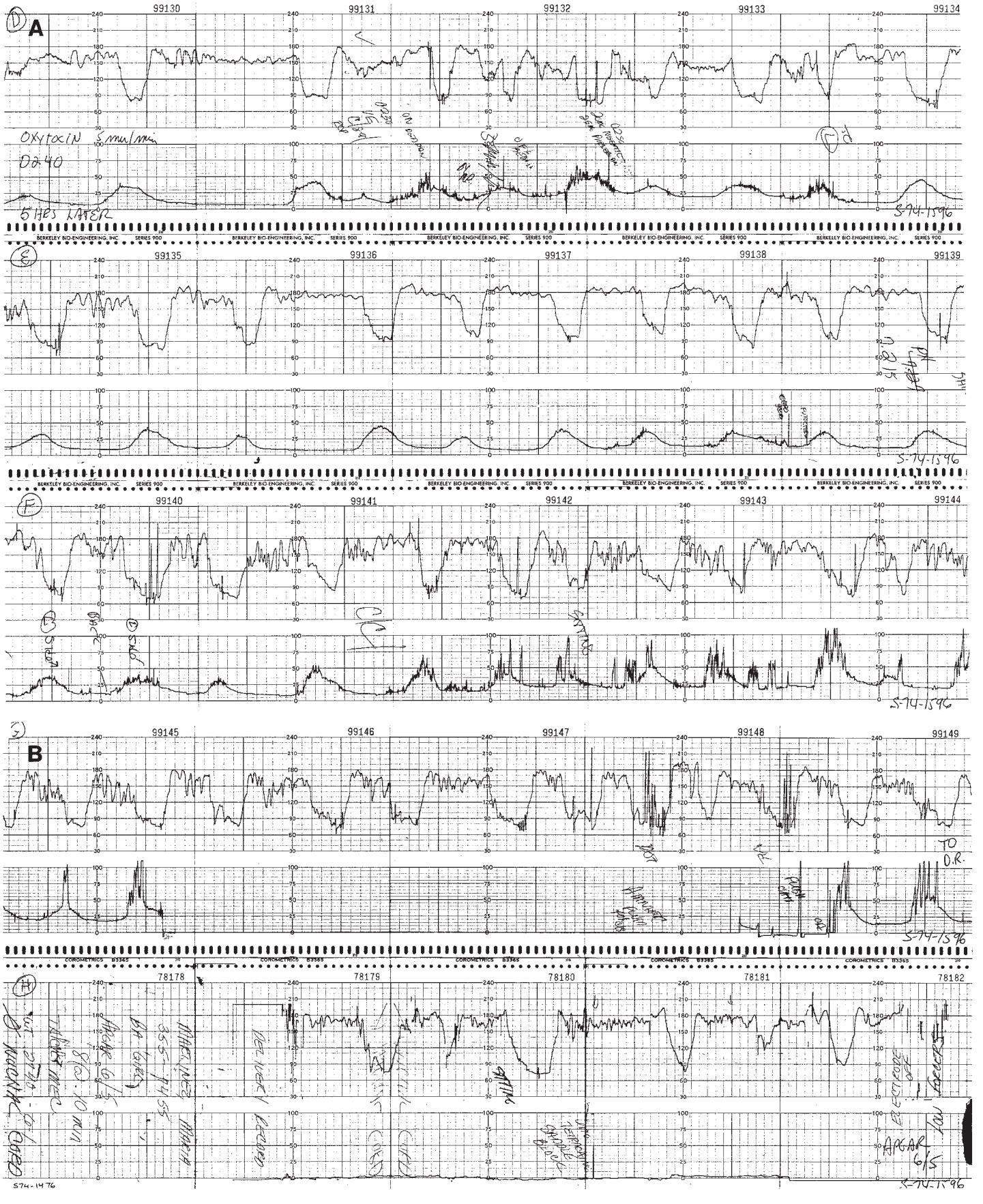
As variable decelerations become more severe and hypoxia is present or more severe, and especially as acidosis develops, additional FHR changes are common. The variable deceleration pattern will begin to appear smoother and rounded or blunted (Fig. 6.23). This change can be partially reproduced with atropine or may be seen in a very premature fetus. In extreme situations, with severe and progressive variable deceleration, the contraction may be followed by a blunt acceleration described by Goodlin and Lowe (14) as "overshoot." This is a transient smooth acceleration lasting more than a minute and occurring after severe variable decelerations. There is no acceleration preceding the deceleration. The overshoot lacks abruptness, is without STV within the acceleration, and returns to baseline very gradually. This pattern is only seen with variable decelerations, usually with a flat baseline and with blunted changes as previously described.

Another finding that may occur when variable decelerations approach 50 to 60 BPM is transient cardiac asystole. Junctional rhythms are common if the FHR decreases below 70 BPM, with ventricular escape beats seen on occasion. Prolonged asystole is quite unusual and sudden death exceedingly rare. These do not seem to alter the likelihood that the decelerations correlate with hypoxia and acidosis over and above the appearance of the remainder of the pattern.

Rather than attempting to quantify variable decelerations and using this as some means to determine intrapartum management, we view variable decelerations as primarily reflex or as indicating developing fetal hypoxia. Reflex variable decelerations do not suggest fetal hypoxia and have the following components:

1. The deceleration lasts no more than 30 to 45 seconds.
2. There is a rapid return to baseline from the nadir of the deceleration.
3. Moderate variability of the baseline FHR is present.
4. The baseline rate is not increasing.

Figure 6.18. Severe variable decelerations are seen in a 14-year-old primigravida admitted at 43 weeks with meconium-stained amniotic fluid. Variable decelerations are seen throughout, becoming progressively deeper and more prolonged. Baseline heart rate is somewhat erratic from 140 to 180 beats per minute. Increased heart rate variability seen in G and H probably represents early hypoxia between contractions. A 2,760-g infant with Apgar scores of 6 at 1 minute and 5 at 5 minutes was delivered. The low 5-minute Apgar score may have been caused by meconium in the airway and difficult ventilation.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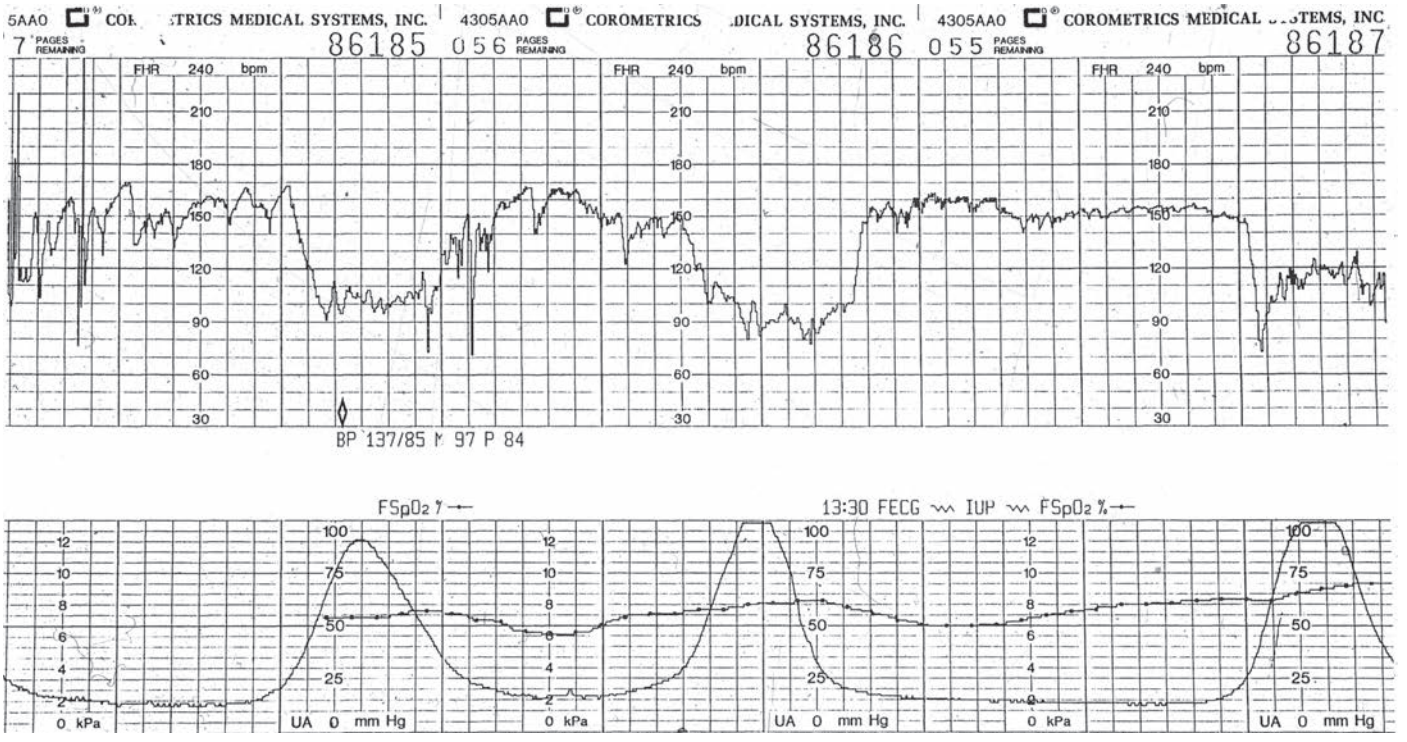

Figure 6.19. These deep variable decelerations dropping to 80 beats per minute and lasting up to 1 minute are seen in conjunction with a fetal pulse oximeter that is showing normal fetal oxygen saturation values between 50% and 60%. Because the inciting stimulus of a variable deceleration is the increase in peripheral vascular resistance in the fetal circulation, and a slowing of the fetal heart rate that is the result of a baroreceptor reflex, even marked decelerations are often not associated with hypoxia.

Variable decelerations that suggest developing fetal hypoxia are those that become deep and long lasting and have delayed return to baseline with or without overshoot. Decreasing baseline variability or an increasing baseline FHR are signs of developing fetal hypoxemia. In contrast to late decelerations, what makes variable decelerations so difficult to manage is their unpredictability. Because the second stage precedes delivery by only a relatively short time, and intervention is usually immediately possible should truly ominous patterns develop, more prolonged and deeper variable decelerations can be tolerated. This is true as long as the baseline heart rate is not rising and moderate variability is maintained (Fig. 6.15). Because cord compression is more frequent in the second stage and is likely to be more severe, having to deal with this issue in practice is a very common problem. To what extent head compression is involved in the production of these decelerations is not totally clear; however, the depth and progression of many of these decelerations do make it clear that cord compression is the main component, and referring to these second-stage

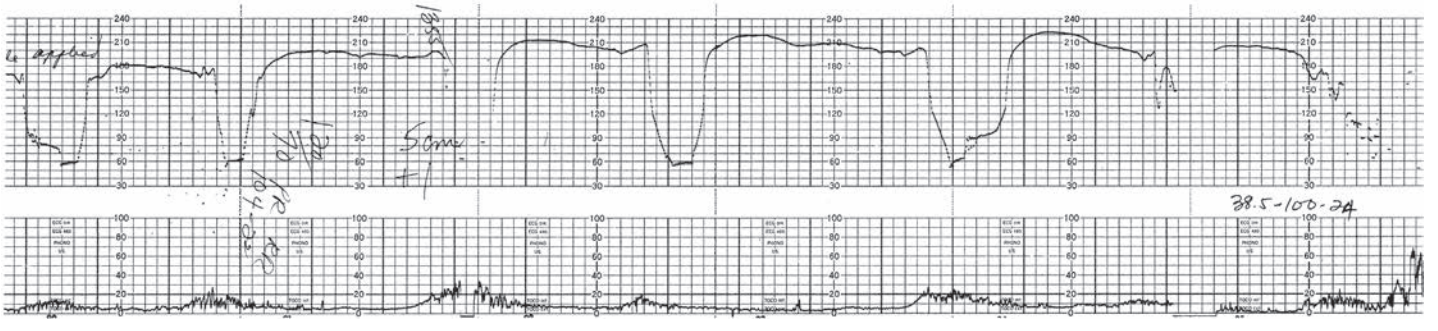

Figure 6.20. Severe ominous deceleration is seen with increasing heart rate to 210 beats per minute and virtually absent variability. A premature baby was delivered by cesarean section with Apgar scores of 1 at 1 minute and 2 at 5 minutes.

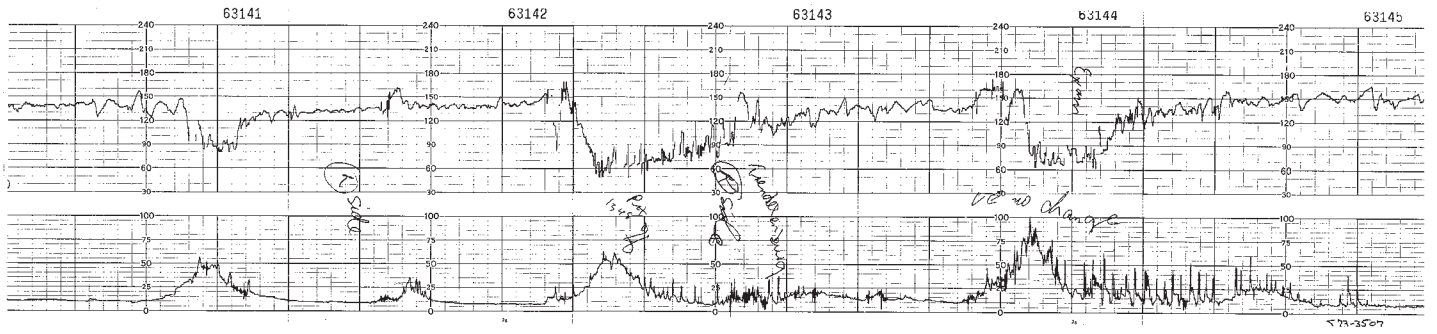

Figure 6.21. Severe decelerations are seen with slow return to baseline. Baseline heart rate and variability are normal.

decelerations as early deceleration is inappropriate. If loss of variability and an increasing baseline develop with the more severe second-stage variable decelerations, expeditious delivery definitely becomes warranted (Fig. 6.24).

Prolonged Decelerations

Prolonged decelerations are isolated decelerations lasting more than 2 minutes. According to the new NIH terminology, a prolonged deceleration that then lasts more than 10 minutes becomes a bradycardia. In either case, these are difficult to classify in terms of pathophysiology because they may be seen in a multitude of situations. As might be expected, cord compression can cause prolonged decelerations. This is generally seen either with progression of severe variable deceleration or with sudden occult cord prolapse (Fig. 6.25) but may also occur solely as recurrent prolonged decelerations. Profound placental insufficiency may cause prolonged decelerations. This is most characteristically seen with hypotension from the supine position or following epidural or spinal anesthesia (Fig. 6.26).

Hypertonic or tetanic uterine contractions may precipitate UPI-induced prolonged decelerations (Fig. 6.27). Tetanic uterine contractions can be seen with oxytocin, breast hyperstimulation, abruptio placenta, or with uterine artery vasospasm. Cocaine ingestion has been implicated in the development of vasospasm and abruptio placenta. Maternal hypoxia causing such decelerations might be seen with seizures, respiratory depression secondary to a

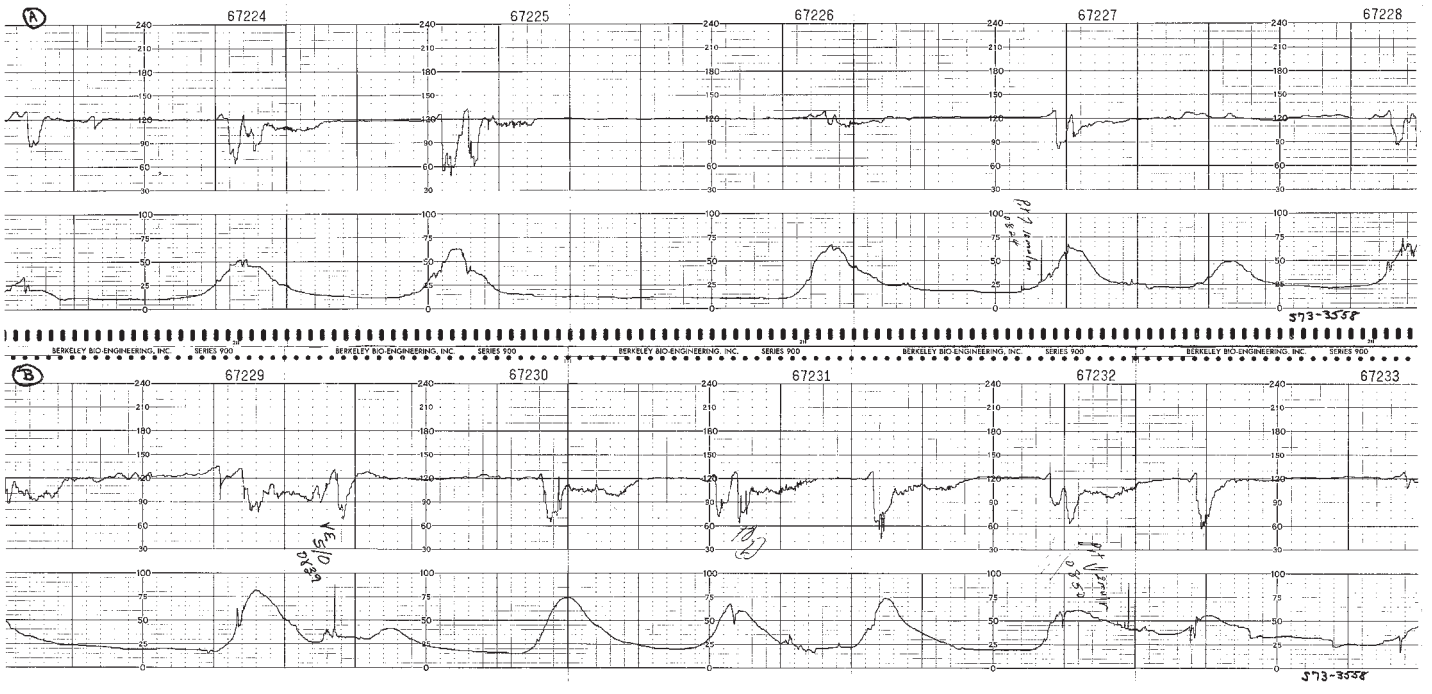

Figure 6.22. Mixed mild variable and late decelerations are seen with most contractions. With such mild variable decelerations, it is unlikely that progressive cord compression has caused the hypoxia, but a coexistent uteroplacental insufficiency probably exists. Poor variability suggests acidosis may also be present.

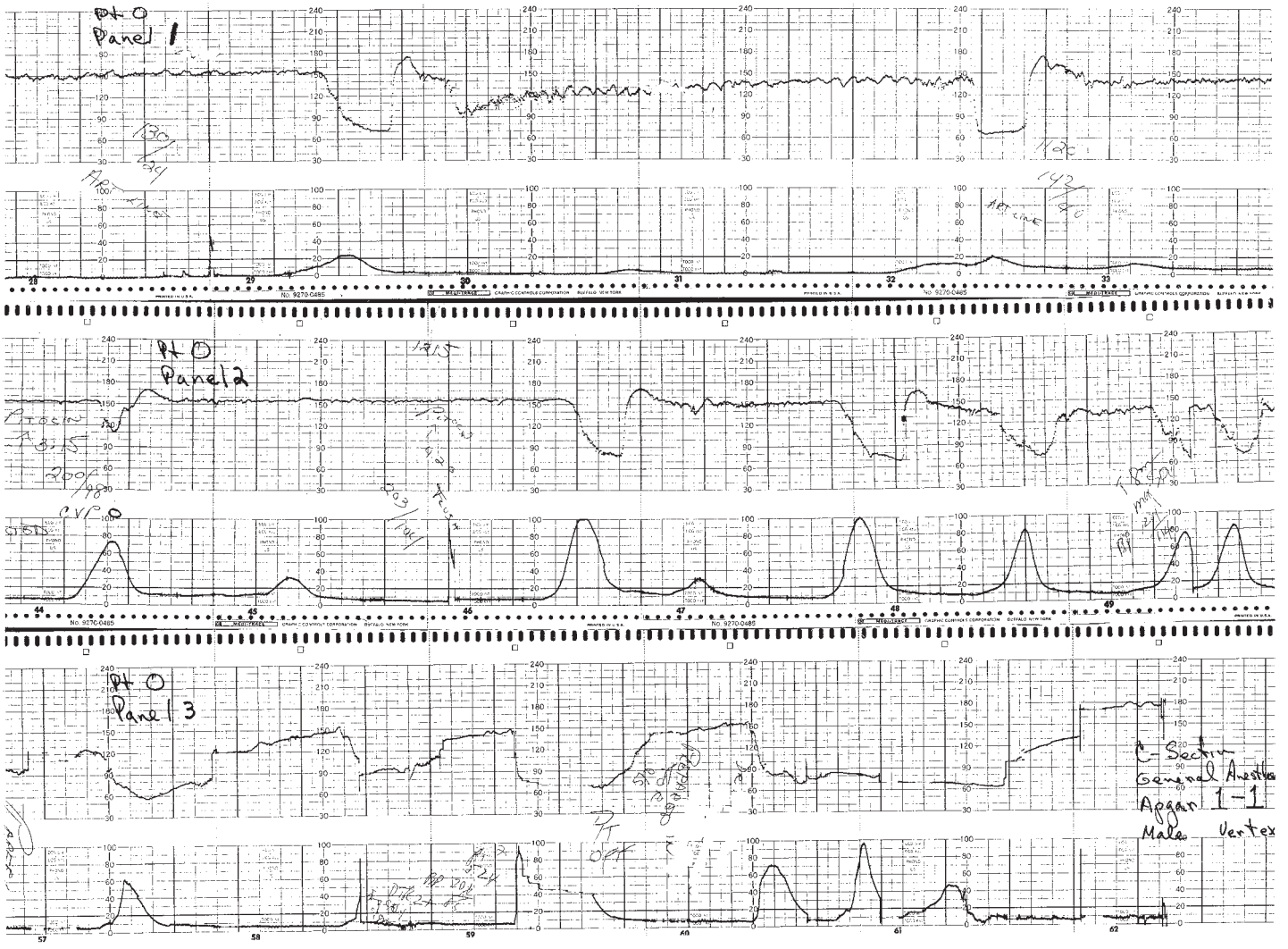

Figure 6.23. Severe variable decelerations are seen throughout these three panels. The blunted (rounded and not abrupt) accelerations seen following the decelerations are not the usual abrupt acceleration and may represent overshoot. Variability is progressively lost and decelerations become ominously prolonged. Delivery in this 30-week severe preeclamptic was by cesarean section and a severely depressed neonate with Apgar scores of 1 at 1 minute and 1 at 5 minutes was delivered.

high-spinal anesthetic, or following an overdose of narcotics or magnesium sulfate. Frequently, prolonged decelerations of the FHR, especially when the duration is more than 4 to 5 minutes, are associated with a rebound tachycardia and loss of variability (Fig. 6.28). This may be due to release of fetal epinephrine or may reflect some degree of fetal CNS depression or injury. If the original insult does not recur immediately, and the fetus was well-oxygenated before the insult, the placenta is very effective in resuscitating the fetus. Generally allowing placental resuscitation is a better choice than operative intervention in these situations. The loss of variability and tachycardia are not necessarily prognostically ominous because the insult may no longer be present and the placenta can effectively restore the fetus to its normal well-oxygenated state. Occasionally, in addition to the loss of variability and tachycardia seen after such prolonged decelerations, there may be a period of late decelerations. These usually clear spontaneously with *in utero* recovery of the fetus.

Prolonged decelerations may not always return to baseline. When seen following a protracted course of severe variable decelerations or a prolonged period of recurrent late decelerations, such a prolonged deceleration may occur just before fetal death (Fig. 6.29). Recurrent prolonged decelerations without apparent etiology probably represent cord compression and are the most difficult of all patterns to manage. This is because one cannot prognosticate fetal tolerance based on previous performance of the FHR in labor or on such parameters of the FHR as variability. Such prolonged decelerations may just continue to recur, and prolonged cord compression may cause fetal death.

There are a few other, more benign, causes of prolonged declarations that merely represent an active fetal vagus

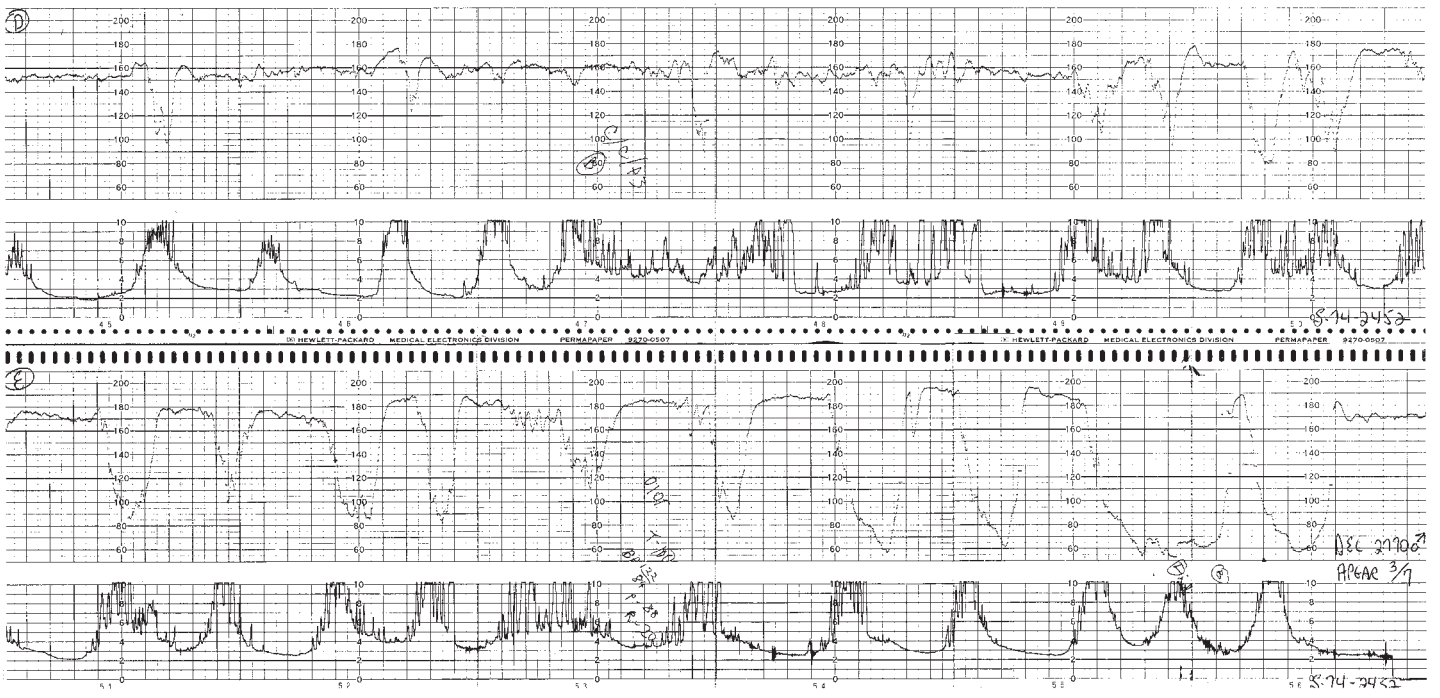

Figure 6.24. The patient is being monitored with an internal electrode and intrauterine catheter. There is a rapid progression of variable decelerations from mild to severe. The baseline heart rate rises from 150 beats per minute (BPM) at the beginning to 190 BPM. The heart rate variability is normal at the beginning of the tracing but is markedly decreased just before delivery. The patient was delivered by low forceps of a 2,770-g male with Apgar scores of 3 at 1 minute and 7 at 5 minutes and a single loop of tight nuchal cord.

nerve and not fetal hypoxia. Occasionally, such decelerations are associated with pelvic examination (Fig. 6.30), application of scalp electrode, rapid descent of the fetus through the birth canal (Fig. 6.31), or with sustained maternal Valsalva maneuver (Fig. 6.32). Such decelerations generally do not last more than a few minutes and are not usually followed by tachycardia or loss of variability. Because these prolonged decelerations are thought to be vagally mediated, therapy with atropine has been suggested. However, this treatment is not recommended, because there is not an associated increase in fetal cardiac output or oxygenation (15).

Accelerations

Because periodic changes are defined as transient changes above and below the baseline, accelerations are the counterpart of decelerations. Accelerations of the FHR occur most commonly in the antepartum period, in early labor, and in association with variable decelerations. There are at least two physiologic mechanisms responsible for accelerations. Accelerations associated with fetal movement or uterine contractions (Fig. 6.33) seem to have the same significance as FHR variability in that their presence represents fetal alertness or arousal states. The other cause of accelerations seems to be

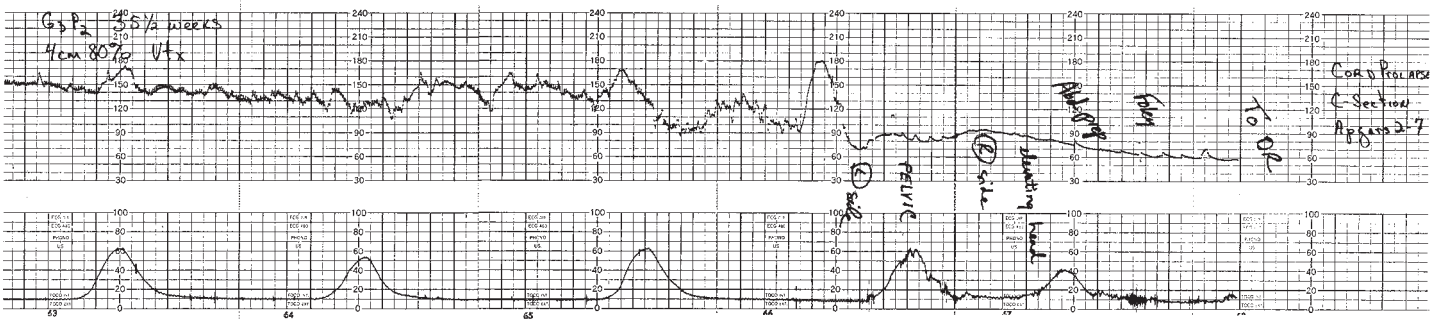

Figure 6.25. A sudden prolonged deceleration is seen in this patient in the early active phase of labor. An immediate pelvic examination revealed cord prolapse, and cesarean section was performed.

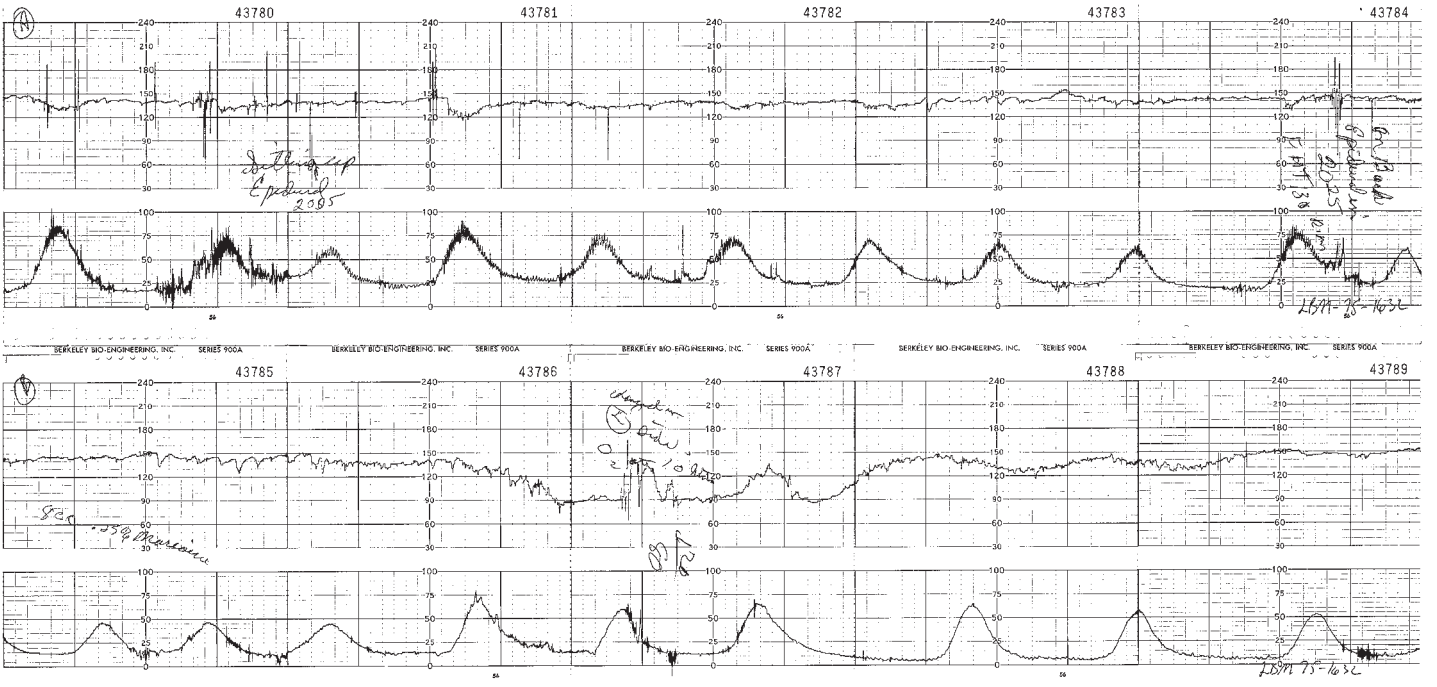

Figure 6.26. A prolonged deceleration is seen after injection of epidural anesthesia with Marcaine. This is followed by several late decelerations, often seen following such epidural-induced decelerations. The pattern subsequently returned to normal and a vigorous newborn was delivered vaginally.

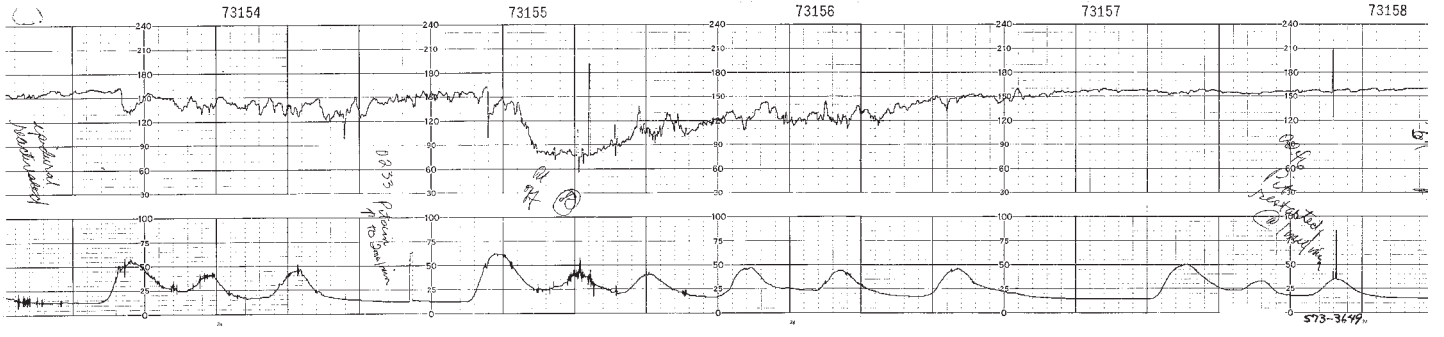

Figure 6.27. Here a prolonged deceleration is seen associated with excessive uterine activity secondary to oxytocin hyperstimulation. Again, a rebound tachycardia with decreased variability follows the prolonged deceleration. Pitocin was stopped and restarted at a lower rate and the heart rate subsequently returned to normal.

partial umbilical cord occlusion. If the low-pressure umbilical vein is compressed and the higher pressure umbilical artery remains patent, a period of decreased placental return and fetal hypotension results in baroreceptor response. The normal baroreceptor response to hypotension or decreased cardiac return is an increase in heart rate, with a resultant acceleration.

The presence of FHR accelerations in the intrapartum period indicates the absence of acidosis. Hypoxia with a normal pH may or may not result in the loss of accelerations. These accelerations may occur with contractions, fetal movement, or without apparent stimulus. In addition, as with decelerations, accelerations may be seen in response to pelvic examination and stimulation of the fetal head (Fig. 6.34). Virtually all of these accelerations are associated with fetal movement. Indeed, as described in Chapter 8, accelerations with intrapartum pelvic examination or fetal scalp stimulation reflect normal fetal pH. This is the rationale for using spontaneous or stimulus-produced accelerations in the presence of FHR patterns with concerning decelerations and/or decreased variability to reassure the clinician that the baby is neither depressed nor acidotic. It cannot be emphasized enough, however, that the absence of FHR accelerations in the intrapartum period is not in and of itself alarming as long as variability is normal and there are no deceleration patterns indicative of possible fetal hypoxia. Thus in labor, fetuses are often inactive, and long periods with the absence

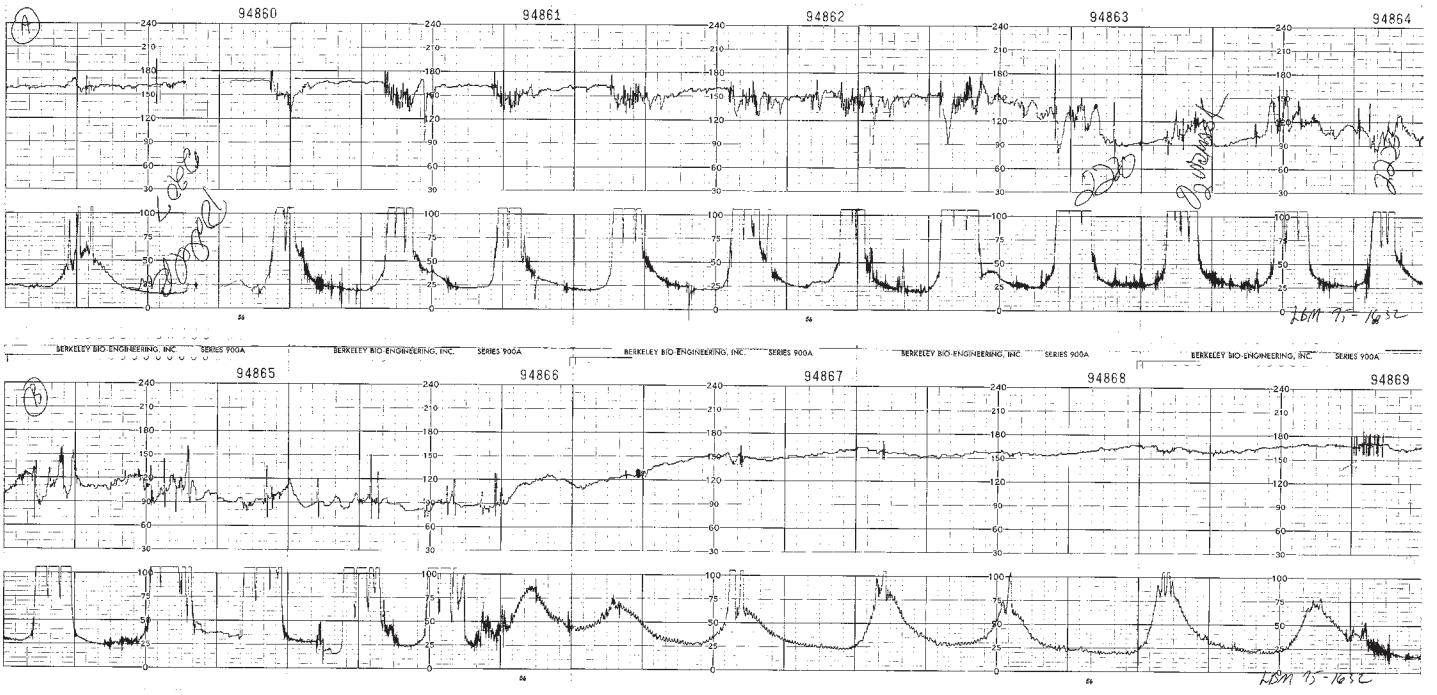

Figure 6.28. A prolonged deceleration from a baseline heart rate of 160 to 90 beats per minute (BPM) is seen lasting 12 minutes in the second stage of labor. An apparent cause is not present. Tachycardia to 170 BPM and decreased variability are seen after this deceleration. Also, some subtle late decelerations are probably present during this time. Again, the heart rate pattern returned to normal. No further significant decelerations recurred and a vaginal delivery of an Apgar 7 infant occurred approximately 15 minutes later.

of accelerations occur that do not, in and of themselves, constitute a concerning scenario. One other problem created by accelerations of the FHR is that, at times, it is difficult to be sure whether one is dealing with decelerations or with accelerations with return to baseline (Fig. 6.35). This is especially true in the beginning of the monitoring period when the baseline has not been established. This is an important practical problem because there may be cases of misinterpretation with intervention for fetal distress when, in reality, there were no decelerations but rather accelerations mistaken for baseline heart rate and return to baseline mistaken for decelerations. There are three clues to help avoid this difficulty. First, accelerations and decelerations are rounded at their peak, whereas the baseline tends to be flat. Second, with accelerations especially, there is usually a period preceding or following without periodic changes when the baseline may be more clearly determined. Third, accelerations are almost always associated with fetal movement that can be documented by patient symptoms, palpation, or ultrasound.

Unusual Patterns

Sinusoidal Pattern

Originally described in separate reports from Shenker (16) in 1973 and Kubli et al. (17) in 1972, the sinusoidal FHR is a rare but distinct baseline pattern. Observed in antepartum, intrapartum, and neonatal FHR monitoring, this pattern is strongly associated with fetal hypoxemia, often resulting from severe fetal anemia (Fig. 6.36). This fetal anemia may result from Rh sensitization, fetal-maternal hemorrhage, or *in utero* fetal hemorrhage and has an associated increased perinatal morbidity and mortality. The sinusoidal FHR pattern has been reported following the intrapartum administration of the analgesics alphaprodine (Nisentil) (18), butorphanol (Stadol) (19), or meperidine (Demerol) (20), and in association with amnionitis (21). In the absence of acidosis or anemia, sinusoidal heart rate following analgesic administration does not appear to have an ominous significance for the fetus, and the mechanism of this heart rate change is unclear. Many times, benign FHR patterns with increased LTV may be easily confused with sinusoidal patterns (Fig. 6.37).

The ability to correctly recognize and manage a sinusoidal pattern unrelated to previous analgesia use is of critical importance. Despite numerous publications on the definition, pathogenesis, and clinical significance of the sinusoidal heart rate pattern, confusion continues regarding the features of sinusoidal heart rate associated with poor perinatal outcome. We consider that the following heart rate features must be present: (a) stable baseline heart rate of 120 to 160 BPM with regular oscillations, (b) amplitude of 5 to 15 BPM (rarely greater), (c) frequency of 2 to 5 cycles per minute (as LTV), (d) fixed or flat STV, (e) oscillation of the sinusoidal

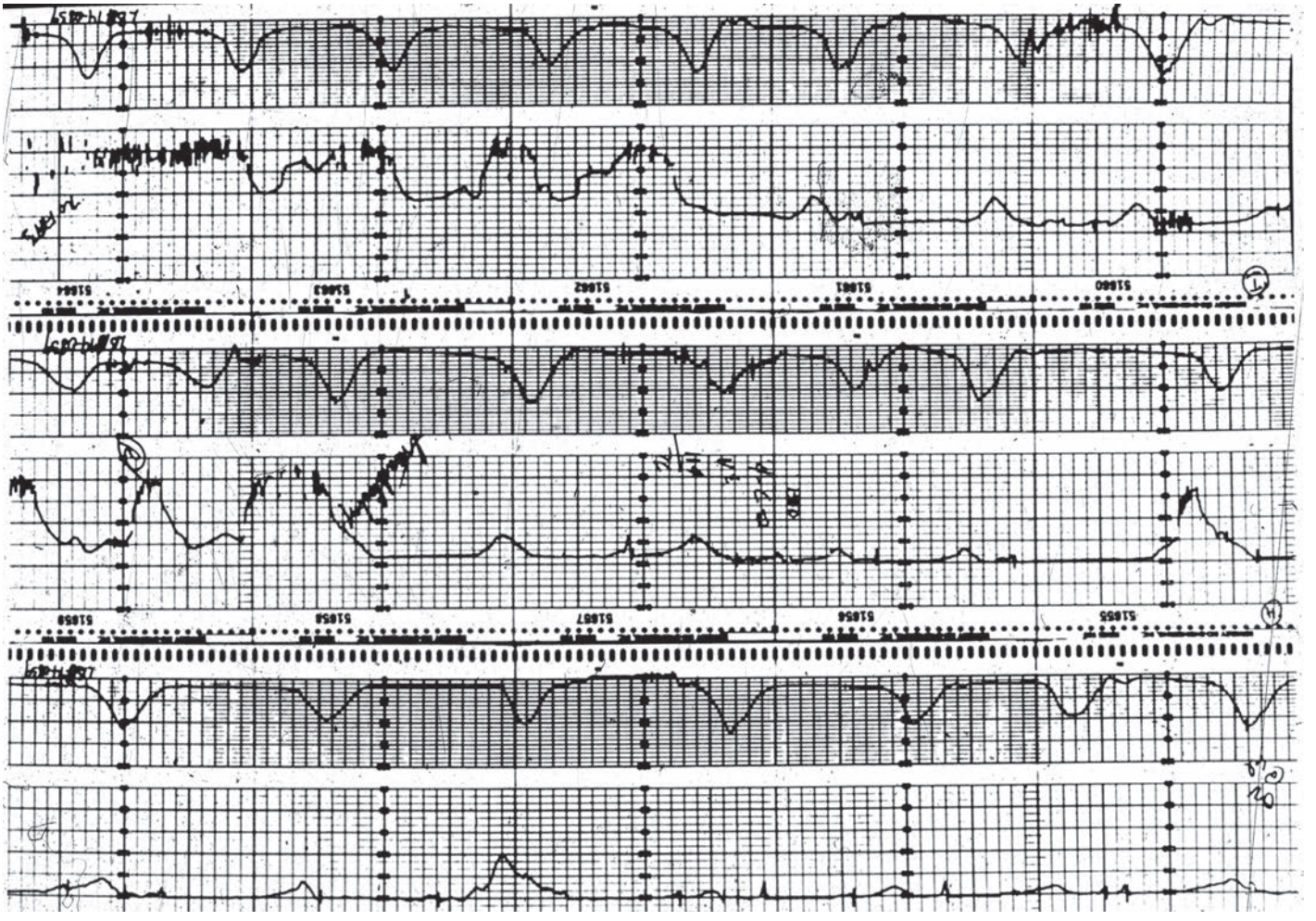

Figure 6.29. This fetus in labor is having recurrent late decelerations with absent variability and tachycardia. At the end of the strip a terminal bradycardia is seen immediately preceding the demise of the fetus.

wave from above and below a baseline, and (f) absence of FHR accelerations (22).

The exact pathophysiology of sinusoidal heart rate remains unknown. The association of fetal anemia with the sinusoidal heart rate is well documented for the human fetus. Young et al. (23) clinically demonstrated an inverse correlation between sinusoidal heart rate amplitude and fetal pH. A derangement of nervous control of the heart secondary to central or peripheral ischemia is hypothesized to result in sinusoidal heart rate (24–26). Murata et al. (27) have

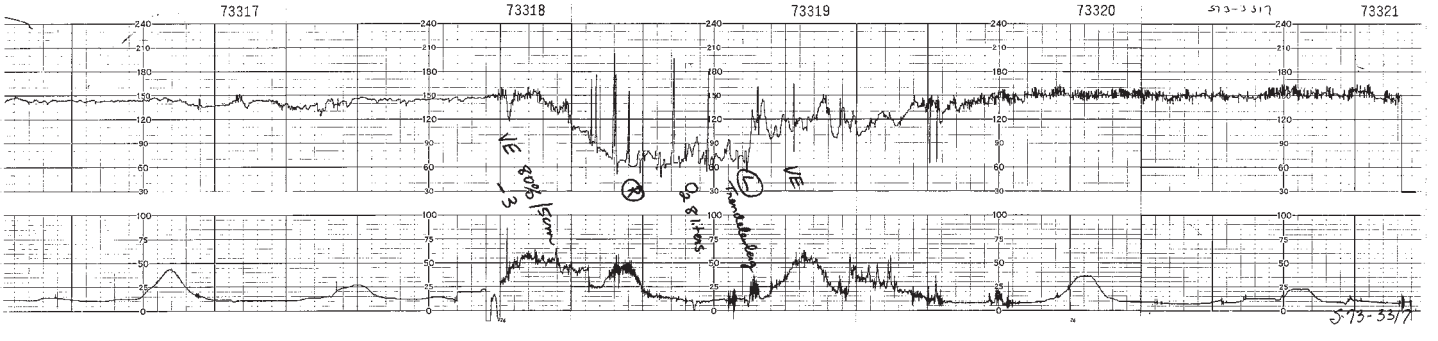

Figure 6.30. A prolonged deceleration is seen after a pelvic examination. Also, note the uterine hyperactivity commonly seen following a pelvic examination (Ferguson reflex?). This probably represents a fetus with an active vagal reflex.

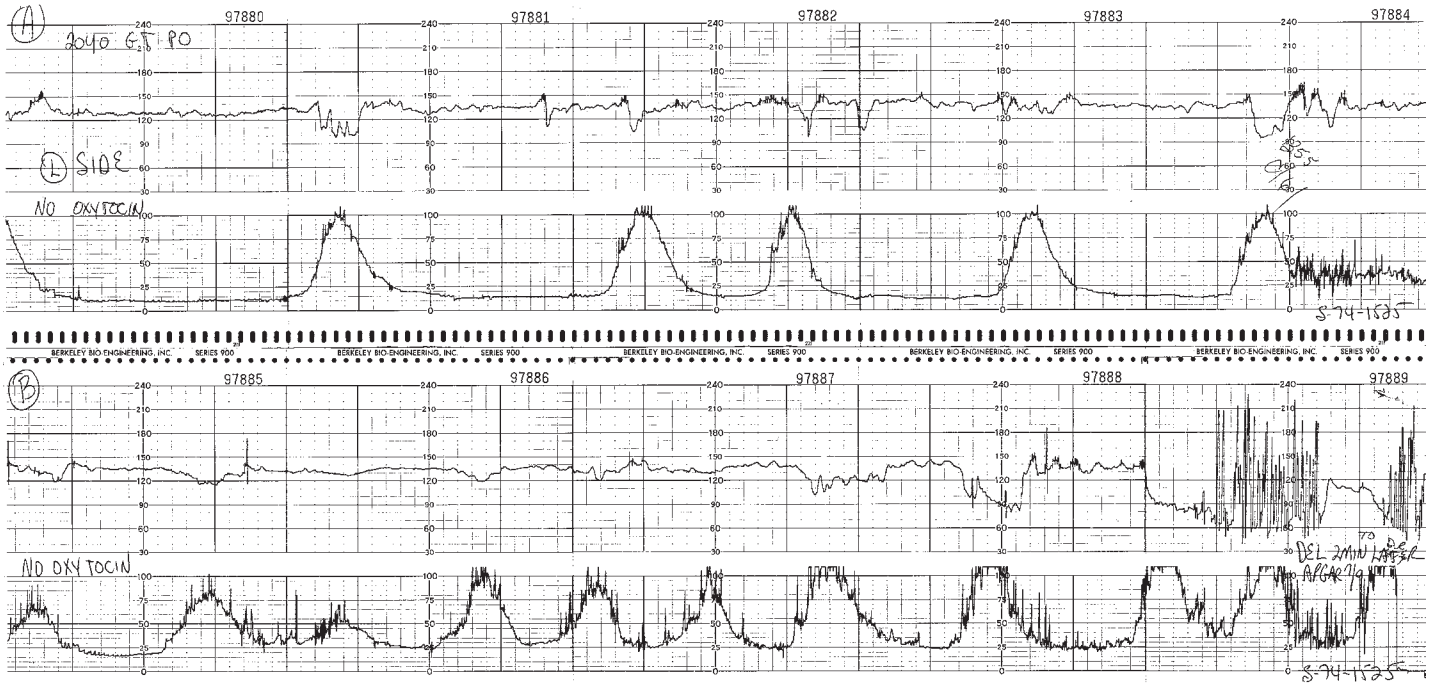

Figure 6.31. In this case, a prolonged deceleration is seen at the end of panel B. This patient progressed rapidly from 6 cm at the end of panel A to delivery 2 minutes after the end of panel B. Such prolonged decelerations should incite performance of a pelvic examination, not only to rule out cord prolapse, but as is often the case, to check for rapid descent as occurred here.

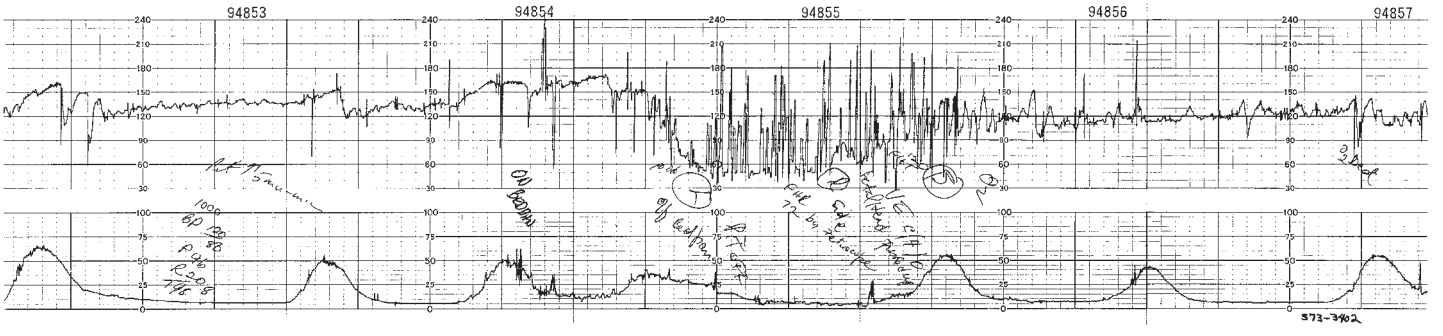

Figure 6.32. Here, a prolonged deceleration occurs while the patient is on a bedpan. Again, a fetal vagal reflex may cause decelerations with maternal Valsalva.

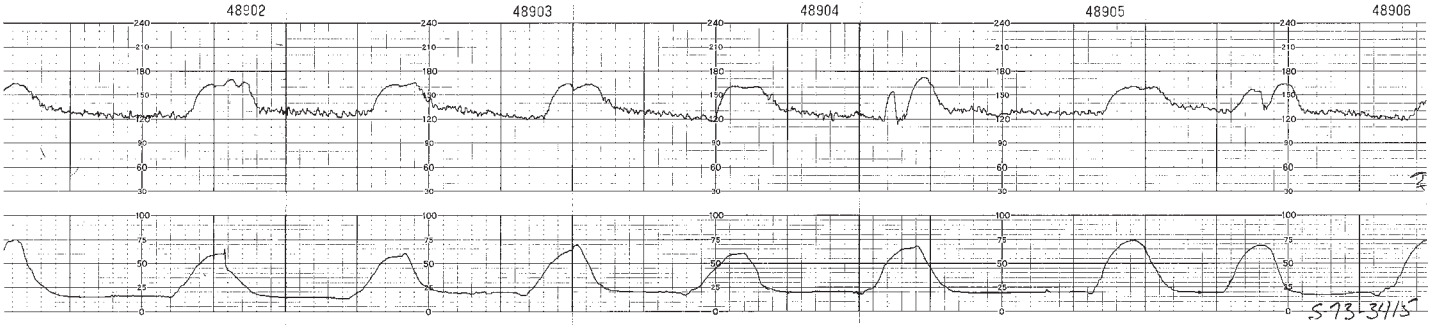

Figure 6.33. Accelerations of the fetal heart rate are seen with each contraction. Baseline heart rate and variability are normal. Such a pattern indicates a normal fetal pH.

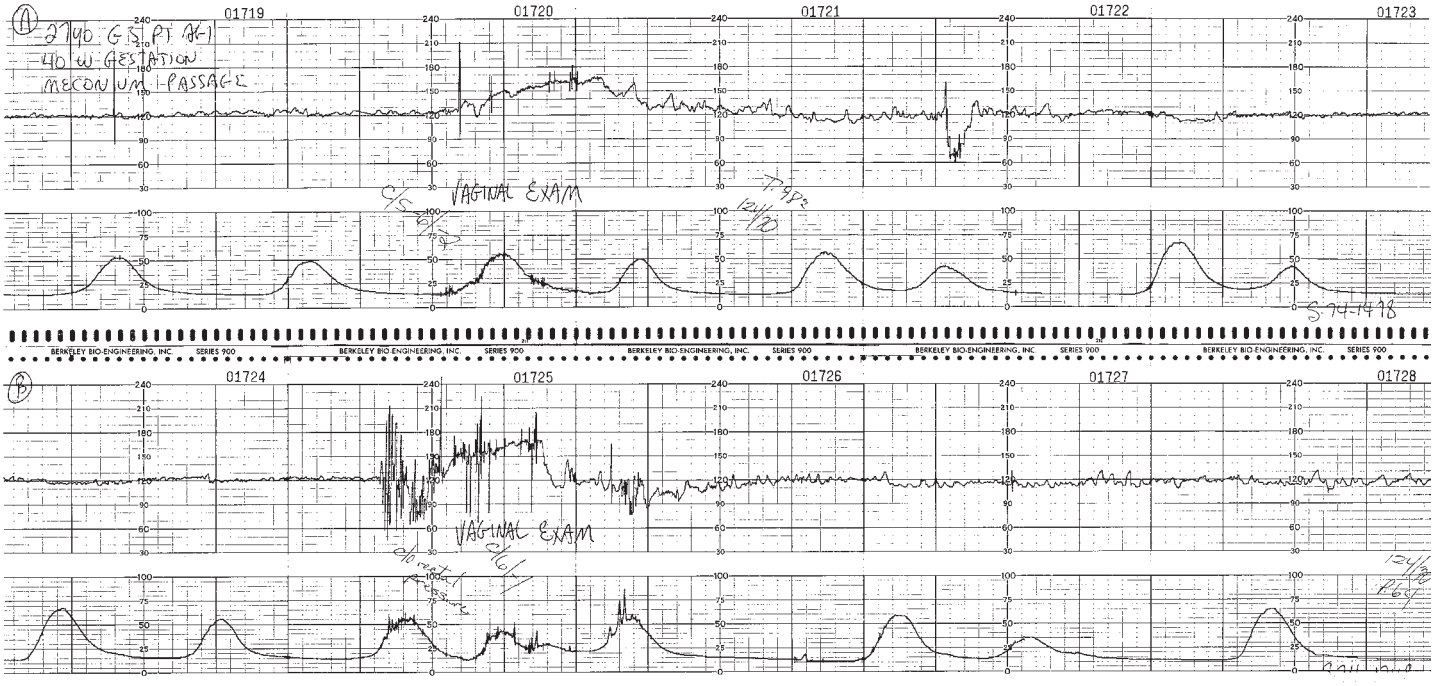

Figure 6.34. Just as a vaginal examination may precipitate prolonged deceleration (Fig. 6.30A), a vaginal examination may stimulate the fetal heart rate to accelerate as occurs in both panels.

reported in an animal model an association between sinusoidal heart rate and plasma arginine vasopressin concentration. Chemical or surgical vagotomy with subsequent infusion of arginine vasopressin produces a sinusoidal pattern. Arginine vasopressin is elevated following hemorrhage or acidosis, and perhaps secondary to direct or indirect effects of this hormone on calcium transfer in the sinus node, sinusoidal heart rate results.

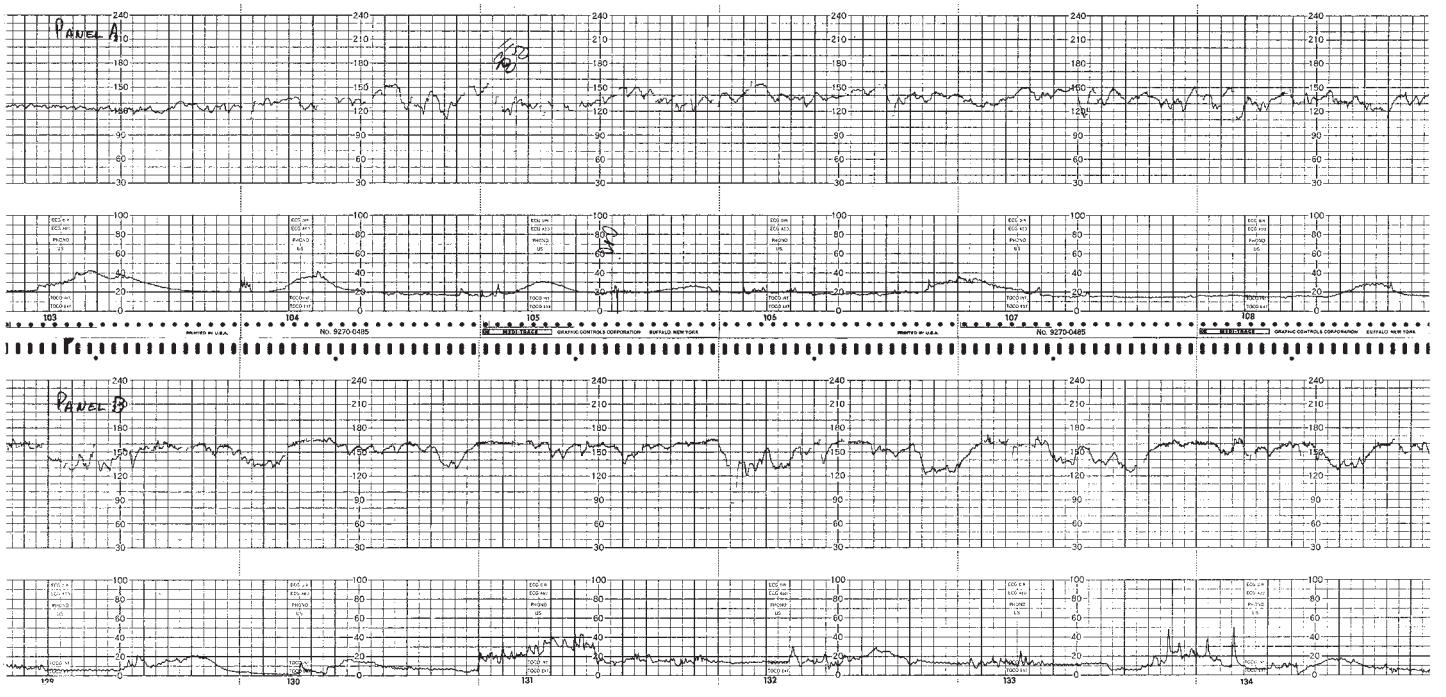

Figure 6.35. In **panel B**, accelerations with a return to baseline could easily be confused for late decelerations. Looking back at **panel A**, it can be seen that the real baseline heart rate is 120 to 130 beats per minute. Also, note that the return to baseline tends to be flat rather than rounded at the nadir as late decelerations would be.

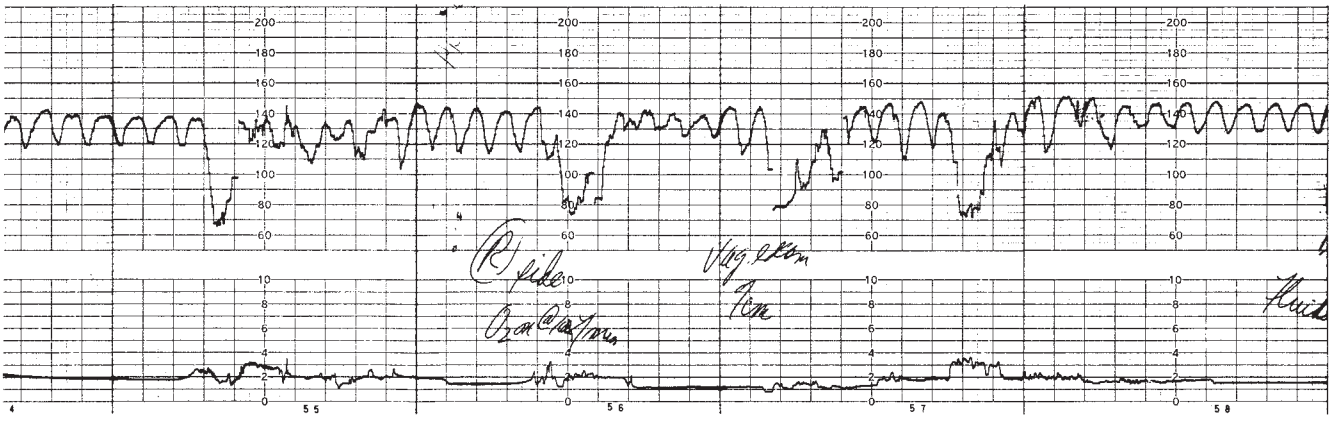

Figure 6.36. Intrapartum sinusoidal fetal heart rate pattern. There are also moderate variable decelerations present. (From Klavin M, Laver A, Boscola M: *Clinical concepts of FHR monitoring*. Hewlett-Packard Co., Boston, 1977:106, with permission.)

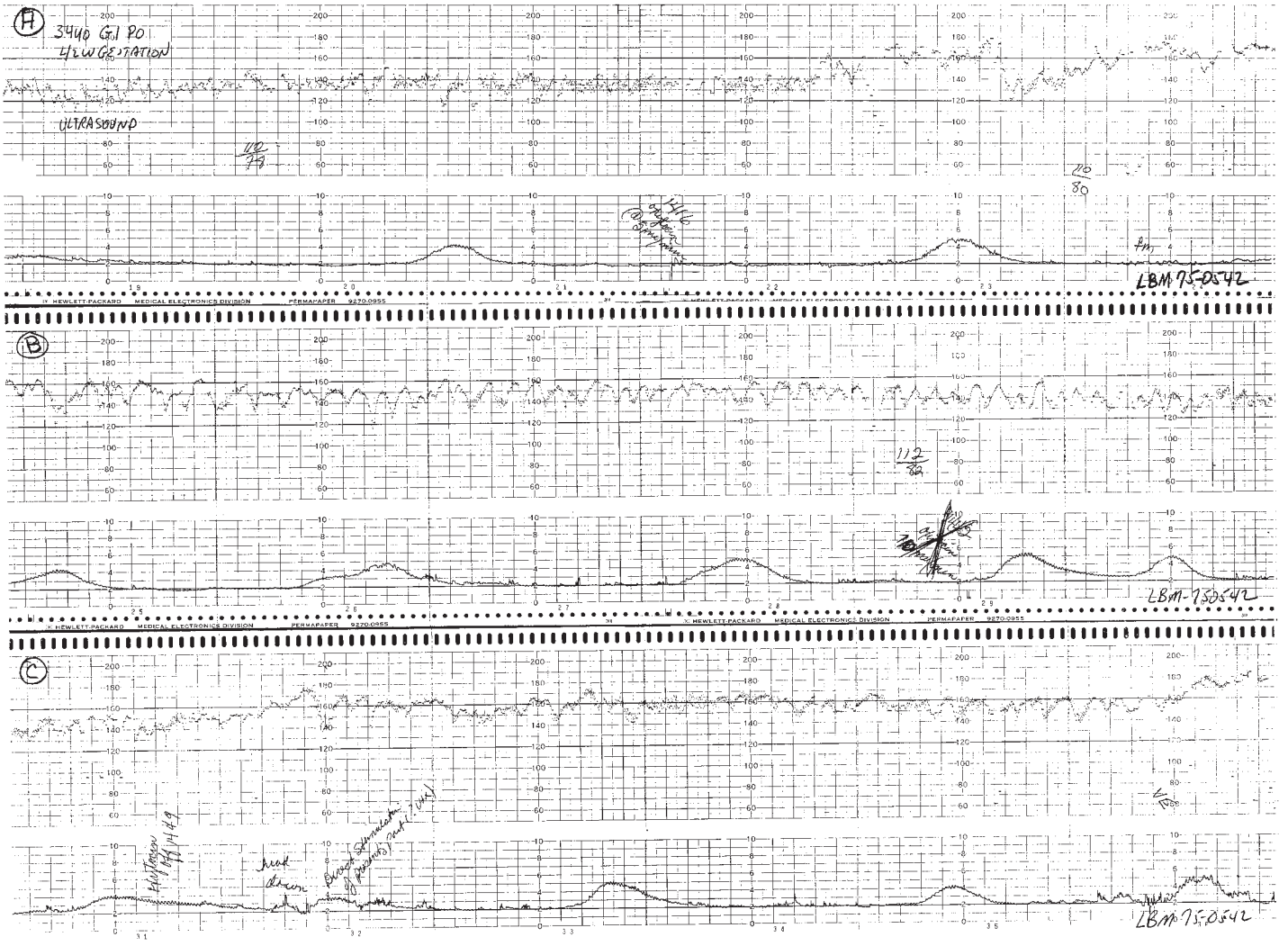

Figure 6.37. As seen in **panel B**, exaggerated long-term variability may resemble sinusoidal heart rate patterns. The presence of short-term variability within the pattern, and the normal patterns before and after **panel B**, distinguish this pseudosinusoidal pattern from a true sinusoidal one.

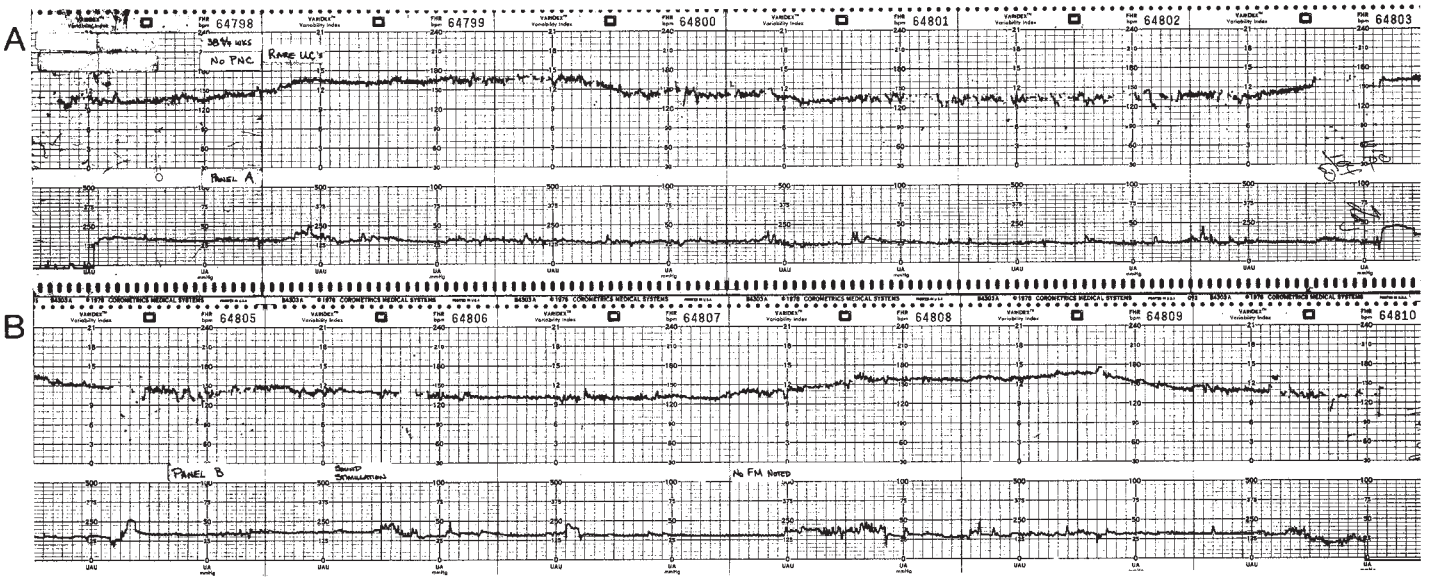

Figure 6.38. As seen in **panel A**, when evaluated in early labor, this patient was thought to have a prolonged acceleration signifying absence of fetal acidosis. However, as the monitoring was continued, there was no evidence of true reactivity (**panel B**). The patient stated that fetal movement had been decreased for 2 weeks. A biophysical profile found no evidence of fetal breathing, tone, or movement. Artificial rupture of membranes revealed thick yellow meconium. Subsequently, persistent late decelerations were noted and emergency cesarean section was performed. The infant was depressed (Apgar scores 2 and 3 at 1 and 5 minutes) but not acidotic (umbilical pH 7.30). Seizure activity was noted in the delivery room. A head computed tomography scan and ultrasound revealed a large cystic area in the left brain consistent with a porencephalic cyst. This example of a wandering baseline resulted from significant preexisting central nervous system insult.

Wandering Baseline

A very rare FHR abnormality is seen when it is impossible to establish the baseline. Although falling within the defined normal limits of 120 to 160 BPM, this baseline wanders and does not remain steady. This rare abnormality is seen in the absence of STV and is highly suggestive of a neurologically abnormal fetus. This wandering baseline will occasionally be seen as a preterminal event (Fig. 6.38).

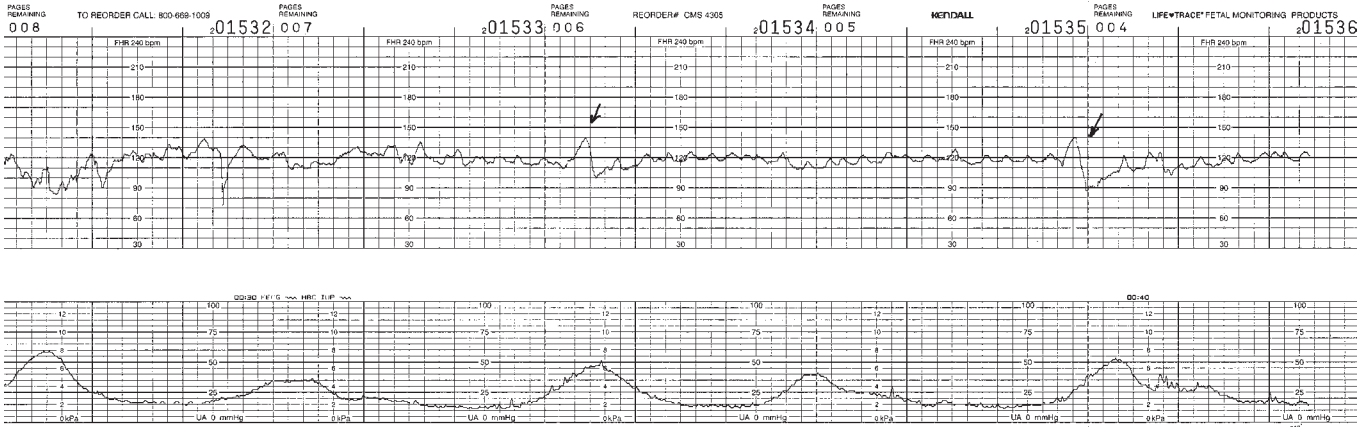

Figure 6.39. The complex of an acceleration immediately followed by a variable deceleration has been termed the "lambda" pattern. Generally, this is similar to a variable deceleration. Such patterns are, benign, often confused with late decelerations, but in general are not persistent and do not portend the appearance of a subsequently deteriorating tracing. Arrows are pointing to accelerations.

Lambda Pattern

First described by Aladjem et al. (28), the "lambda pattern" is an FHR pattern involving an acceleration followed immediately by a deceleration (Fig. 6.39). Although neither rare nor ominous, the problem with this pattern is the potential for confusing it with late deceleration or other abnormal patterns (29). This pattern most typically appears early in labor and does not persist. The appearance of this pattern does not predict an increased likelihood of subsequent development of ominous variable or other concerning patterns. The mechanism responsible for causing this pattern is unknown but may result from intermittent mild cord compression or stretch.

CONCLUSION

A knowledge of the physiology and pathophysiology of the FHR, coupled with experience in pattern recognition, is essential for the appropriate use of electronic FHR monitoring. Pattern recognition is a process of recognizing FHR changes, such as decelerations, that suggest the type of pathophysiologic process occurring, determining how the fetus is tolerating that process at any given moment by such parameters as variability and baseline heart rate, and of prognosticating how long such a process might be allowed to continue without significant fetal depression or damage by the severity, repetitiveness, and duration of the pattern. Unfortunately, such a process is difficult to analyze quantitatively, making the task of pattern recognition and integration all the more important.

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CHAPTER 7

Umbilical Cord Blood Gases to Assess Fetal Condition at Birth

The assessment of the fetal condition at birth had traditionally been restricted to the Apgar score. It is interesting to note that this score, proposed originally by its namesake, Virginia Apgar, an anesthesiologist, was developed primarily to assess the need for newborn resuscitation (1). However, as investigators became interested in the influence of the process of labor and delivery on the long-term development of affected newborns, investigators began to assess the utility of the Apgar score in predicting adverse neurologic development. It became apparent that low Apgar scores, except in the extreme, had little predictive ability for such adverse outcomes. For example, an Apgar score of 0 to 3 at 5 minutes only increases the risk of cerebral palsy (CP) by 1%, whereas with an Apgar score of 0 to 3 even at 10 minutes, 90% of children did not develop CP (2,3). This should not be surprising as the primary complications in term babies, which are likely to adversely affect long-term development of the brain, are hypoxia and acidosis (perinatal asphyxia). But a low Apgar score is only a reflection of neurologic depression, of which acidosis may certainly be one cause, but is far from the only cause. Other causes of newborn depression include drugs or anesthetic agents; anomalies affecting the fetal brain, lungs, airway, or heart; airway obstruction; sepsis; pulmonary hypoplasia; and even the resuscitation process itself, as with intubation and suctioning for meconium. Thus, there should be and is an alternative for assessing the fetus at birth and that is the evaluation of umbilical cord blood gases. The assessment of the fetus for the presence, absence, and degree of acidosis at birth not only is potentially useful for the prediction of long-term sequelae but also has other important implications. For the depressed baby who is not acidotic, it guides the pediatrician to look for other causes of depression. If the baby is acidotic, it will assist the pediatrician in tailoring the resuscitative efforts. For the obstetrician, the assessment of umbilical blood gases will help, at least in retrospect, with the interpretation of the fetal heart rate (FHR) tracing, as will be discussed later in this chapter. In the case of a baby who later develops any neurologic dysfunction, the cord blood gases can be used to refute or confirm the possibility of perinatal asphyxia as a contributing cause, and this has implications for the treating neurologist as well as in the courtroom if the child's damage results in a malpractice suit. Thus, the evaluation of umbilical cord blood gases has become an important and common method of evaluating the depressed baby at birth.

PHYSIOLOGY OF FETAL ACID-BASE BALANCE

pH is a logarithmic representation of the quantity of hydrogen ions in fluid. Specifically, pH is the negative logarithm of the hydrogen ion concentration. So a pH of 7.0 is equal to a molar hydrogen ion concentration of 10^{-7} or, expressed numerically, .0000001 M H^+. In humans, it is critical that hydrogen ions are kept in a close balance both by the rate of production and by a process of either conversion or buffering with substances in blood and tissue, which prevents excess hydrogen ions from doing harm. The major buffers in blood are hemoglobin and bicarbonate. Hemoglobin essentially absorbs hydrogen ions and thus has a finite capacity to simply absorb acid and carry it to the kidneys where it can be excreted. In the fetus, the kidney's ability to excrete acid is relatively immature, and therefore, its role in acid buffering is minimal once the hemoglobin capacity has been saturated. The other major method of buffering excess H^+ is by the conversion of bicarbonate to carbonic acid (H_2CO_3) by the formula:

$$H^+ + CO_2 \leftrightarrow H_2CO_3 \leftrightarrow CO_2 + H_2O$$

Under normal circumstances, metabolism of sugars produces carbon dioxide (CO_2) and water, and CO_2 produced by the fetus is eliminated across the placenta. Under conditions of hypoxia, as with the adult, the fetus converts energy with a process known as anaerobic metabolism, which involves the conversion of sugars and fatty acids into pyruvic and lactic

acids resulting in excess H^+ ion production and a lowering of the pH. To prevent harm from these hydrogen ions to cells, tissues, and organs, these excess H^+ ions must be buffered by the mechanisms described above. As the pH drops during such anaerobic metabolism, the shift in the equation results in reduced bicarbonate and loss of this buffering capacity. Thus, the H^+ content increases (a drop in pH), and when this falls out of the normally very tight range of safe pH, an *acidosis* results. This type of acidosis resulting from an increased production of hydrogen ions by anaerobic metabolism (or by the direct production of other organic acids as in diabetic ketoacidosis) is called *metabolic acidosis*. Both hydrogen ions and the bicarbonate that buffers them are highly charged ions and move very slowly across the placenta. Thus, with a metabolic acidosis, the placenta's ability to clear hydrogen ions from the fetus or replenish bicarbonate from the mother is a relatively slow process, and a significant metabolic acidosis, when it is finally resolved, will take many hours for the placenta alone to reverse it.

The other type of acidosis seen in the fetus is known as *respiratory acidosis*. This results from an inability to clear from the blood stream the normally produced CO_2. In the fetus, the most common reason for this is compression or other reduction in flow in the umbilical cord vessels; although if the mother develops a respiratory acidosis, the fetus will accumulate more CO_2 and the gradient is changed as well. The fetus, as with the adult, produces CO_2 as a result of oxidative metabolism of sugars and fatty acids under normal well-oxygenated circumstances. The CO_2 produced by the fetus cannot be eliminated, as in the adult, through the lungs, so it is eliminated through the placenta into the maternal circulation and out of the maternal respiratory tract. As long as there is normal umbilical blood flow, placental exchange, and maternal respiration, the CO_2 diffuses very rapidly across the placenta. Normal umbilical arterial pCO_2 (reflecting that blood exiting the fetus to the placenta) has a value of about 50 mm Hg, whereas the venous pCO_2 exiting the placenta has a value of about 40 mm Hg. This relatively large difference demonstrates how rapidly CO_2 is cleared through the placenta. A respiratory acidosis reflecting retained CO_2 in the fetus, most commonly due to umbilical cord compression or other obstruction to umbilical blood flow, will clear very rapidly when the compression is released as the CO_2 in the umbilical artery is rapidly diffused across the placenta, the equilibrium of the equation is shifted away from the production of hydrogen ions, and the pH moves toward normal.

When the pH is low, it is important to distinguish between a respiratory acidosis and a metabolic acidosis; the bicarbonate value that can be measured is not an accurate reflection in and of itself. The concept of base excess or base deficit is used for this purpose. The pH value and the measured pCO_2 are plotted on a curve known as the Siggaard-Andersen normogram (Fig. 7.1). Now that measurement of the pH and pCO_2 is automated, this calculation is most commonly done on the same instrument that has a computer built in for this purpose. As seen in Figure 7.1, a line is drawn through the pCO_2 (point E) and the pH (point A), and the base excess is read from the plot at point C. The plot is variable based on the fetal hemoglobin concentration, and the base excess is read from the plot. An elevated base excess for an umbilical cord is >−9, and thus a low pH with an elevated base excess would be indicative of a metabolic acidosis and a low pH with a normal base excess would be a respiratory acidosis. In the latter case, the pCO_2 would be elevated, and in the former, the pCO_2 would be normal. A mixed respiratory and metabolic acidosis will have both an elevated base excess and an elevated pCO_2. The clinical picture of the baby at birth with a respiratory acidosis, depending on the severity, will be a low 1-minute Apgar score and, following resuscitation, a relatively normal 5-minute Apgar score, as the efforts of resuscitation will clear the elevated CO_2 rapidly and allow the pH to return to normal. A metabolic acidosis, when relatively severe, will be reflected in both low 1- and 5-minute Apgar scores. This is because with resuscitation, only after oxygen (O_2) reaches the cells and tissues is the anaerobic metabolism reversed and buffers are restored that the pH becomes normal, and this process takes considerably longer.

It is important to review the terminology as this is often confusing, and care must be used to express the status of fetal blood gases and their results as accurately as possible (Table 7.1).

Technique for Obtaining Umbilical Cord Blood Gases

To collect umbilical cord blood for analysis, a doubly clamped segment of the cord is cut away from the placenta immediately after delivery of the baby. Delay in clamping the cord segment for obtaining cord blood gases can significantly alter the pH and pCO_2. The blood samples can be obtained with disposable plastic syringes that have been flushed with heparin (1,000 U per cm^3). It is important to carefully expel all the heparin from the syringes, as heparin can alter the pH. Samples are obtained from both the umbilical artery and vein whenever possible. Umbilical arterial blood is often more difficult to obtain. If this proves to be the case, blood can be obtained from an artery on the surface of the placenta. The arteries are identified as they override the vein on the placental surface. Samples should be carefully labeled as umbilical artery or vein and with the time they are obtained. The samples are stable either in the clamped segment of the cord or in the syringe for up to 60 minutes and do not have to be transported to the laboratory on ice.

Normal Cord Blood Gas Values

The blood from the umbilical artery reflects information on the blood returning to the placenta from the fetus, so it is most informative about the state of the fetus at the time

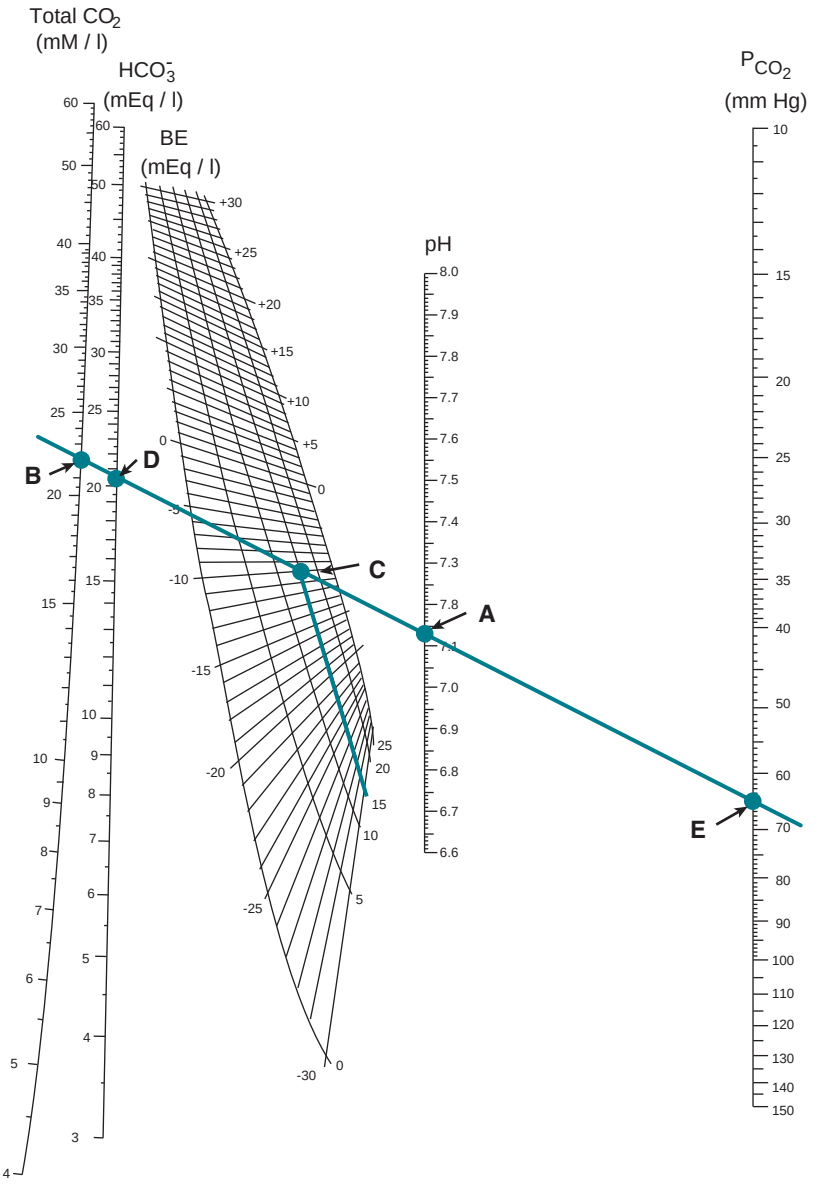

Figure 7.1. This is the Sigaard-Anderson normogram used to calculate base excess (or base deficit, the negative of base excess). A line is drawn through the pCO_2 (point E) and the pH (point A), and the base excess is read from the plot at point C. The plot is variable based on the fetal hemoglobin concentration, and the base excess is read from the plot.

TABLE 7.1 Terminology used to describe blood gases in the fetus

- Acidemia—increased H^+ in blood
- Acidosis—increased H^+ in tissue
- Asphyxia—hypoxia with metabolic acidosis
- Base deficit—[HCO_3] below normal
- Base excess—[HCO_3] above normal (the negative of base deficit)
- Hypoxemia—decreased O_2 in blood
- Hypoxia—decreased O_2 in tissue
- pH—negative log of [H^+]

of delivery. Blood from the venous side will be useful in confirming the accuracy of the values obtained from the artery and, as will be discussed, if an acidosis does exist confirming whether the acidosis is metabolic or respiratory. The values that are most important are the pH and the pCO_2. The pO_2 is only useful in confirming that the specimen obtained was actually from the umbilical cord and not from an umbilical venous catheter obtained from the newborn sometime later, as fetal pO_2 is substantially lower than newborn values. The pO_2 in and of itself is not informative because the level fluctuates so much minute to minute, especially in the final minutes before birth, and even very vigorous and subsequently normal fetuses can have very low pO_2 values.

Normal values for the umbilical cord artery and vein are shown in Table 7.2 (4–6). Values for term and preterm babies are similar (7).

It should be pointed out that these normal values have been derived for the most part from babies who have experienced labor and delivery. The labor process itself is associated with a decrement in pH and elevation of base excess (Fig. 7.2) (8). Blood gas values obtained from cordocentesis or from babies delivered electively by cesarean section without labor confirm this general trend toward a lower pH and higher base excess as labor progresses, as values thus obtained from nonlaboring patients and via cordocentesis show higher pH and lower base excess values (Table 7.3) (9–11). Thus, if a baby is delivered by cesarean section without labor, or in early labor, the normal values will be somewhat different, with the pH somewhat higher and the base excess somewhat lower.

Correlating Umbilical Cord Blood Gases with FHR Patterns

The pathophysiology of FHR patterns has been well established in animals and, in some cases, in humans with the use of fetal pulse oximetry and ultimately with the correlation of FHR patterns with umbilical cord blood gases at birth. For example, it is well established that the only trigger for late decelerations is a fall in fetal pO_2 usually due to inadequate exchange or delivery of O_2 within the placenta. As with the adult lung, the placenta's capacity for exchanging CO_2 far exceeds its ability to exchange O_2. Thus, with late decelerations, if the O_2 delivered to the baby is insufficient to support aerobic metabolism, anaerobic metabolism results in a buildup of organic acids, and a metabolic acidosis results. In such a case, the cord gases will exhibit a low pH, a normal pCO_2, and an elevated base deficit that exceeds 9. Since metabolic acidosis is only slowly corrected by the placenta, the blood gases in the umbilical vein will be similar to those in the umbilical artery. In the case of variable decelerations, most often due to umbilical cord compression, if the frequency and duration of the cord compression is severe enough, as in airway obstruction in the adult, accumulation of CO_2 will be the first event. If severe enough, this will result in a respiratory acidosis (12). If even more severe and the fetus can progress to an O_2 deficit as well, a combined respiratory and metabolic acidosis will result. In the former case of the respiratory acidosis, there will be a low pH, an elevation of pCO_2, and a normal base excess. A combined respiratory and metabolic acidosis would result in the umbilical artery in a low pH, an elevated pCO_2, and a base deficit of >9. Interestingly with a respiratory acidosis due to umbilical cord compression, most often there will be a disparity in pCO_2, and thus the pH, between the umbilical artery and vein. This occurs because, as previously explained, CO_2 can be rapidly cleared in the placenta and the pCO_2 in the umbilical vein will be considerably lower than in the artery as the blood flows through the placenta and the CO_2 diffuses into the maternal circulation. Thus, the artery will have a lower pH and a more elevated pCO_2, and the vein will have a lower or more normal pCO_2 and the pH will have risen back toward normal. Prolonged decelerations leading to bradycardia, tachycardia, and/or absent or minimal variability will all have unpredictable or variable umbilical cord blood gases because their etiologies and pathophysiology can vary from one patient to the next. For example, a bradycardia due to umbilical cord prolapse when intervention occurs promptly may result in only a respiratory acidosis at birth, but if intervention is more delayed, there can be a combined respiratory and metabolic acidosis. Abruptio placentae may have only a metabolic acidosis or may have a combined metabolic and respiratory acidosis. A tachycardia associated with fetal sepsis usually will have a normal pH, but as the sepsis becomes severe and the baby develops shock with cellular hypoperfusion, a metabolic acidosis followed by an acidemia may result. Thus, the combination of umbilical artery and umbilical vein blood gases along with their correlation with their associated FHR pattern can give insight into the cause of an acidosis, can help the clinician determine if the interpretation of the pattern was correct in some cases, and can assist the pediatrician with the resuscitation of the newborn, assist with determining the etiology of any newborn depression or subsequent complications, and possibly help guide any newborn treatment required. To some extent, the severity and the type of the acidosis (whether respiratory or metabolic) can assist, along with the complications occurring in the newborn, with the prognosis for long-term neurologic development of the child, the discussion of which follows.

TABLE 7.2 Normal values for umbilical cord blood gases

	Mean value	Normal range
Artery		
pH	7.26	7.15–7.38
pCO_2	50	35–70
Base excess	–3.0	–2 to –9
Venous		
pH	7.34	7.20–7.41
pCO_2	40	33–50
Base excess	–2.6	–1 to –8

Values are a composite of four studies evaluating cord blood gases in babies with normal Apgar scores and normal newborn courses (4–7).

Correlation of Umbilical Blood Gases with Long-term Outcome

As covered in Chapter 3, neurologic damage resulting from intrapartum asphyxia is only one of the many causes of CP and other encephalopathies. Prior to using umbilical cord

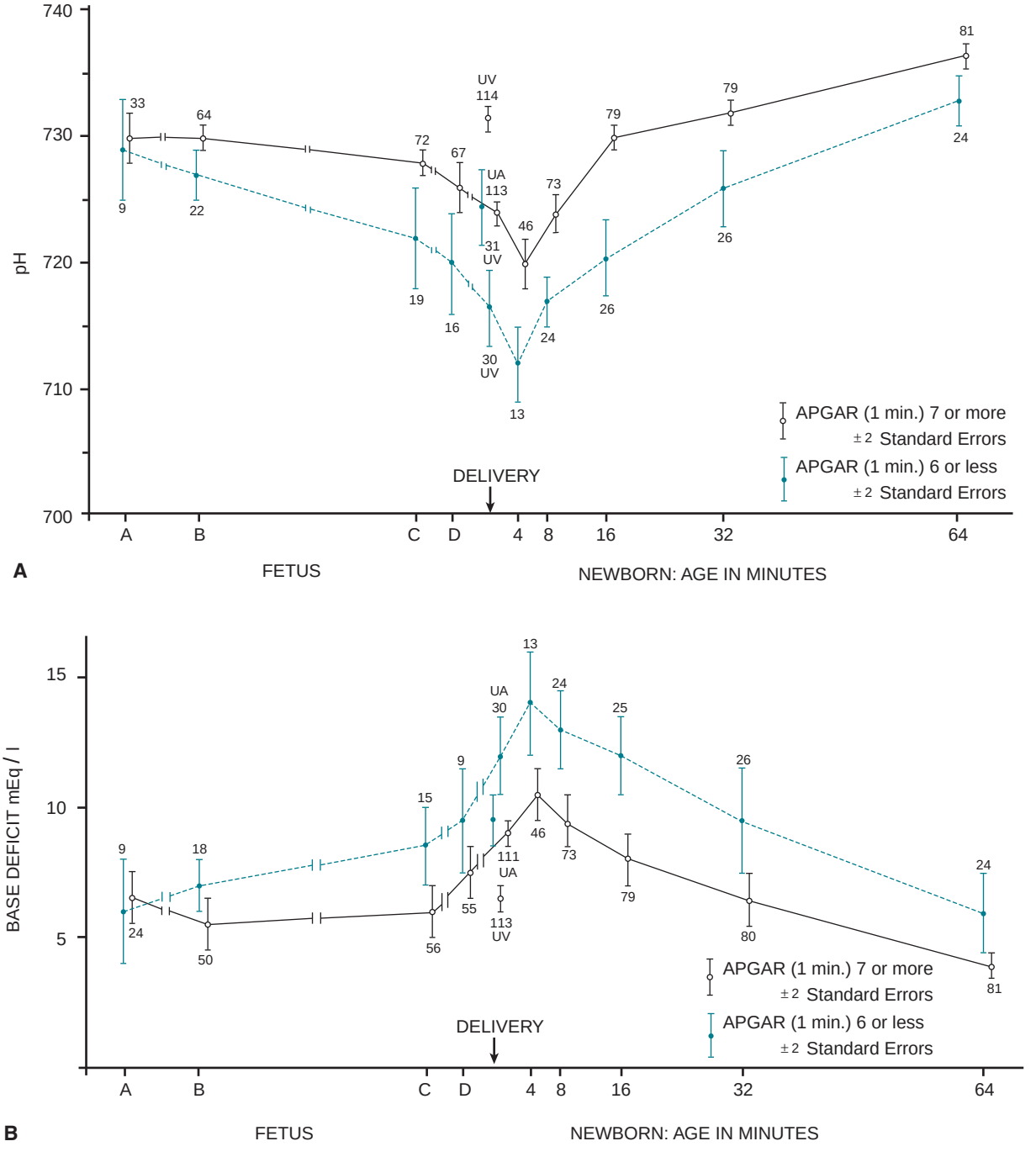

Figure 7.2. **A**: Normal pH (*solid line*) during labor, at delivery, and in the early newborn period is shown. In addition, the *dotted line* represents pH from fetuses and newborns delivered with 1-minute Apgar scores of 6 or less. Point A is early labor, B is at 5 cm dilation, C is at complete dilation, and D is just prior to delivery. 2B The *dark line* is the normal of base deficit in labor, and the *dotted line* represents the base deficit from depressed babies in labor. **B** is the normal course of base deficit (base excess is the negative of base deficit) in labor and in the early newborn period. As in A, the *dotted line* is from depressed newborns (From Modanlou H, Yeh SY, Hon EH, et al.: Fetal and neonatal biochemistry and Apgar scores. *Am J Obstet Gynecol* 117:942, 1973.)

blood gas assessment, it was common to base the prognosis of the fetus on the Apgar score and thus the need for resuscitation. Subsequent to the realization of the poor predictive value of the Apgar score as well as its nonspecific nature for understanding the cause of any depression, it became a practice to utilize umbilical cord pH in assessing fetal status at birth. As should be apparent now, the problem with using pH alone, especially when using pH alone from the umbilical

TABLE 7.3 Normal fetal blood gases and pH obtained by cordocentesis

Parameter (mean value)	22 Wk	28 Wk	34 Wk	40 Wk
UV (Ph)	7.416	7.407	3.398	7.388
UV (Po_2)	47.6	42.0	36.3	30.6
UV (PCO_2)	33.6	34.9	36.2	37.5
UV (HCO_3)	22.3	23.0	23.7	24.3
UA (pH)	7.390	7.379	7.368	7.357
UA (PO_2)	28.3	26.3	24.3	22.3
UA pressure (mm Hg)	3.8	5.2	6.5	—

From Creasy RK, Resnik R: *Maternal-fetal medicine: principles and practice*. 5th ed. Elsevier, Inc., Philadelphia, 2004.

artery, is that an elevated pCO_2 causing a respiratory acidosis cannot be distinguished from a metabolic acidosis. Because a respiratory acidosis has virtually no correlation with, and thus is not a cause of, neurologic damage (13), a pH alone does not provide the degree of information that a pH along with a calculated base deficit does. Thus, in studies that correlated pH with long-term outcome, as seen in Table 7.4, even in the extreme with a value of <7.0, there is little, if any, correlation between a low pH value and neonatal death or seizures, the best single neonatal indicator of neurologic damage (14).

On the other hand, when a base deficit is used in addition to the pH, there is a much stronger predictive value of a true metabolic acidosis in predicting neurologic damage. Low et al. (13) followed 233 term newborns, equally divided into babies with base deficits of <8, 6 to 12, 12 to 16, and >16. They found that moderate and severe encephalopathies were seen in 10% of those babies with base deficits 12 to 16 in 40% of those with a base deficit of >16, and in no babies with respiratory acidosis alone. These increases represent a 200-fold (12 to 16) and 800-fold (>16) increase in neurologic damage, and thus, the use of both pH and base deficit is the most highly predictive of damage and the most revealing of the cause of the acidosis.

WHICH PATIENTS SHOULD HAVE UMBILICAL CORD BLOOD GAS ASSESSMENT?

There is some controversy in the United States over whether all babies should have assessment of umbilical cord blood gases or just selected babies either depressed or at risk for hypoxia and acidosis. The argument in favor of testing all babies is that it may be of utility in defending against an allegation of failure to deliver a baby earlier and preventing an acidosis in a situation that results in a lawsuit. From a medical standpoint, there is no value in this practice because it has been shown that a baby with a normal Apgar score, even when

TABLE 7.4 Neonatal morbidity and mortality according to pH cutoff

pH	Neonatal deaths	Seizures	Both
7.15–7.19 (n = 2236)	3 (0.1%)	2 (0.1%)	1 (0.05%)
7.10–7.14 (n = 798)	3 (0.4%)	1 (0.1%)	0
7.05–7.09 (n = 290)	0	0	1 (1.1%)
7.00–7.04 (n = 95)	1 (1.1%)	1 (1.1%)	1 (1.1%)
<7.00 (n = 87)	7 (8.0%)[a]	8 (9.2%)[a]	2 (2.3%)

[a] P <.05.
From Goldaber KG Gilstrap LC Leveno KJ, et al: Pathologic fetal academia. *Obstet Gynecol* 78:1103–1107, 1991.

TABLE 7.5 Indications for umbilical cord pH: a proposed checklist

- Premature delivery <34 wk
- Moderate or thick meconium
- Any of the following nonreassuring patterns with 1 h of delivery:
  Persistent late decelerations
  Fetal tachycardia
  Absent variability (≤5 BPM)
  Severe variable decelerations
  Recurrent prolonged decelerations
  Bradycardia
- Intrapartum vaginal bleeding more than "bloody show"
- Maternal fever (≥100.4°F)
- Apgar score <7 at 1 and/or 5 min
- Unexpected fetal anomaly
- Instrumented vaginal delivery
- Shoulder dystocia
- Breech or other malpresentation delivered vaginally
- Any nonelective cesarean section
- Severe IUGR
- Maternal thyroid disease

a low pH is found (probably mostly due to laboratory error), shows no difference in immediate neonatal outcome, including complications and duration of stay, than a baby with a normal Apgar score and a normal pH. Thus, this practice is purely defensive medicine and adds substantial expense, potential worry, and unnecessary tests and interventions over a low pH value. The American Congress of Obstetricians and Gynecologists recommends cord blood sampling and analysis on babies with cesarean section for suspected fetal compromise, those with low 5-minute Apgar scores, with severe intrauterine growth restriction (IUGR), and with abnormal FHR tracings and on babies from mothers with maternal thyroid disease, intrapartum fever, and multifetal gestations. Because "abnormal FHR tracings" can be ambiguous, and since this means Category II FHR tracings can be ambiguous, an alternative list of indications for umbilical cord sampling and testing is provided in Table 7.5.

SUMMARY

Umbilical cord blood gas analysis is the most specific and accurate method of determining the condition of the baby at birth. An understanding of the physiologic basis of umbilical cord blood gases and their correlation with FHR patterns will enhance one's ability to predict and understand the baby's status at birth and in retrospect improve and validate the interpretation of the FHR pattern that preceded birth. Selected babies who are depressed at birth or otherwise at risk for acidosis should be sampled immediately following delivery. Both umbilical arterial and venous blood should be obtained whenever possible and should be analyzed for pH and pCO_2 and a base deficit/excess calculated.

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CHAPTER 8

Clinical Management of Abnormal Fetal Heart Rate Patterns

The general premise of electronic fetal heart rate (FHR) monitoring is that when the FHR pattern is normal, it is essentially certain that the fetus at that point in time is neither hypoxic nor acidotic. The opposite is, however, often not the case; that is, when the FHR is abnormal, the fetus is often vigorous and not significantly acidotic at birth. The previously used term "fetal distress" was often inaccurate. As recommended by the ACOG Committee on Obstetric Practice in 2005, the nonspecific and imprecise term "fetal distress" should be replaced by "nonreassuring fetal status," which is to say that all of the data available for assessing the fetus do not reassure the clinician (1). In addition, this should be followed by a further description of findings (e.g., types of FHR patterns, baseline changes, biophysical profile score). In addition, there is now an entirely new recommendation for nomenclature of FHR tracings (see Chapter 6).

There is no agreed definition of fetal distress because the term has various meanings for different people. Although not interchangeable, the terms fetal distress, fetal asphyxia, and asphyxial trauma are often freely substituted and erroneously equated. Such inconsistencies and inaccuracies create problems for caregivers, particularly in the increasingly litigious climate of our society. *Dorland's Medical Dictionary* defines asphyxia as a "lack of oxygen and respired air resulting in impending or actual cessation of life." Its Greek origin, "a stopping of the pulse," gives the definition of birth asphyxia an imprecise meaning at best. In the past, asphyxia has been the assumed cause of depressed newborns with low Apgar scores. In the fetus, asphyxia refers to a lack of oxygen resulting in metabolic acidosis (2). Asphyxial trauma suggests cellular damage, particularly but not exclusively to the central nervous system (CNS), subsequent to some antepartum or intrapartum compromise. Neither fetal hypoxia nor asphyxia is necessarily associated with cellular death leading to fetal morbidity or mortality (3). Indeed, when recognized, the hope is that intervention significantly mitigates the risk of adverse outcomes. For any number of reasons, the correlation between an abnormal FHR and adverse outcome is poor.

The American College of Obstetricians and Gynecologists carefully reviewed the relationship between FHR patterns, Apgar scores, umbilical cord gases, newborn course, and subsequent outcome. Specific criteria to establish an acute intrapartum event being sufficient to cause cerebral palsy are as follows:

1. Evidence of metabolic acidosis in fetal umbilical artery blood at birth (pH <7.00 and base deficit ≥12 mmol/L)
2. Early onset of severe or moderate encephalopathy in infants born at or beyond 34 weeks' gestation
3. Cerebral palsy of the spastic quadriplegic or dyskinetic type
4. Exclusion of other identifiable etiologies such as trauma, coagulation disorders, infectious conditions, or genetic disorders (4)

Obtaining an umbilical artery blood gas assessment is critical in establishing this condition (5). We do not recommend routinely obtaining a blood specimen pH assessment on all deliveries as often (about 5% to 10%) an abnormal cord pH will be found in a baby with normal Apgar scores. These babies rarely have complications known to be correlated with acidosis or other problems in the newborn period. Hence we do not find that a cord pH is helpful in a newborn with normal Apgar scores. We do recommend obtaining a specimen of umbilical arterial blood for pH, pCO_2, and base deficit in the following situations: unexpectedly depressed newborns; Category III FHR pattern immediately before delivery, especially when operative delivery has been performed for that indication; babies with anomalies or other situations where the baby is likely to go to the neonatal intensive care unit; and very premature infants (≤32 weeks).

Ideally, the goal of FHR monitoring is to detect fetal hypoxia at its earliest stage and to attempt to prevent asphyxic damage resulting from prolonged and severe hypoxia. The

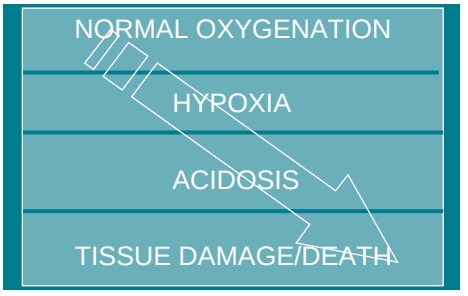

Figure 8.1. Model for declining fetal oxygenation with progressive development of hypoxia, metabolic acidosis, damage, and death.

progression of normoxia to hypoxia to metabolic acidosis to asphyxic damage and ultimately death is believed to occur in that order (Fig. 8.1). When the FHR pattern suggests hypoxia, all measures short of operative delivery (hydration, position change, supplemental oxygen, decreasing/discontinuing oxytocin, etc.) should be used to try to reverse the situation. If the hypoxic pattern cannot be reversed, then the fetus should be delivered expeditious. Clinical judgment needs to be stressed since if birth occurs because the fetus is thought to be hypoxic but not acidotic, then far too many unnecessary operative deliveries will be done, for the majority of hypoxic babies do not become acidotic, are vigorous at birth, and have normal outcomes.

Normal FHR patterns include those with accelerations, normal baseline rate, and normal variability. Early decelerations and mild variable decelerations are associated with normal Apgar scores, no acidosis at birth, and normal perinatal outcome. Abnormal patterns include the more severe forms of variable deceleration, persistent late decelerations, prolonged decelerations, and various atypical or preterminal patterns. Such patterns are usually associated with normal Apgar scores, but low Apgar scores may accompany such patterns (6).

A most important concept that must be realized in evaluating and managing abnormal FHR patterns is the issue of the normal progression of these changes when they are due to hypoxia. For example, tachycardia can be a result of hypoxia (see Fig. 6.20), but only when there are associated decelerations suggestive of hypoxia. That is to say in the laboring patient, persistent late, moderate-to-severe variable, or prolonged decelerations are the first indicators of hypoxia and virtually always precede tachycardia if the increase in heart rate is due to hypoxia. Similarly, loss of variability or disappearance of accelerations will not be the first sign of hypoxia and does not require further evaluation or intervention unless associated decelerations suggest a progressive hypoxia. The only exception to this can be the fetus who has tachycardia, absent or decreased variability, and/or no accelerations when the monitor is first placed on the patient. In this situation, one cannot be sure that no hypoxic deceleration pattern preceded these findings.

LATE DECELERATIONS

ACOG Definition (2010)

Late deceleration

- Visually apparent usually symmetrical gradual decrease and return of the FHR associated with a uterine contraction
- A gradual FHR decrease is defined as from the onset to the FHR nadir of 30 seconds or more.
- The decrease in FHR is calculated from the onset to the nadir of the deceleration.
- The deceleration is delayed in timing, with the nadir of the deceleration occurring after the peak of the contraction.
- In most cases, the onset, nadir, and recovery of the deceleration occur after the beginning, peak, and ending of the contraction, respectively.

Late decelerations are found in association with uteroplacental insufficiency and imply some degree of fetal hypoxia (7). A decrease in pO_2 detected by the fetal brain is the only trigger for late decelerations. In their mildest form, late decelerations are associated with normal or increased FHR variability. The pattern is characteristically described as uniform, appearing consistently from one contraction to the next. This rule is common once fetal hypoxia is established since in the early stages of developing fetal hypoxia, the pattern may be intermittent (Fig. 8.2). While it would seem prudent to take measures to optimize uterine blood flow at this stage, intermittent late decelerations with good or increased FHR variability require close watching and may not become any more severe. In this situation, the patient should be laboring in the lateral position, she should be well hydrated, oxytocin should be decreased or discontinued if contractions seem excessive, and she should be receiving oxygen (Fig. 8.3). If the late decelerations have occurred in association with decreased maternal blood pressure following conduction anesthesia or following the administration of an antihypertensive agent in a patient with hypertension, appropriate measures should be taken to restore blood pressure and placental perfusion (Table 8.1).

If persistent late decelerations develop despite maximizing uterine blood flow, the physician is obligated to be sure that a metabolic acidosis has not developed. The simplest and most straightforward way of ruling out acidosis is to look for spontaneous accelerations (Fig. 8.4) or, in their absence, attempt to elicit an acceleration with acoustic or scalp stimulation (Fig. 8.5A). In the presence of late decelerations, FHR accelerations, whether spontaneous or elicited, rule out acidosis. The absence of any acceleration is associated with an approximate 50% incidence of acidosis. When no acceleration is elicited, previous clinical alternatives included continuous fetal pulse oximetry monitoring (Fig. 8.5B) or intermittent scalp pH (Fig. 8.6). Unfortunately, fetal pulse oximetry monitoring is no longer available in this country,

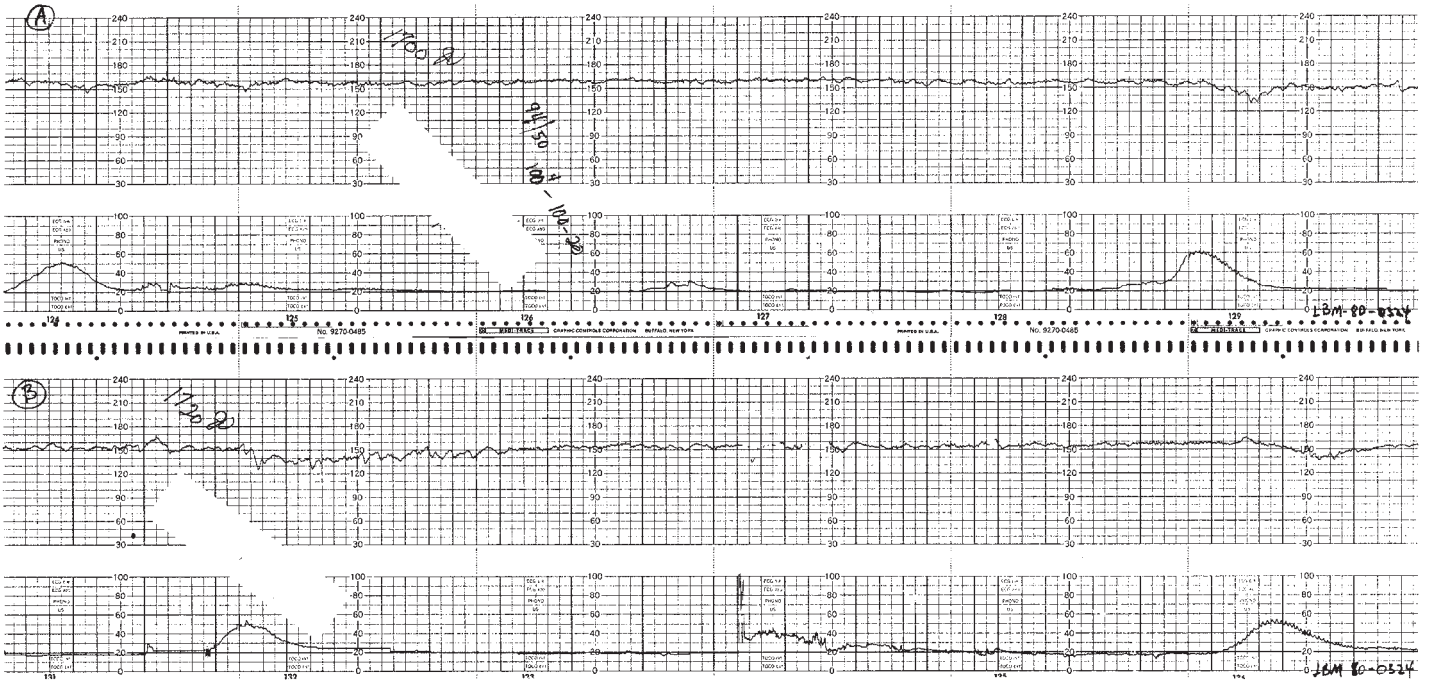

Figure 8.2. Intermittent late decelerations in early labor. Note lack of accelerations.

and fetal scalp blood sampling is rarely performed. As long as the late decelerations persist and the pattern unchanged, efforts to rule out acidosis must be repeated at least every 30 minutes. The presence of repeated accelerations allows the clinician to follow such patterns as long as the labor is progressing satisfactorily.

The discussion thus far has concerned the management of late deceleration in association with good variability. An FHR pattern of persistent late decelerations with complete absence of variability is much more ominous and almost always associated with fetal acidosis (Fig. 8.7). When encountered in either the antepartum or intrapartum period, our approach is to discontinue uterine stimulation and expedite delivery. This is most often via cesarean section since these patients are unable to tolerate labor without continuing to experience persistent late decelerations (8).

A common cause of late decelerations is excessive stimulation of contractions. Frequently, discontinuation of oxytocin results in improvement of the FHR (Fig. 8.8). The careful reinstitution of oxytocin at a lower rate often results in continued labor progress without continued late decelerations.

Another clinical situation that arises not infrequently is when late decelerations are persistent but when the oxytocin is discontinued, the decelerations resolve. Yet, without augmentation, the labor is inadequate. Following some period of time, the oxytocin is restarted, and late decelerations appear again. The clinician may choose to monitor the fetus closely and allow labor to continue as long as there is no evidence of acidosis. Alternatively, the choice may be to deliver the fetus expeditiously. This has been termed fetal intolerance to labor and has become a common indication for cesarean delivery for patients in labor.

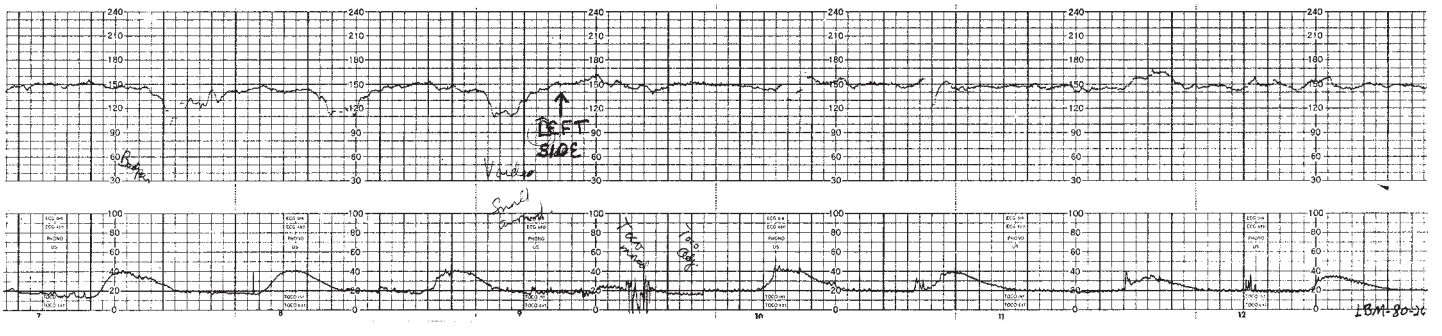

Figure 8.3. Late decelerations corrected by turning patient on her side.

TABLE 8.1 Medical management of late deceleration

1. Place patient on side.
2. Administer O_2 (100%) by tight face mask.
3. Discontinue oxytocin.
4. Correct any hypotension.
   (a) Appropriate position change
   (b) Intravenous hydration with appropriate fluid
   (c) Reserve pharmacologic pressor treatment (ephedrine) for severe or unresponsive hypotension due to conduction anesthesia.

VARIABLE DECELERATIONS

ACOG Definition (2010)

Variable deceleration

- Visually apparent abrupt decrease in FHR
- An abrupt FHR decrease is defined as from the onset of the deceleration to the beginning of the FHR nadir of <30 seconds.
- The decrease in FHR is calculated from the onset to the nadir of the deceleration.
- The decrease in FHR is 15 beats per minute (BPM) or greater, lasting 15 seconds or greater, and <2 minutes in duration.
- When variable decelerations are associated with uterine contractions, their onset, depth, and duration commonly vary with successive uterine contractions.

Variable decelerations are most frequently the result of umbilical cord compression. It is the most common periodic change observed in laboring patients. The pattern is characterized by abrupt decreases in FHR to levels as low as 50 BPM. The pattern varies from one moment to the next and may be influenced by such simple things as maternal position changes. While the pattern is the most profound appearing of the periodic FHR changes, the vast majority of the time it cannot be considered nonreassuring, because, unless the cord occlusion is frequent, prolonged, and severe, there is no increased risk of low Apgar scores, fetal metabolic acidosis, or other significant neonatal morbidity. The problem with variable decelerations is that they may suddenly become severe. Variable decelerations are less predictable than late decelerations, which tend to follow a slowly deteriorating course, allowing their development to be watched over time. Also, because variable decelerations can change from one contraction to the next, it is hard to know what to expect in the near future. This makes the management of this pattern especially difficult, particularly when working in a setting without the capability to move rapidly to operative delivery.

We have found that it is much easier to define the limits of reassuring variable decelerations than it is to give absolute criteria for when variable deceleration represents sufficient evidence of hypoxia to demand operative intervention. The following four criteria may be used as a guide to the limits of reassuring variable deceleration (Fig. 8.9):

1. The FHR decelerations last no more than 40 to 60 seconds on a repetitive basis.
2. The return of the FHR to the baseline is abrupt. There is no persistent "late component" manifested by a slow return or a late deceleration after the return.
3. The baseline FHR is not increasing.
4. The FHR variability is not decreasing.

While severe variable decelerations, increasing baseline heart rate, and loss of variability in the face of progressive variable decelerations are reasonably reliable indicators of progressively severe cord compression and developing hypoxia, this may not be the case with a slow return to baseline. As described in the chapter on the physiology of FHR patterns, there may be two and possibly three potential causes of a slow return to baseline. First, progressively severe cord compression may result in sufficient residual fetal hypoxia as to result in this pattern (see Fig. 6.21). The other primary mechanism is a combination of late and variable decelerations caused by two different mechanisms: cord compression and placental insufficiency. Finally, there may be a third, more benign cause, perhaps due to slower than usual release of the cord compression. In a recent review of variable decelerations

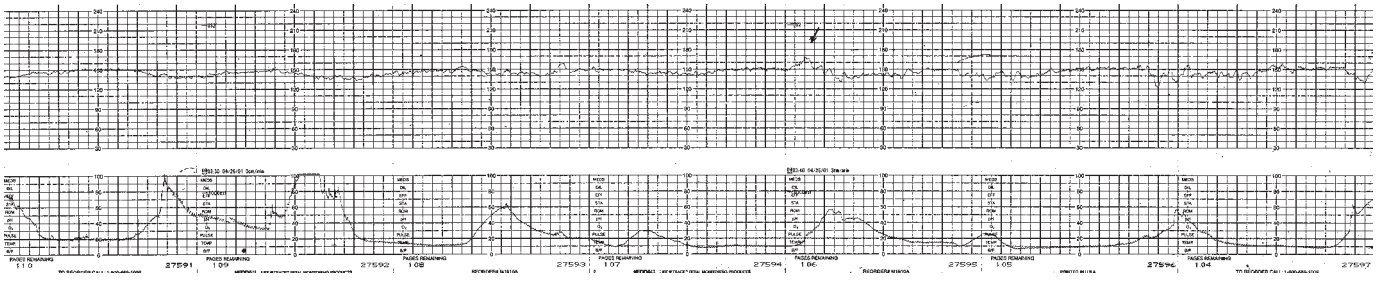

Figure 8.4. Persistent late decelerations. Note the presence of a spontaneous acceleration at panel 27595 (*arrow*). The presence of the acceleration virtually eliminates, at this point in time, any likelihood of a metabolic acidosis.

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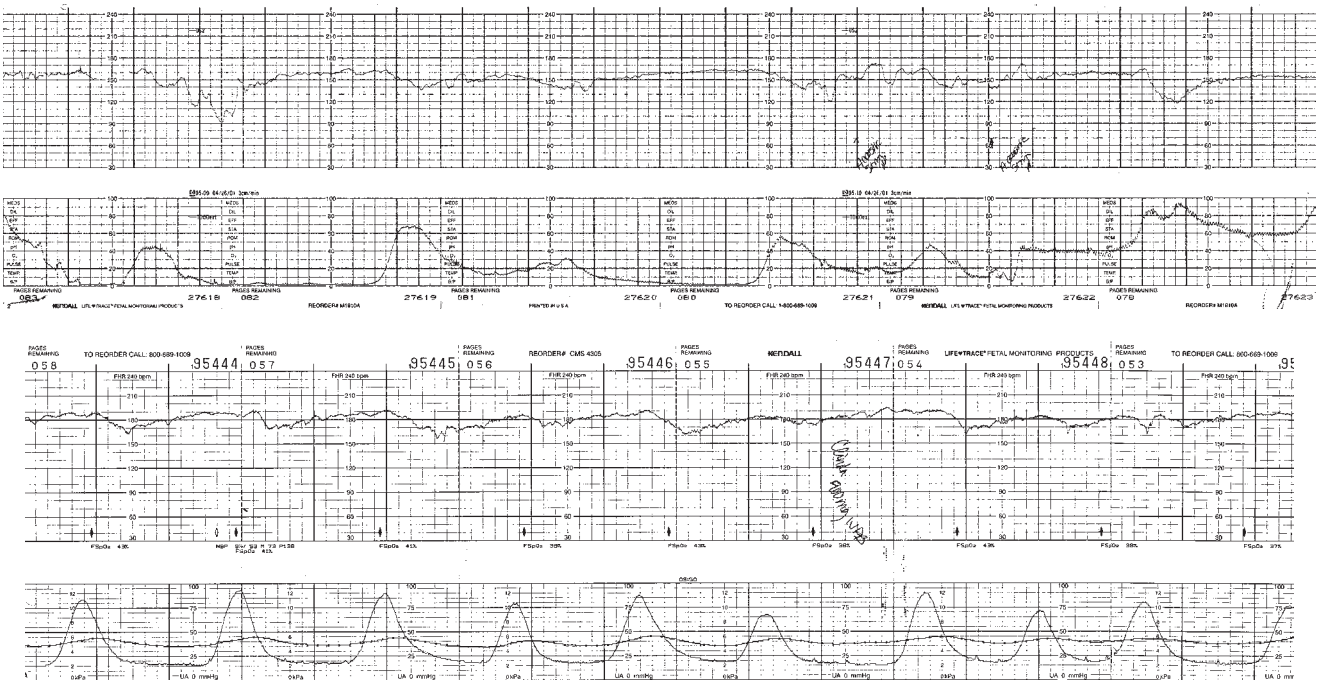

Figure 8.5. **A, B:** Both cases illustrate persistent late decelerations. In A, the clinician is reassured of the absence of a metabolic acidosis with an acceleration following acoustic stimulation. In B, reassurance is attained using fetal pulse oximetry, which reveals a continuous fetal oxygen saturation above 30%.

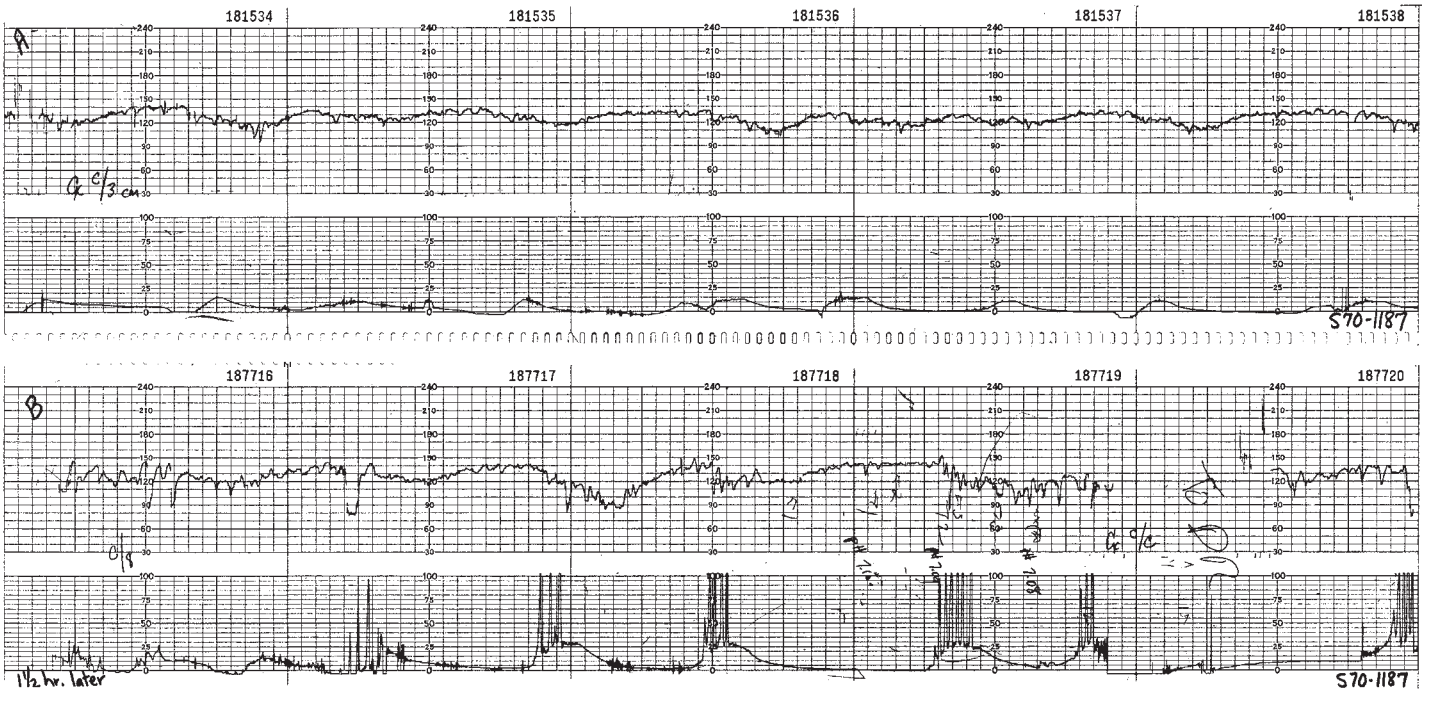

Figure 8.6. Late decelerations with good variability are seen in the **upper panel**. The **lower panel** is 1½ hours later, showing a continuation of late deceleration with good variability. The pH taken at this time was 7.08, suggesting that earlier intervention might have been advisable.

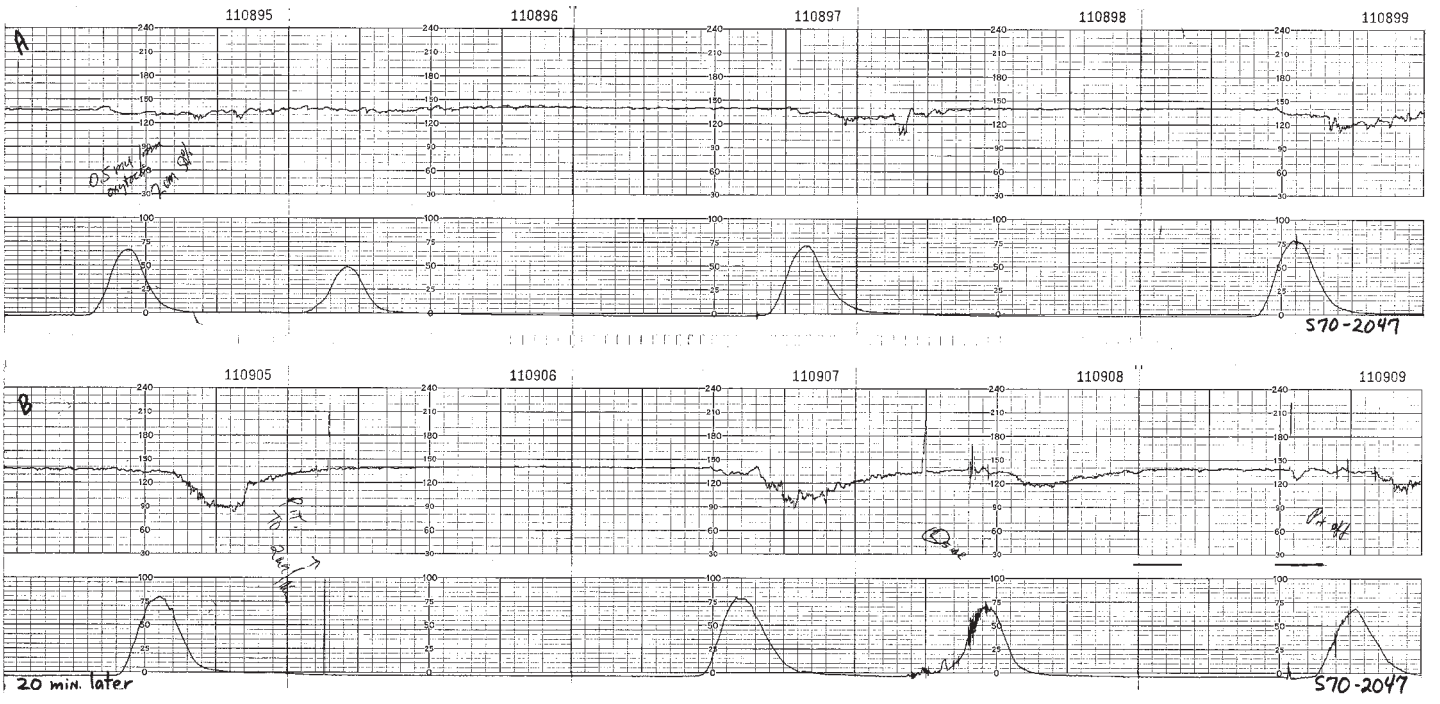

Figure 8.7. Late deceleration with poor variability. (From Paul R, Freeman R: *Selected records of intrapartum fetal monitoring with self instruction*. USC Publishers, Los Angeles, 1971, with permission.)

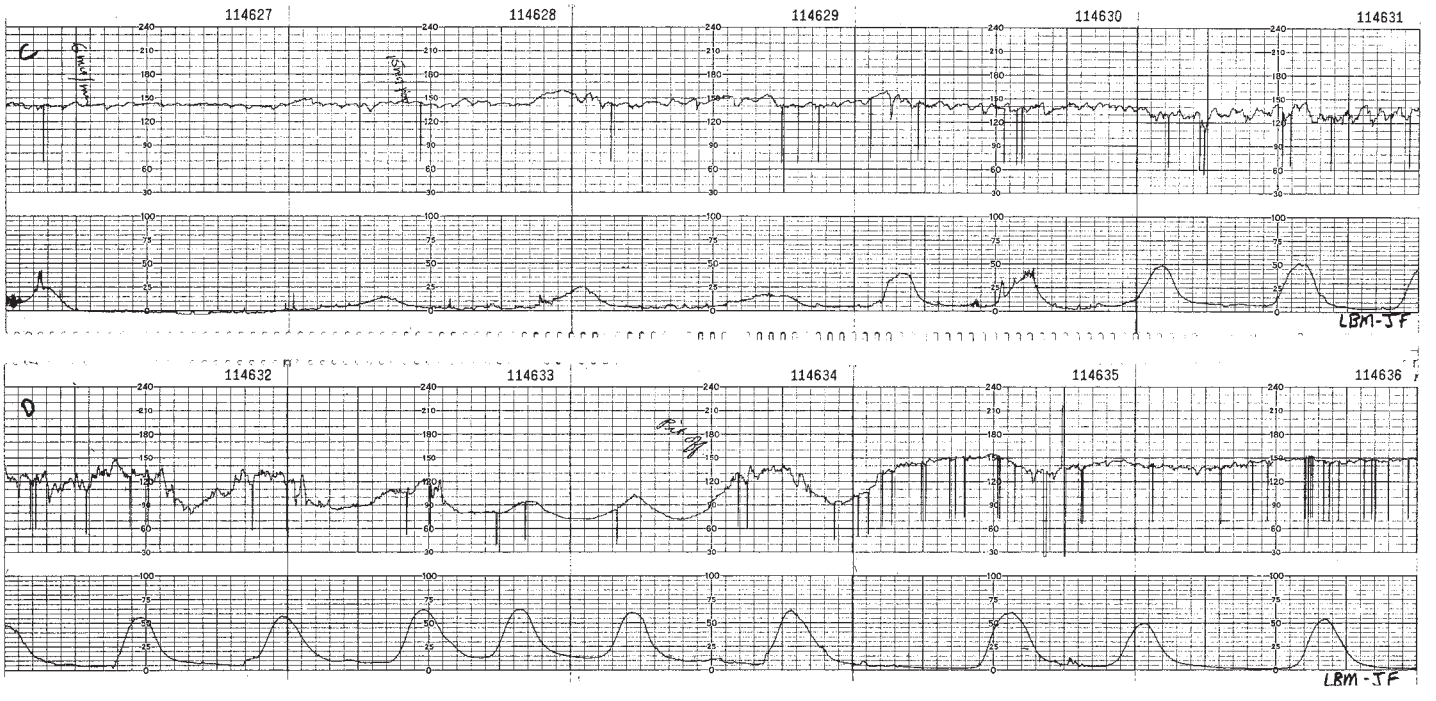

Figure 8.8. Late deceleration due to oxytocin and corrected by stopping the oxytocin infusion when it says "Pit off" in **section 114634**. (From Paul R, Freeman R: *Selected records of intrapartum fetal monitoring with self instruction*. USC Publishers, Los Angeles, 1971, with permission.)

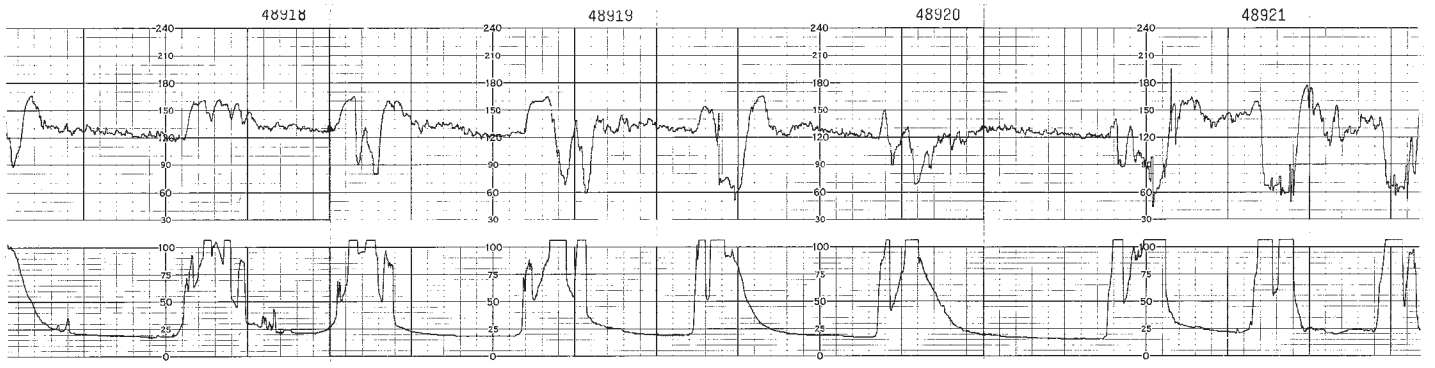

Figure 8.9. Reassuring variable decelerations.

with slow return to baseline in patients who were also being monitored with pulse oximetry, we found that when these patterns were preceded by severe variable or persistent late decelerations, there was indeed an increased likelihood of significant hypoxia. However, when such patterns were preceded by neither, there was no more likelihood of hypoxia than in patients with mild variable decelerations (9).

When criteria for nonreassuring variable decelerations are met and the pattern persists, the ability to effect immediate delivery is crucial. Increasing baseline, loss of variability, and/or delayed return to baseline can evolve in a short time with variable decelerations (Figs. 8.10 and 8.11). Similar to late decelerations, when variable decelerations meet any of the previous criteria as a nonreassuring pattern, one must either be reassured of the absence of acidosis, using the presence of spontaneous or elicited accelerations or move expeditiously to delivery.

It is useful from a management perspective to categorize variable decelerations based on their most likely cause (Table 8.2). Variable decelerations due to oligohydramnios usually appear relatively early in labor, and these are best reversed using amnioinfusion. Probably the most common cause of variable decelerations are nuchal cords. Nuchal cords probably cause decelerations, not due to compression, but due to the cord tightening, becoming stretched as the fetus descends into the pelvis. Because in most labors, the rapid phase of descent usually begins between 8 and 10 cm of dilation, it is common for nuchal cords to manifest variable decelerations at this stage (Fig. 8.12). These patterns are not likely to benefit from amnioinfusion or maternal repositioning. If the pattern becomes nonreassuring, the patient will often benefit by having her push less frequently, giving the fetus more time to recover from the cord compression between contractions (Fig. 8.13).

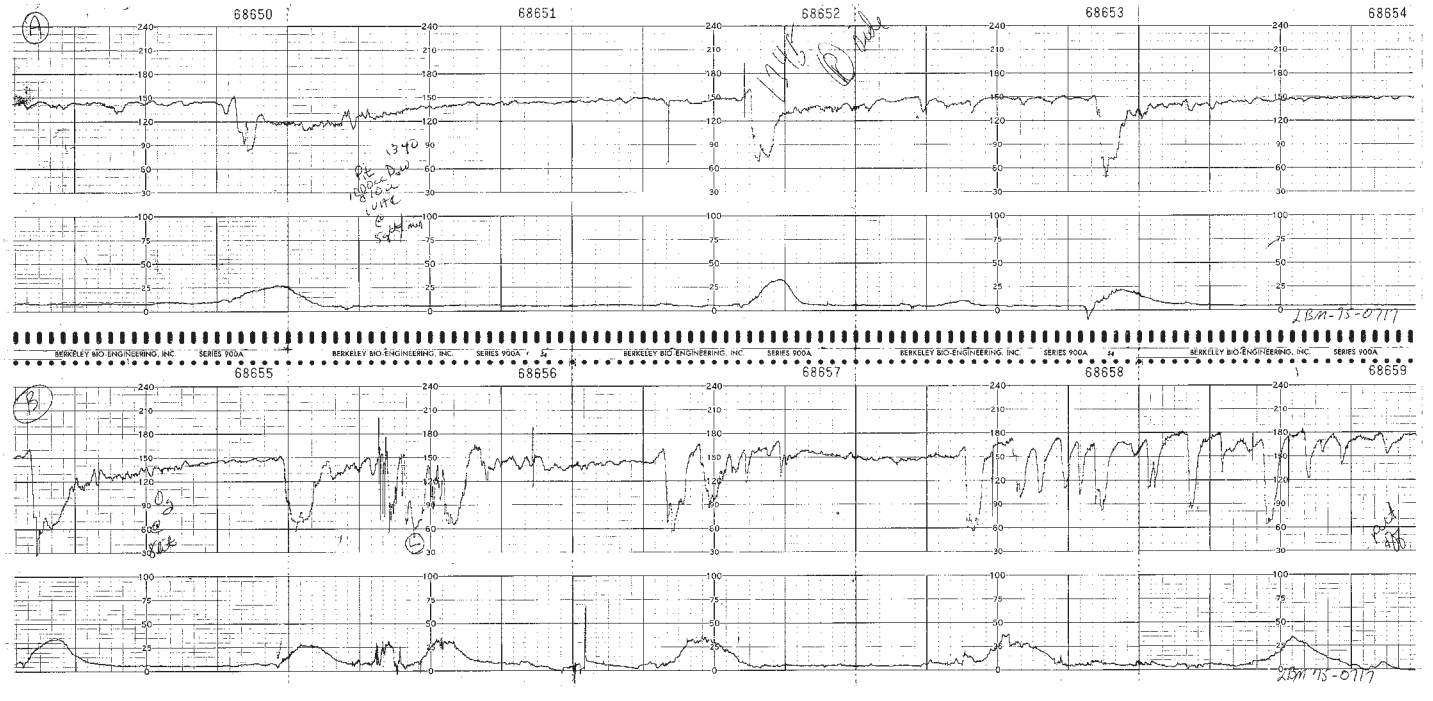

Figure 8.10. Variable deceleration with a rising baseline and slow return to baseline. This is an abnormal pattern.

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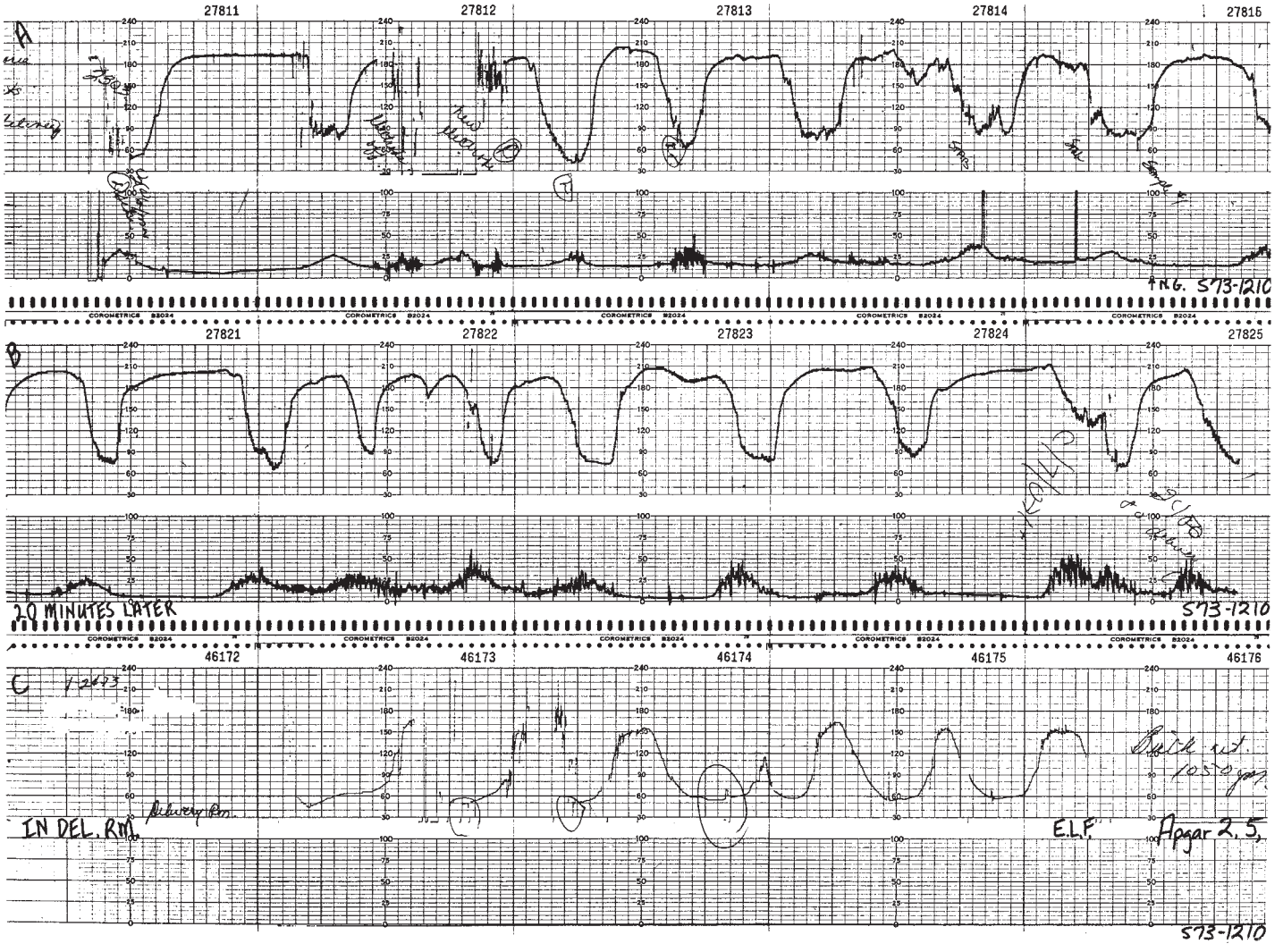

Figure 8.11. Variable deceleration with tachycardia and loss of fetal heart rate variability. This pattern is concerning. (From Paul R. Petrie R: *Fetal intensive care current concepts*. USC Publishers, Los Angeles, 1973, with permission.)

TABLE 8.2 Management classification of variable declarations

Etiology of cord compression	Typical appearance	Treatment
Nuchal cord	8–10-cm dilation	May consider altering pushing efforts to allow more recovery between contractions
Oligohydramnios	Early in labor	Amnioinfusion
Cord prolapse	Sudden onset, often at the time of membrane rupture	Elevation of presenting part, immediate cesarean section
Unusual cord compression	Variable timing and shape	Repositioning
Short cords	—	—
Other cord entanglement	—	—
True knots	—	—

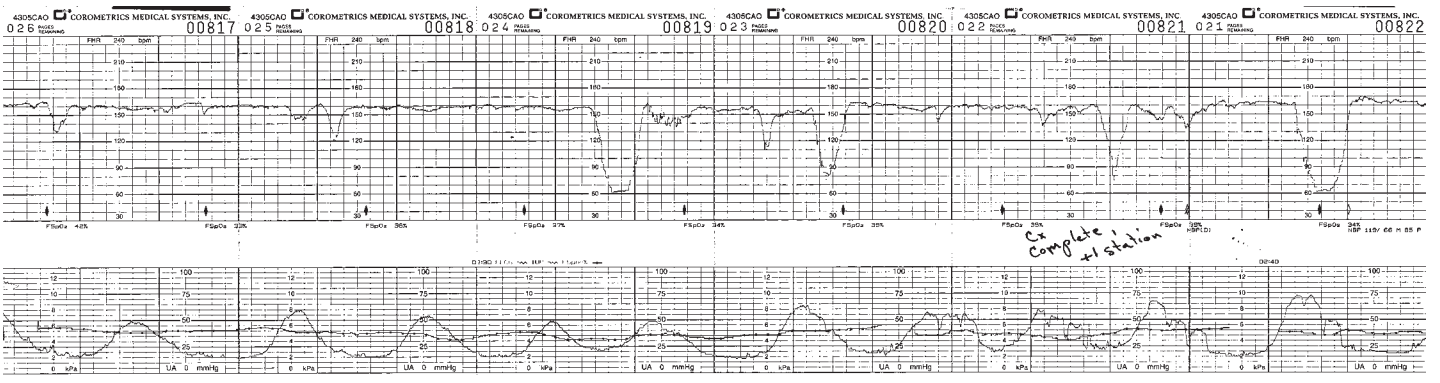

Figure 8.12. The new onset of variable decelerations occurring late in labor often heralds the transition to the second stage of labor. As seen in this patient, shortly after the appearance of the first deep variable deceleration, she begins spontaneously pushing and is found to be completely dilated.

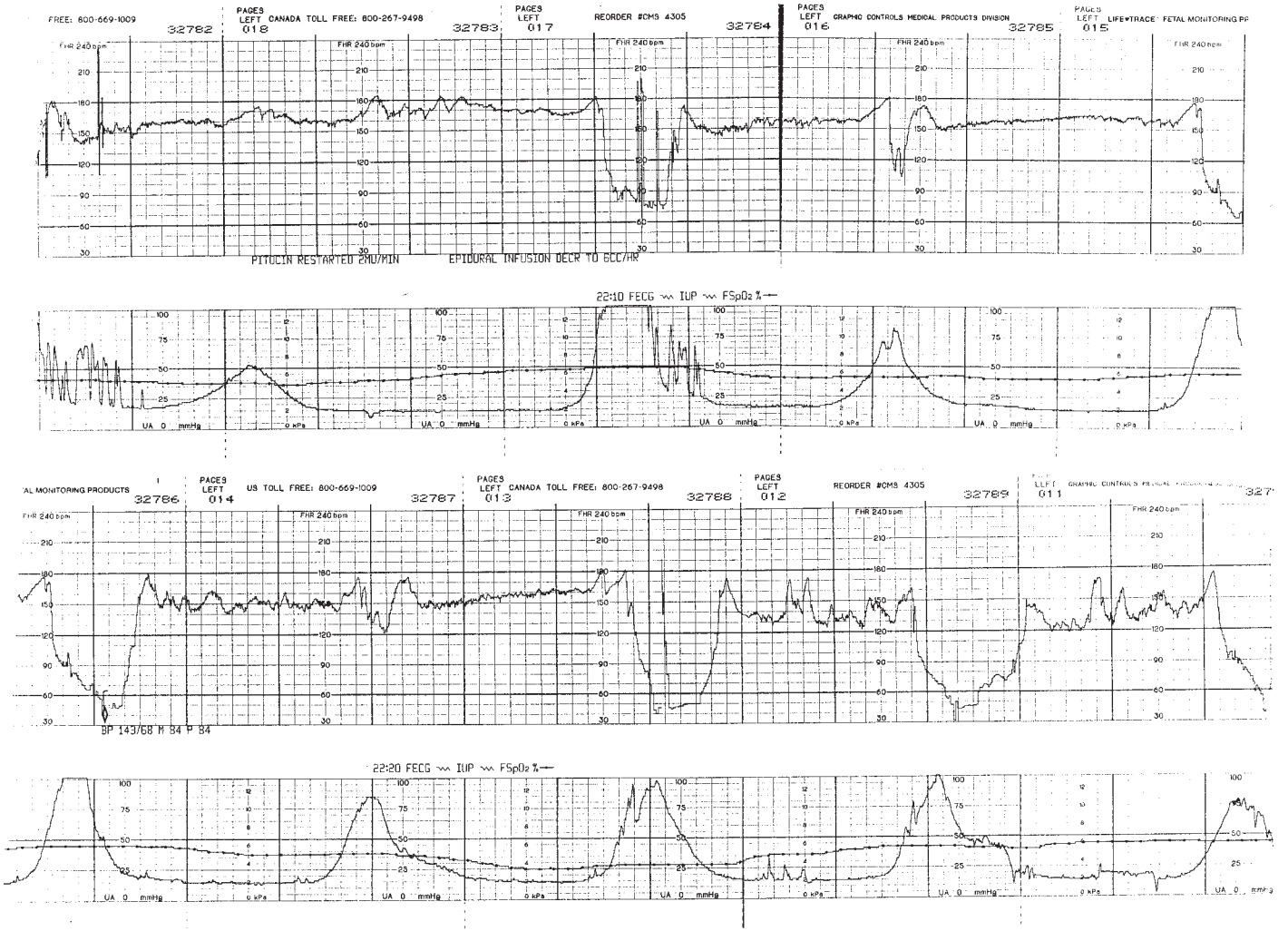

Figure 8.13. This patient is having rather severe variable decelerations in the second state of labor. Note that the depth and duration of the deceleration diminishes when the patient is not pushing. This is an effective method to allow additional recovery when significant variable decelerations are occurring in the second stage of labor.

A rare cause of variable or prolonged deceleration is umbilical cord prolapse. When significant variable or prolonged decelerations suddenly appear, this etiology should be considered and a pelvic examination performed immediately because cord prolapse virtually always requires elevation of the presenting part and immediate cesarean section. Finally, there is a group in which no good treatment other than repositioning may help relieve the cord compression. This group includes cord entanglement other than nuchal cords, such as around a fetal extremity or body, a true knot in the cord, or a short cord. The presentation of these may occur at any time during labor (Fig. 8.14).

Generally speaking, persistent and uncorrectable nonreassuring variable decelerations in early labor are best managed with delivery. On the other hand, during the second stage of labor, it is very common to see variable decelerations with maternal pushing. With continued pushing, these decelerations may appear to be concerning (Fig. 8.15). However, if progress is being made with descent of the presenting part, and the baseline heart rate level and variability remain unchanged, it is better to allow labor to progress than to attempt a difficult midpelvic delivery. It is common to see variable decelerations lasting more than a minute at 2- to 3-minute intervals during the second stage of labor. Other than position change and attempting to have the mother not push with each contraction, we continue to monitor and allow labor to continue as long as the baseline rate is not increasing and baseline variability is present.

The clinical guideline for management of patients with variable decelerations is, then, to be reassured if the pattern does not exceed the previously stated criteria for nonreassuring variable decelerations and to treat the patient expectantly. If these criteria are exceeded, the general rule is that the closer one estimates vaginal delivery to be, the higher

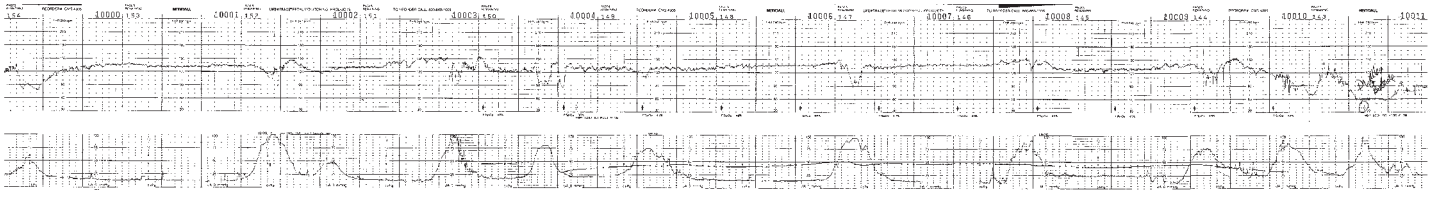

Figure 8.14. These variable decelerations are typical of "unusual cords." Their frequency, depth, duration, and shape are inconsistent, and they do not bear a regular relationship to contractions. At delivery the umbilical cord was noted to be quite short (11 in).

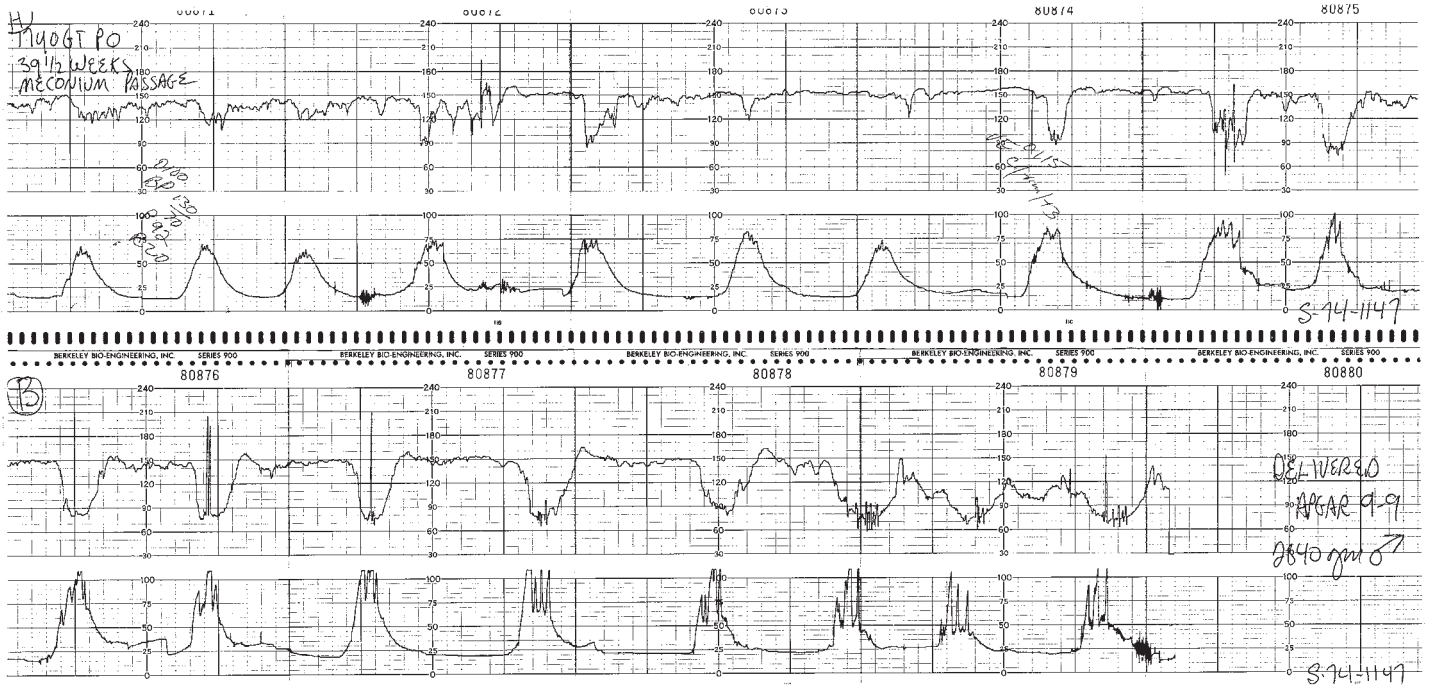

Figure 8.15. Variable deceleration of progressive severity occurring at the end of the second stage of labor.

TABLE 8.3 Medical management of severe variable decelerations

1. Change position to where FHR pattern is the most improved. Often Trendelenburg is helpful.
2. Discontinue oxytocin if running.
3. Check for cord prolapse or imminent delivery by vaginal examination.
4. Administer 100% O_2 by tight face mask.
5. Consider therapeutic amnioinfusion.

FHR, fetal heart rate.

will be the threshold for intervention (Table 8.3). With the introduction of both prophylactic and therapeutic amnioinfusion, the frequency of variable decelerations should be decreased, often making management decisions much easier. Variable decelerations are responsible for most unnecessary cesarean sections performed when the physician is not experienced with fetal monitoring, and they must have been a major reason for operative intervention when only auscultatory FHR monitoring was available.

PROLONGED DECELERATIONS

ACOG Definition (2010)

Prolonged deceleration

- Visually apparent decrease in the FHR below the baseline
- Decrease in FHR from the baseline that is 15 BPM or more, lasting 2 minutes or more but <10 minutes in duration
- If a deceleration lasts 10 minutes or longer, it is a baseline change.

Prolonged decelerations lasting several minutes and occurring as more or less isolated events will be observed either in association with identifiable causes or without apparent etiology. If there is no known cause, prolonged decelerations may represent umbilical cord compression of a severe degree or may represent a catastrophic event such as placental abruption, uterine rupture, or bleeding from vasa previa. The following is a list of potential causes of prolonged decelerations that may be identified and managed during the intrapartum period:

1. Hypertonic or prolonged contractions (spontaneous or oxytocin-induced) (Fig. 8.16)
2. Vaginal examination (Fig. 8.17)
3. Application of internal fetal scalp electrode
4. Fetal scalp blood sampling
5. Prolapsed umbilical cord (Fig. 8.18)

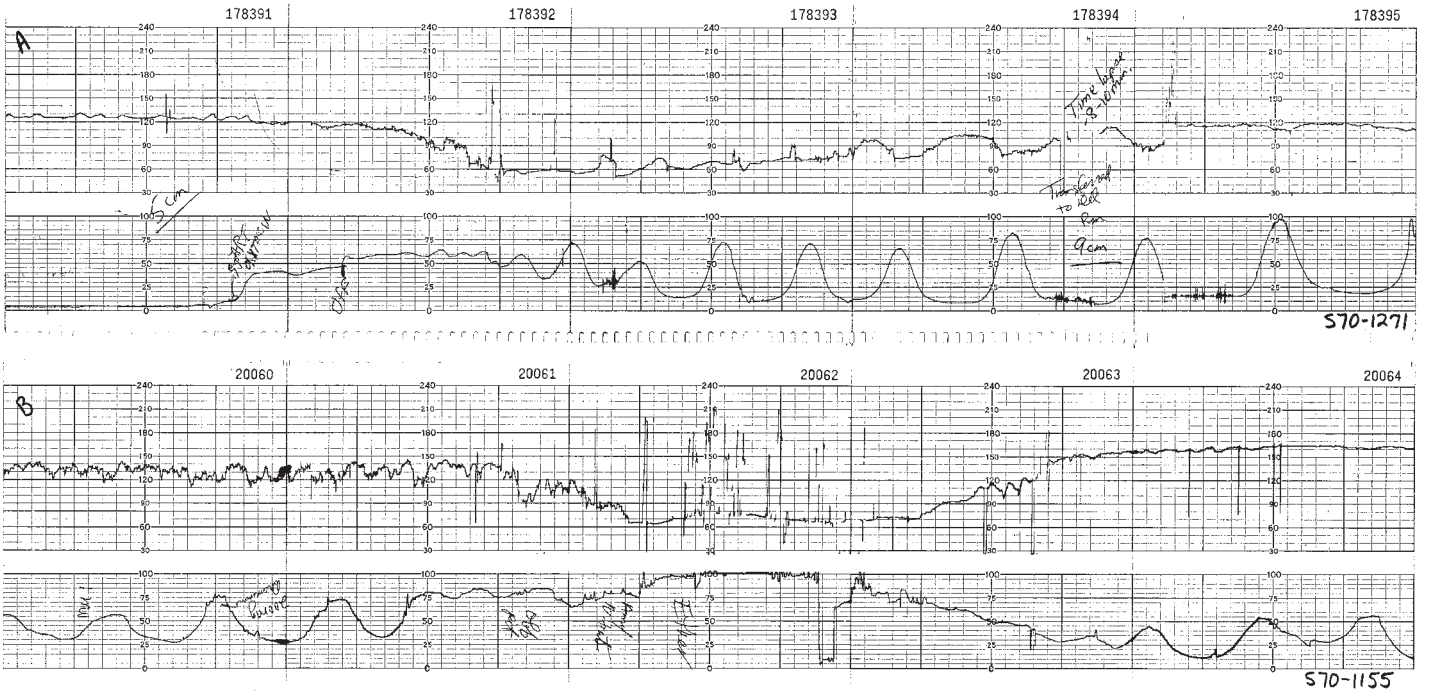

Figure 8.16. The **upper panel** demonstrates a tetanic contraction with severe prolonged fetal heart rate (FHR) deceleration after institution of oxytocin. The **lower panel** represents a tetanic contraction with a severe prolonged FHR deceleration after intravenous dimenhydrinate (Dramamine). (From Paul R, Freeman R: *Selected records of intrapartum fetal monitoring with self instruction*. USC Publishers, Los Angeles, 1971, with permission.)

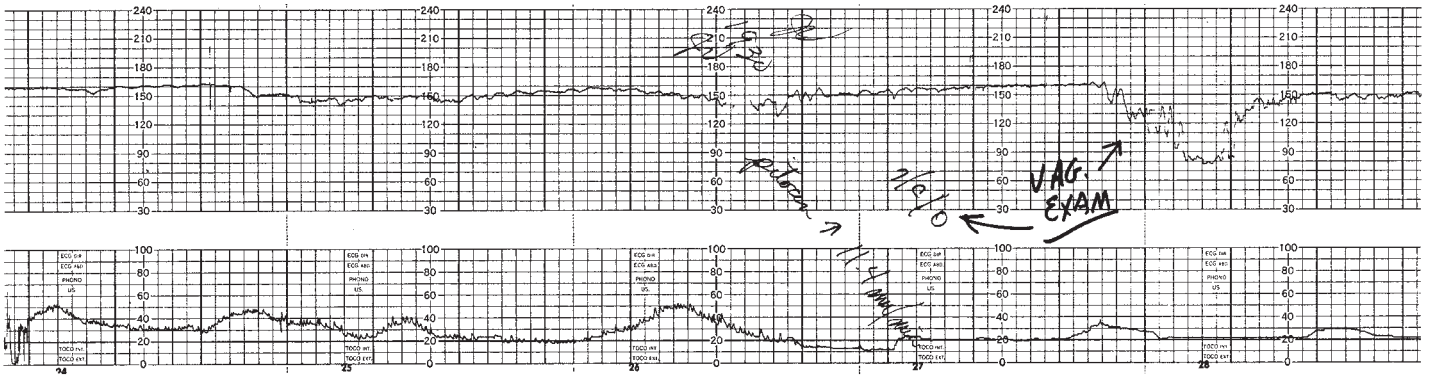

Figure 8.17. A prolonged deceleration associated with a vaginal examination.

6. Maternal seizure
7. Epidural or spinal block (Fig. 8.19)
8. Supine hypotension
9. Fetal CNS anomalies
10. Prolonged umbilical cord compression, often associated with rapid descent of the fetus
11. Maternal respiratory arrest (high spinal, intravenous [IV] narcotic) or cardiac decompensation
12. Uterine rupture (Fig. 8.20)
13. Abruptio placentae (Fig. 8.21)

When late or prolonged decelerations occur in patients soon after receiving a conduction anesthetic, initial management should be directed at correcting maternal hypotension by positioning the patient either in the lateral position or, by leaving her supine, instituting sustained left uterine displacement, and raising her legs in the air. These measures will give the patient an "autotransfusion" of several hundred milliliters of blood. In addition, the patient should receive a rapid infusion of IV fluids (preferably she would have been prehydrated before the block) and be started on oxygen inhalation.

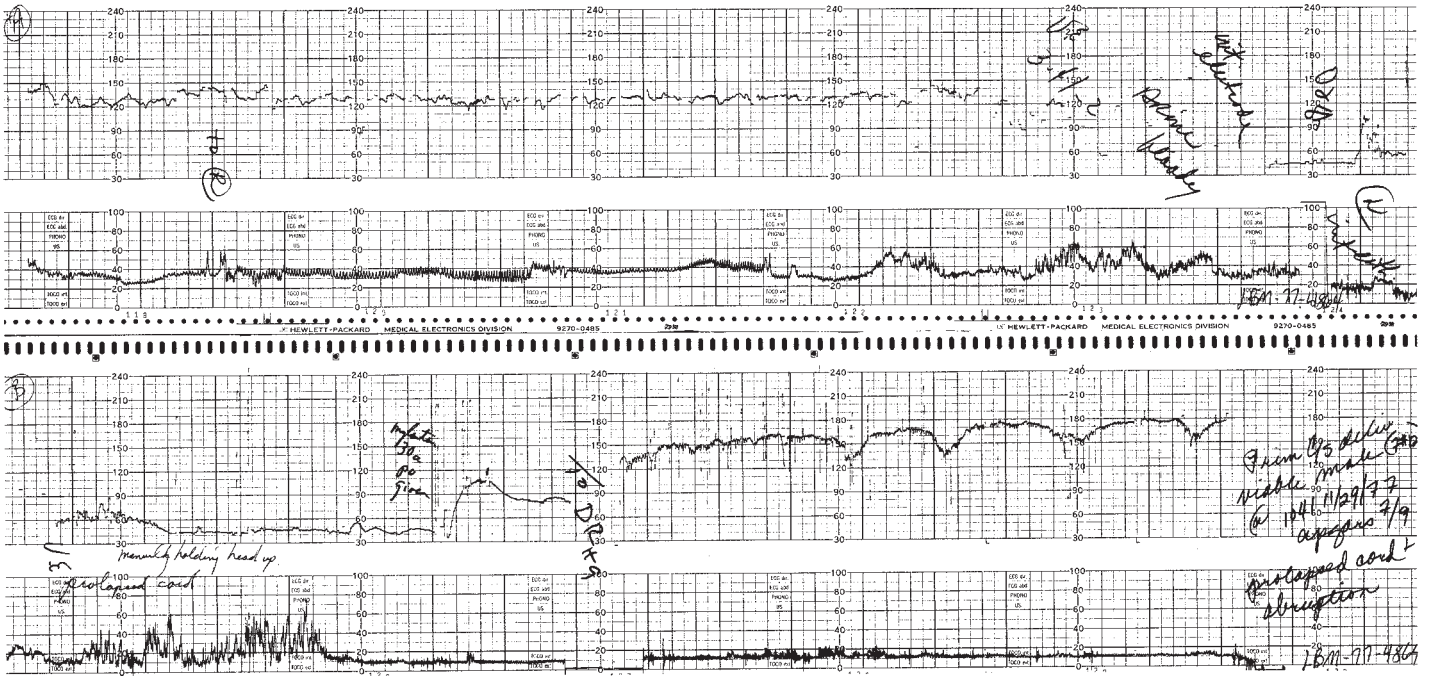

Figure 8.18. Toward the end of the **upper panel**, the membranes are ruptured and a fetal scalp electrode is applied because of severe vaginal bleeding. The fetal heart rate (FHR) is noted to be 40 beats per minute (BPM) following the previous 130-BPM rate from the previous Doppler recording. Reexamination at the beginning of the **lower panel** reveals a prolapsed umbilical cord. After elevating the presenting part, the FHR shows a marked tachycardia just before a cesarean section revealed an abruption. The Apgar scores were 7 and 9 at 1 and 5 minutes, respectively.

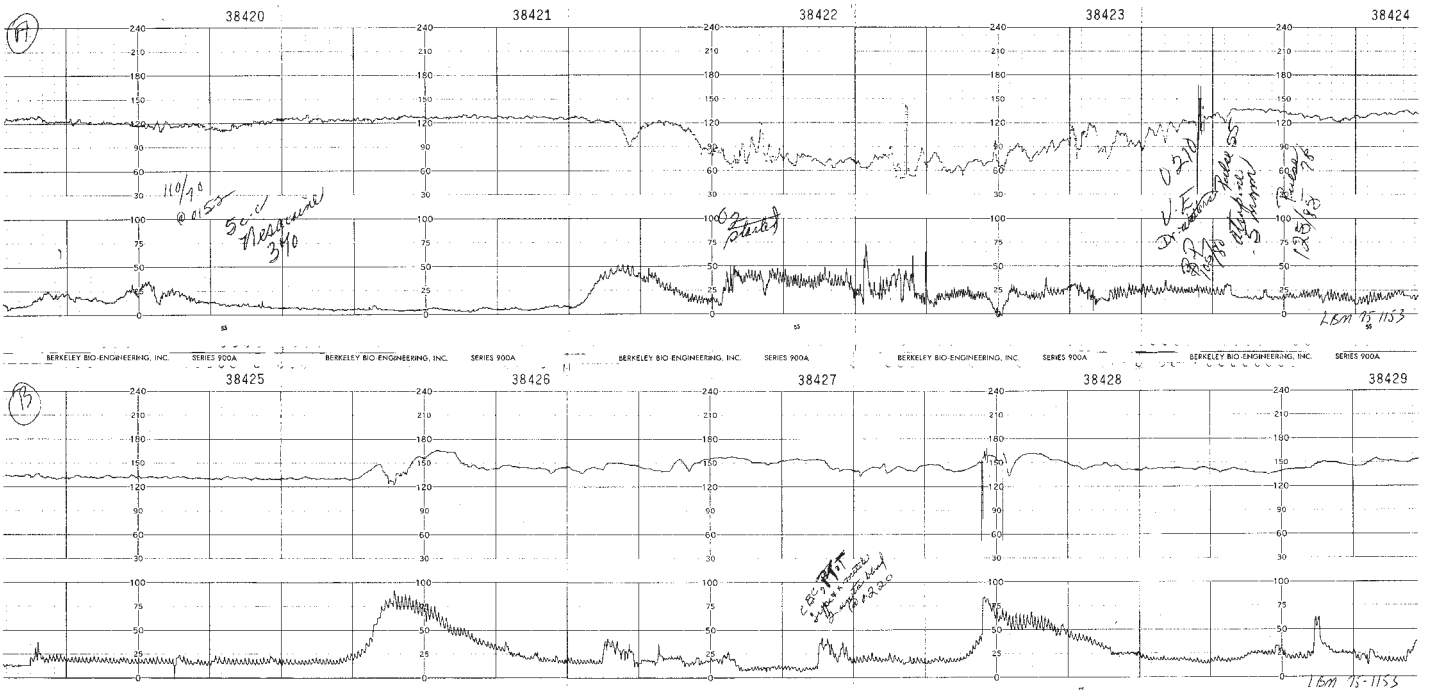

Figure 8.19. A prolonged fetal heart rate (FHR) deceleration after activation of an epidural block. During the deceleration, the mother's blood pressure was not low, but her pulse rate was only 55, so the anesthesiologist administered 0.5 mg of atropine intravenously. Note the subsequent increase in the FHR and the loss of short-term variability.

If necessary, IV ephedrine is administered in boluses (10). In addition, attention should be given to uterine tone because this may result from uterine ischemia. With tetanic contractions and prolonged FHR decelerations, consideration should be given to parenteral tocolysis with a beta-agonist such as terbutaline (Fig. 8.22).

The clinical response to prolonged decelerations in general should be as follows: First, consider your response. The few minutes that you do not overreact will frequently result in two favorable events: The deceleration will usually spontaneously recover, and you are less likely to unnecessarily frighten the patient. If the deceleration persists for more than a few minutes, a vaginal examination should be performed. This will reveal if either rapid descent and impending delivery or a prolapsed cord as the etiology. Obviously, these are the two most critical issues to identify immediately. The next step is to carefully review the clinical status of the patient and the fetal monitor for possible explanations. Commonly, a treatable etiology will be identified and the FHR will return to normal.

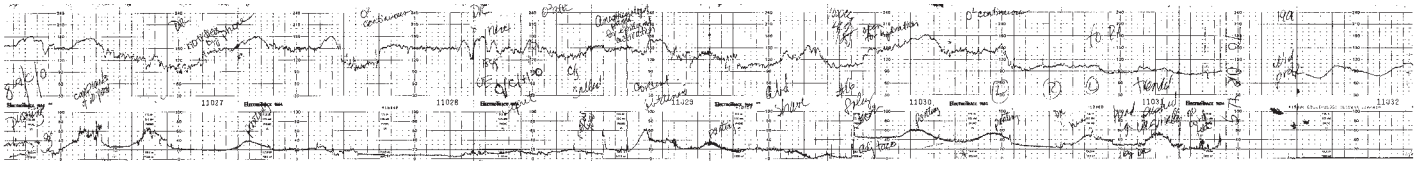

Figure 8.20. This is a gravida 2, para 1 with a previous low transverse cesarean section in spontaneous labor at term. Upon reaching 9-cm dilation, the fetus began having variable decelerations. Because of her complaint of the onset of severe lower abdominal pain in conjunction with these decelerations, the physician became concerned over the possibility of a uterine rupture and decided upon cesarean section. In the process, the variable decelerations progressed to an ominous prolonged deceleration. An urgent cesarean section was performed, and a large uterine rupture was found. The fetus had Apgar scores of 1 at 1 minute, 4 at 5 minutes, and 7 at 10 minutes, with an umbilical cord arterial pH of 6.90, pCO_2 90, BE −14 and venous pH of 7.10, pCO_2 50, BE −9. This mixed metabolic and respiratory acidosis is characteristic of uterine rupture. The baby ultimately survived without apparent neurologic damage.

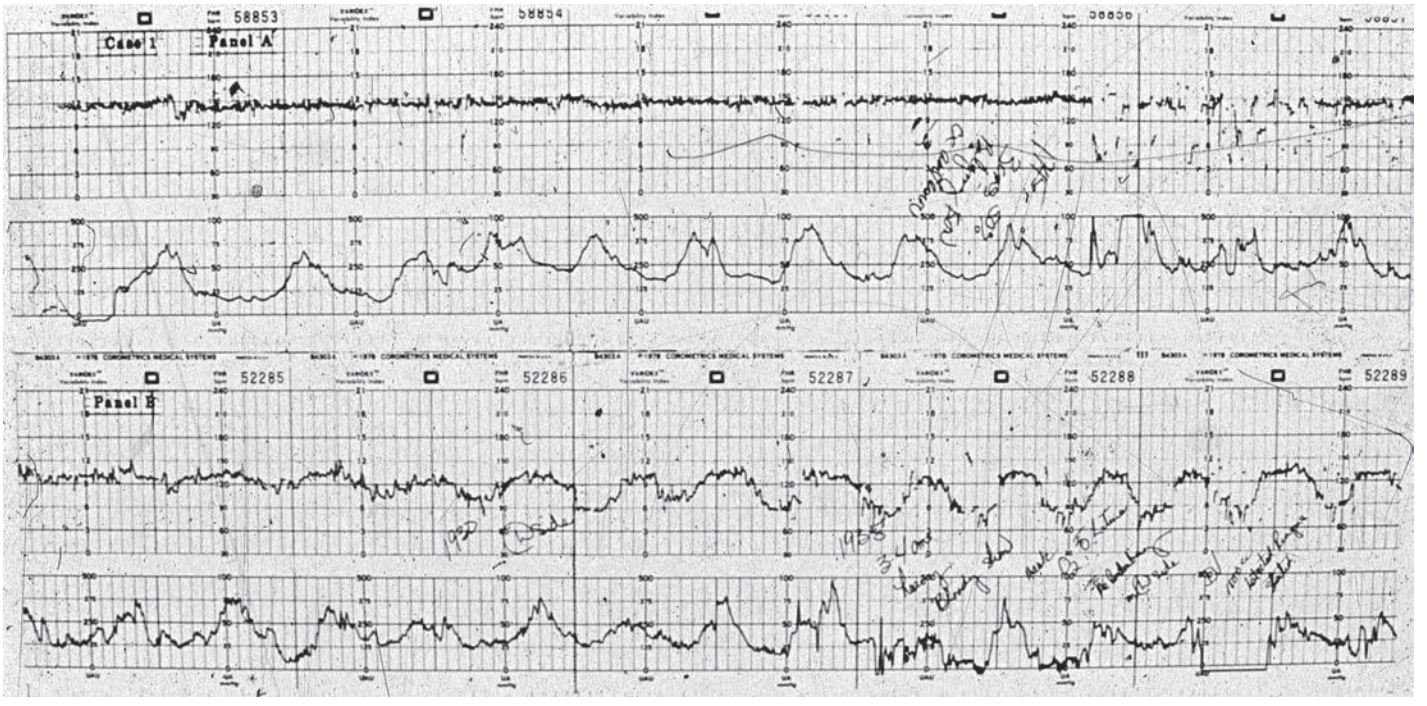

Figure 8.21. This patient presents in active labor at term. Other than being in an unusual amount of pain, she had no other historical factors that alert the clinician to a problem. However, on the monitor it is apparent that she is having frequent (tachysystolic) contractions. In the **lower panel**, late decelerations appear, and in the latter half of that panel, the patient begins having vaginal bleeding. A forceps delivery yielded a baby with Apgar scores of 4 at 1 minute and 7 at 5 minutes, and a large abruptio placentae was revealed when the placenta was delivered. Abruption may present either with prolonged decelerations or persistent later decelerations that may evolve into a prolonged deceleration, depending on the severity of the abruption and the degree of uterine hyperactivity.

However, evidence of bleeding and uterine tachysystole or tetany associated with prolonged deceleration may represent abruption or uterine rupture and require immediate delivery.

If a treatable cause is not identified and the deceleration persists, the next step, usually now after 5 to 7 minutes, is to move the patient to the operating room and mobilize the nursing and anesthesia team. Upon arrival in the operating room, the FHR monitor should be reconnected, preferably with a fetal scalp electrode in place to monitor the FHR directly. If the FHR remains low, immediate delivery is indicated. The rapidity with which this must be accomplished depends on a number of variables including what the FHR looked like before the onset of the deceleration; whether there is any suggestion of abruption or uterine rupture, which are the most rapidly progressing insults; the depth and duration of the deceleration; how much variability is lost during the deceleration; and whether the FHR appears to be returning to baseline periodically or just stays down. Often following recovery of the prolonged deceleration, if the insult is sufficient, a period of tachycardia and loss of variability and even late decelerations will appear (Fig. 8.18). If the insult is resolved, there is no need for immediate intervention. If the decision is made to proceed to delivery anyway, allowing for intrauterine resuscitation before abdominal or vaginal delivery will in many cases be in the best interest of both the mother and her fetus. How long this will take will vary with the etiology and duration of the insult and the status of the baby before the insult, but 20 to 40 minutes may elapse before the rate and variability return to normal.

The other common management dilemma with this pattern is with recurrent prolonged decelerations. These are usually due to prolonged cord compression. If they occur in the earlier part of labor and are associated with oligohydramnios, they may resolve with amnioinfusion. If not, they are one of the most difficult patterns to manage. Unlike other patterns, where the basic premise with a nonreassuring pattern is to rule out acidosis, the situation here is quite different. The concern is that the next deceleration will not return to baseline and the clinician will be faced with the urgency of the prolonged deceleration that does not remit. Thus, one must integrate how often they are occurring, how long they stay down below baseline, whether variability is lost, what the pattern looks like when the deceleration resolves, and how long before the patient is expected to deliver. In addition, consideration must be given to the individual facility's response time.

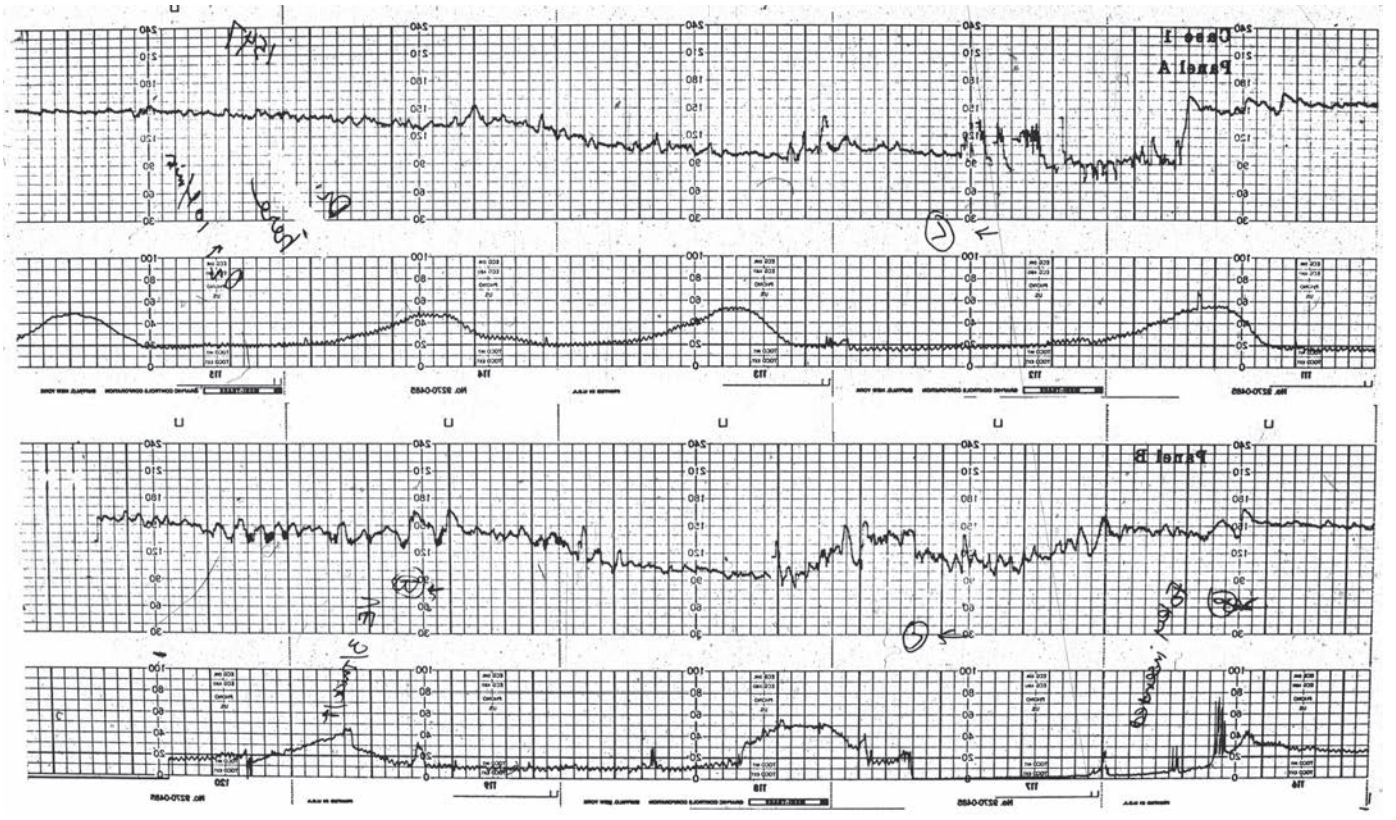

Figure 8.22. Recurrent prolonged decelerations are seen in this fetal heart rate (FHR) tracing in early labor. While variability remains normal and there are no signs that the fetus may be becoming acidotic, this remains a concerning pattern as there is a substantial likelihood that the FHR may again go down and not recover or recover as well.

INTERVENTIONS FOR ABNORMAL FETAL HEART RATE

The ideal intervention for fetal hypoxia is a cause-specific, noninvasive one that permanently reverses the problem. While not always possible, this should certainly be the goal. Obviously, the first step in achieving this goal is to recognize the cause of the abnormal FHR pattern. A thorough knowledge of the pathophysiology of FHR changes coupled with a careful clinical patient evaluation and a knowledge of common causes of specific FHR changes will maximize the opportunity for this goal to be achieved. In addition to cause-specific types of interventions, theoretically all cases of hypoxia should benefit by interventions that maximize oxygen delivery and placental function.

Nonsurgical Interventions

Oxygen Administration

One of the most common ways of attempting to maximize oxygen delivery to the fetus is to give supplemental oxygen to the mother. While diffusion across the membrane is driven by pO_2 as opposed to oxygen content, and while maternal pO_2 can be raised substantially with mask oxygen, it is controversial as to whether or not fetal pO_2 is clinically significantly increased by routine maternal oxygen administration. In a 2003 review for the Cochrane Review, Fawole and Hofmeyr (11) concluded that there was not adequate evidence showing that administration of oxygen to the laboring patient improved fetal oxygenation, and such administration could not be supported by the evidence at that time. Other studies from fetuses being monitored with pulse oximetry also do not substantiate a significant rise in fetal SpO_2 using a regular face mask, while there may be some effect with a tight-fitting nonrebreather mask (12). More recently, Haydon et al. found in normally oxygenated fetuses a clinically insignificant increase of fetal oxygen saturation of some 5% using a simple mask and 6.5% using a nonrebreather face mask. However, in fetuses with hypoxia, there was a clinically significant 20% increase in fetal oxygen saturation using a simple mask and a 37% increase with a nonrebreather mask (13).

Lateral Positioning

It is considered optimal for all patients to labor in the lateral recumbent position, at least from the standpoint of maximizing uterine perfusion. The reasons for this are in being inactive and recumbent, the body is required to deliver the least amount of blood flow to other muscles. More importantly, in

either lateral position, there is minimal to no compression by the uterus of the vena cava or aorta, thus maximizing venous return and cardiac output.

Hydration

Most patients in labor are either restricted or prohibited from taking oral fluids for fear of a requirement of an urgent operative delivery in the presence of a full stomach. If not fluid restricted, individuals involved in sustained exercise, and possibly by inference in active labor, do not voluntarily ingest adequate amounts of fluid and become relatively dehydrated due to this phenomena called "autodehydration." Recent evidence would suggest that the usual amount of IV fluid of 125 mL/hour is a gross underestimate of the replacement required in labor (14). Thus, by increasing fluid administration, there is potential to maximize intravascular volume and thus uterine perfusion. The type of fluid given intravenously should also be considered. With reports of increased rates of neonatal hypoglycemia, acidosis, jaundice, and tachypnea following IV bolusing of the mother with glucose-containing parenteral solutions, caution should be exercised with the use of glucose solutions. In a recent randomized study that demonstrated a shortening of the duration of labor in patients receiving IV glucose compared with normal saline, the use of glucose-containing solutions in labor was not associated with any deleterious effects in the newborn including bilirubin levels, blood glucose, or pH at birth (15). These solutions were administered as continuous infusion rather than bolus as in the prior concerning reports.

Oxytocin

In a patient with an abnormal FHR, the more time there is between contractions, the more time there is to maximally perfuse the placenta and deliver oxygen. In patients receiving oxytocin, there is a potential to improve oxygenation by decreasing or discontinuing oxytocin. As previously discussed, however, this may become a difficult situation as many patients will stop progressing in labor if the oxytocin augmentation is discontinued. Once the FHR pattern improves or resolves, it is often necessary to restart or increase the oxytocin. If the abnormal pattern returns, the clinician has a significant dilemma. At this point, it may be appropriate to continue the oxytocin despite the presence of FHR decelerations, especially if there are accelerations present, either spontaneous or elicited. Written documentation of the clinical plan explaining the necessity and appropriateness of continuing oxytocin in this situation is especially important.

Tocolytic Therapy for Abnormal Fetal Heart Rate Patterns

Although first reported by Caldeyro-Barcia et al. in 1969, the use of tocolytic therapy as an approach to treatment of acute fetal compromise has not been widely pursued (16). In the initial case reported, continuous IV infusion of a beta-mimetic agent (metaproterenol) was used, and sequential scalp pH samples were drawn. Once pH recovered to >7.30, the patient was delivered by cesarean section of a vigorous baby. Other drugs reported to be used successfully in relieving acute fetal distress include hexoprenaline (17), terbutaline (18–20), ritodrine (21), and magnesium sulfate (22). Improvement in both FHR pattern and fetal pH was seen in the majority of treated patients. In many of these cases, delivery was by cesarean section, although vaginal delivery following acute tocolytic therapy commonly occurs.

Although adverse fetal effects are not commonly reported following the efforts at pharmacologic *in utero* resuscitation, there are certain maternal conditions in which such agents should not be used. Because beta-sympathetic agonists will increase maternal pulse, stroke volume, systolic blood pressure, and blood glucose and decrease diastolic blood pressure and serum potassium, their use is contraindicated in women with cardiac disease, arrhythmias, hemorrhage, severe hypertension, or hyperthyroidism. FHR patterns of prolonged or persistent late decelerations associated with unresponsiveness to position change and cessation of oxytocin infusion may be treated with a subcutaneous or IV injection of 0.125 to 0.250 mg of terbutaline (Fig. 8.23).

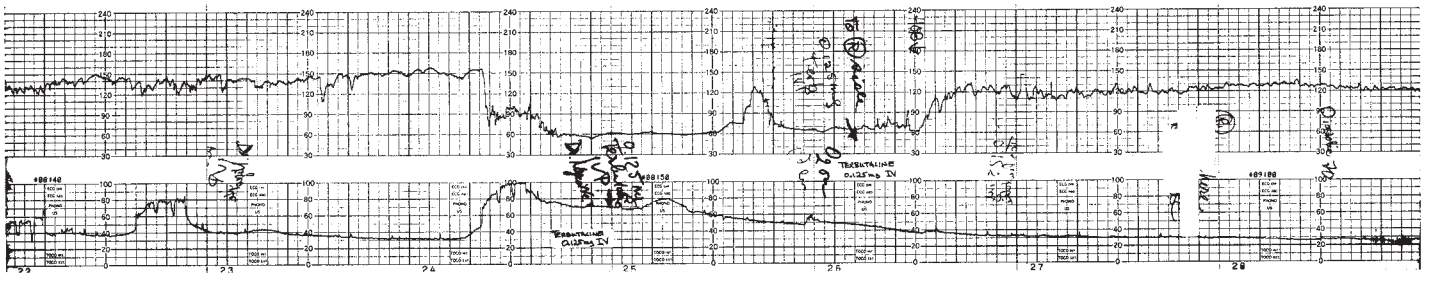

Figure 8.23. Tetanic contractions may result from excessive oxytocin administration or may occur spontaneously. In this example, the patient is in spontaneous labor at term. A prolonged deceleration is seen associated with a tetanic uterine contraction. Beta-agonist therapy is administered twice (terbutaline, 0.125 mg intravenously) with subsequent fetal heart rate return to baseline. The patient delivered a healthy baby 3 hours later.

In a setting of recurrent prolonged or severe variable decelerations where there is uterine tachysystole or an overall dysfunctional hypertonic uterine contraction pattern, tocolytic administration may result in decreased frequency and strength of contractions and resolution of the FHR pattern (Fig. 8.24).

Amnioinfusion

Variable decelerations are the most frequent and, in many ways, the most difficult FHR abnormality to manage. Variable decelerations indicate umbilical cord compression and may rapidly deteriorate. In addition, they are a source of patient, nurse, and physician distress that may lead to inappropriate intervention on behalf of an otherwise well-oxygenated fetus. During early labor, variable decelerations are most commonly seen in association with oligohydramnios either in a postterm or growth-retarded gestation or following rupture of membranes. In patients with preterm premature rupture of membranes, the risk for developing abnormal FHR patterns is increased with the most common concerning pattern being variable decelerations (23). Vintzileos has shown that the frequency and severity of variable decelerations vary directly with the severity of oligohydramnios (24) in the setting of premature rupture of membranes. Gabbe showed in fetal monkeys that removing amniotic fluid resulted in variable decelerations, and when the fluid was restored, the decelerations resolved (25).

Miyazaki and Taylor (26) first reported on the acute use of saline amnioinfusion in the treatment of variable or prolonged decelerations. Rapid infusion of normal saline through the intrauterine pressure catheter was effective in relieving the majority of repetitive variable or prolonged decelerations without apparent adverse maternal or fetal risk (Fig. 8.17). This was followed by a confirmatory prospective study from the same institution (27). Several subsequent randomized trials of therapeutic amnioinfusion have confirmed its benefits in reducing cesarean section for fetal distress and in improving Apgar

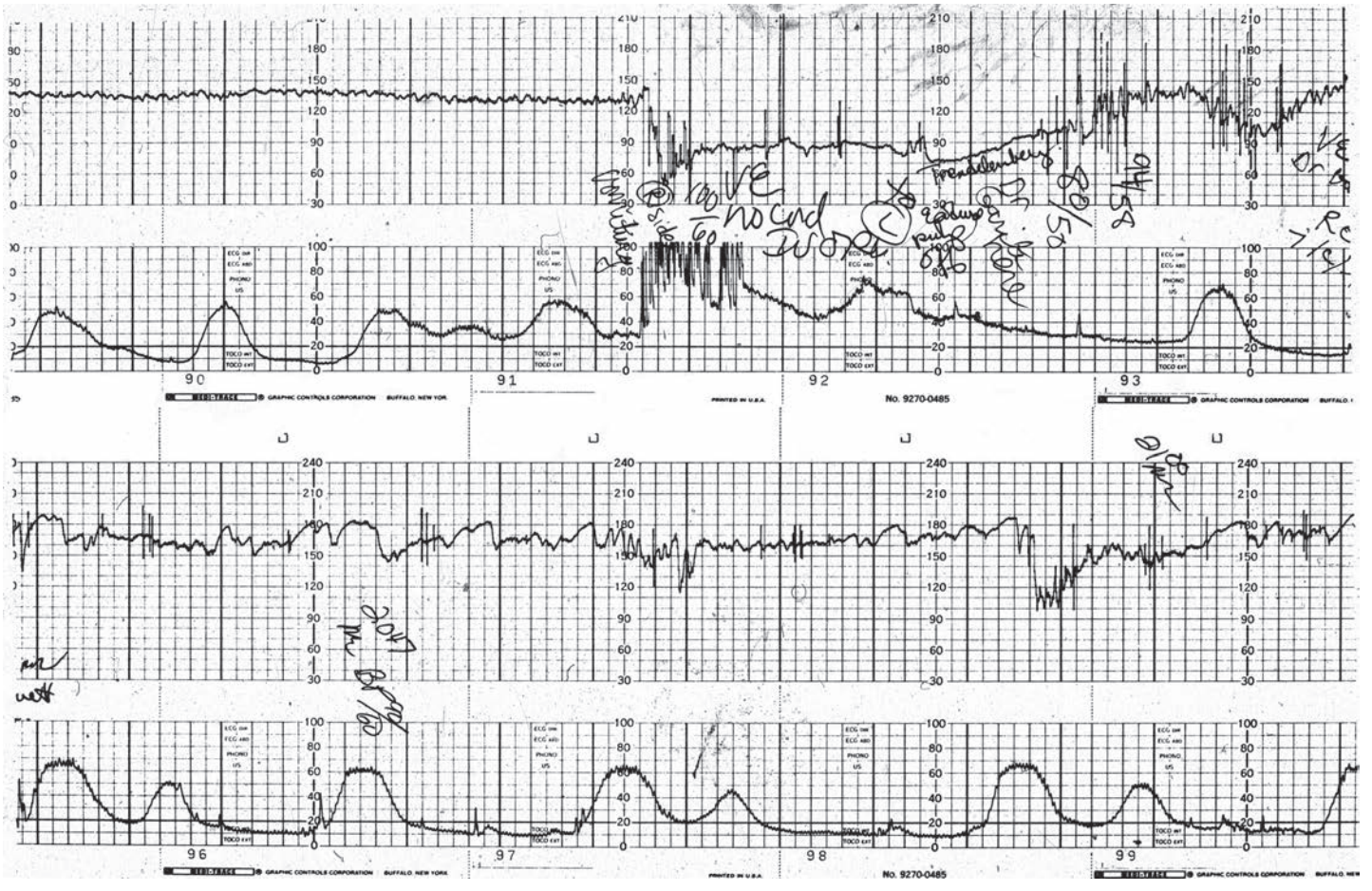

Figure 8.24. This gravida 2, para 1 with a previous cesarean section is at term in spontaneous labor. She is having recurrent prolonged decelerations as seen in the **upper panel** associated with spontaneous prolonged contractions (not on oxytocin). Terbutaline was administered allowing a more spaced out contraction pattern that subsequently resulted in a spontaneous vaginal delivery of a vigorous newborn.

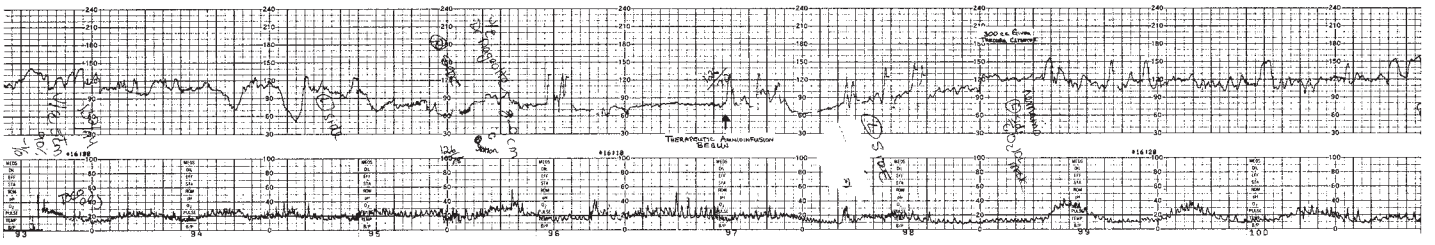

Figure 8.25. Prolonged decelerations in active labor may be unaccompanied by uterine hypertonus. Presumably due to continuous compression of the umbilical cord, various maneuvers are recommended: position change, cervical examination for cord prolapse, administration of oxygen, and rapid hydration. Acute amnioinfusion is another potentially therapeutic modality that can be used, particularly when cord entrapment is suspected. In this example, the fetus was having recurrent moderate-to-severe variable decelerations without evidence of uterine hypertonus.

scores and/or umbilical cord pH values (28–30). Thus, in patients in whom variable and/or prolonged decelerations appear in early labor, the use of amnioinfusion will often result in the improvement or resolution of such patterns (Fig. 8.25).

Nageotte et al. (28) prophylactically infused warm saline intrapartum in women in early labor with preterm premature rupture of membranes and documented oligohydramnios. In this prospective randomized study, preterm patients with premature rupture of membranes receiving prophylactic amnioinfusion experienced fewer decelerations in both the first and second stages of labor, had higher mean umbilical artery and vein pH, and had a clear trend toward a decreased cesarean section rate for abnormal FHR patterns when compared with control patients. Additionally, amnioinfusion may be started prophylactically when there is an unusually high risk for the development of variable decelerations from oligohydramnios, such as with fetal growth restriction. In a meta-analysis of randomized trials allocating patients who were experiencing variable decelerations in labor to either amnioinfusion or routine monitoring, those receiving amnioinfusion had a significant reduction in both the frequency of variable decelerations and cesarean delivery for abnormal FHR (31). Further, amnioinfusion is equally effective when administered in either bolus or continuous infusion (32).

OPERATIVE INTERVENTION FOR ABNORMAL FHR

When the fetus is determined to have a persistently abnormal FHR pattern and backup methods cannot provide reassurance that the fetus is not acidotic, operative intervention is indicated. Several questions arise when such a decision has been made. What is the best choice, operative vaginal delivery or cesarean section? How much time do we have to perform the delivery? What anesthetic should be used? What is the prognosis? Are there situations where the fetus is too compromised or otherwise not likely to benefit from this intervention?

Choosing operative vaginal delivery or cesarean section is not difficult if the patient is in early labor. For the patient near or at complete dilation, this becomes a greater challenge. Which route is more likely to create the more rapid delivery while at the same time result in the least complications for mother and baby? Will the attempt at operative vaginal delivery be successful? This decision will depend not only on the variables that predict success of operative vaginal delivery (station, clinical pelvimetry, size of the baby, skill of the clinician, etc.) but also on the severity of the FHR pattern and whether there is time to find out whether an operative vaginal delivery will succeed. The time for intervention also is a question of judgment. Except for the situation of a prolonged deceleration that will not recover, most other situations require judgment and integration of the entire clinical picture of mother and fetus. The question of how much time is available to perform operative intervention in the face of an abnormal FHR pattern is a complex one, which includes the unpredictability of the FHR pattern, and the medical-legal concerns that may arise in these difficult cases. The American College of Obstetricians and Gynecologists recommends that "all hospitals have the capability of performing a cesarean delivery within 30 minutes of the decision to operate" but that "not all indications for a cesarean delivery will require a 30 minute response time" (32). The examples they give of those situations that mandate an expeditious delivery include hemorrhage from placenta previa, abruptio placentae, prolapse of the umbilical cord, and ruptured uterus. In some situations (e.g., sustained prolonged deceleration to <70 BPM with loss of variability), 30 minutes may be too long to avoid damage; in others, this 30-minute dictum may be too restrictive and result in suboptimal anesthetic choices and compromised preoperative preparation. Thus, a judgment based on the severity of the FHR pattern and the overall clinical status of mother and baby must be integrated into this difficult decision.

ADDITIONAL ISSUES IN THE MANAGEMENT OF ABNORMAL FETAL HEART RATE PATTERNS

Meconium

The presence of meconium has historically been a confusing issue in evaluating the fetus in labor. The quandary arises from the fact that while a hypoxic insult eliciting a significant vagal response from the fetus may result in the passage of meconium from the fetal gut, passage of meconium can also occur in the absence of any significant or sustained hypoxia and be an entirely normal finding. The meconium is a potential toxin if the fetus aspirates this particulate matter. The thickness of the meconium is also a reflection of the amount of amniotic fluid. Thick meconium generally reflects some degree of oligohydramnios.

Amnioinfusion also has been proposed in several prospective randomized trials in an effort to avoid the fetal/neonatal pulmonary problems associated with meconium (33,34). However, a recent large prospective trial of amnioinfusion in term labors complicated with thick meconium staining of the amniotic fluid failed to reduce the risk of moderate or severe meconium aspiration syndrome, perinatal death, or other significant neonatal or maternal abnormalities (35). When meconium is present, with delivery of the fetal head, immediate deep suctioning of the nasopharynx and oropharynx using a bulb syringe and a DeLee suction trap is usually performed. Most obstetric units will call for a neonatal resuscitation team in the presence of meconium passage. Once delivery is completed, the vocal cords may be visualized with a laryngoscope and endotracheal intubation and tracheal suctioning performed. It should be emphasized that meconium suctioning and vocal cord inspection may affect the Apgar scores, which may be depressed in response to such efforts. While meconium aspiration syndrome has varied degrees of severity and may result in perinatal death, it appears that most cases of meconium aspiration syndrome are not related to aspiration of meconium but rather result from other pathologic *in utero* processes such as chronic hypoxia and infection (36,37). Since meconium in the lungs clearly alters pulmonary function, clearing of the newborn's airway of particulate meconium should be attempted if possible. In fact, however, such therapeutic interventions immediately following birth appear to have no effect on altering the course of severe meconium aspiration syndrome.

Preterminal Patterns

The dying fetus does not always have a specific FHR pattern. However, loss of variability is virtually always present if the mechanism of fetal death is cord compression or placental dysfunction. In addition, most fetuses will have a period of prolonged deceleration as a terminal event. Periodic changes may resemble variable deceleration, but the patterns appear rounded and blunted (Fig. 8.26). Tachycardia may or may not precede fetal death. Rarely, sinusoidal patterns are present in terminal fetuses. Although the baseline FHR in the terminal fetus is usually unstable and may be characterized by a blunted slow wandering, some terminal fetuses will have a fixed baseline heart rate that appears to have been drawn with a ruler.

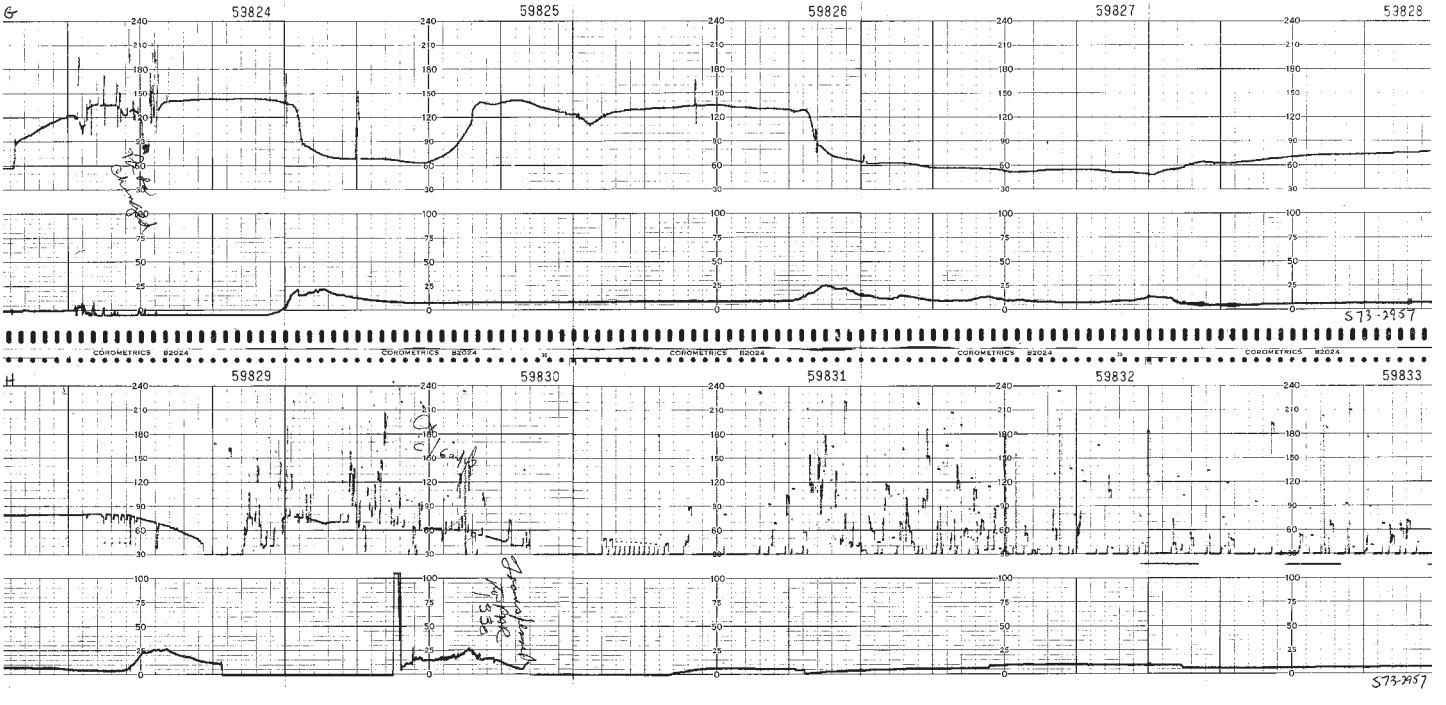

Figure 8.26. A terminal fetal heart rate pattern with "blunted" variable decelerations.

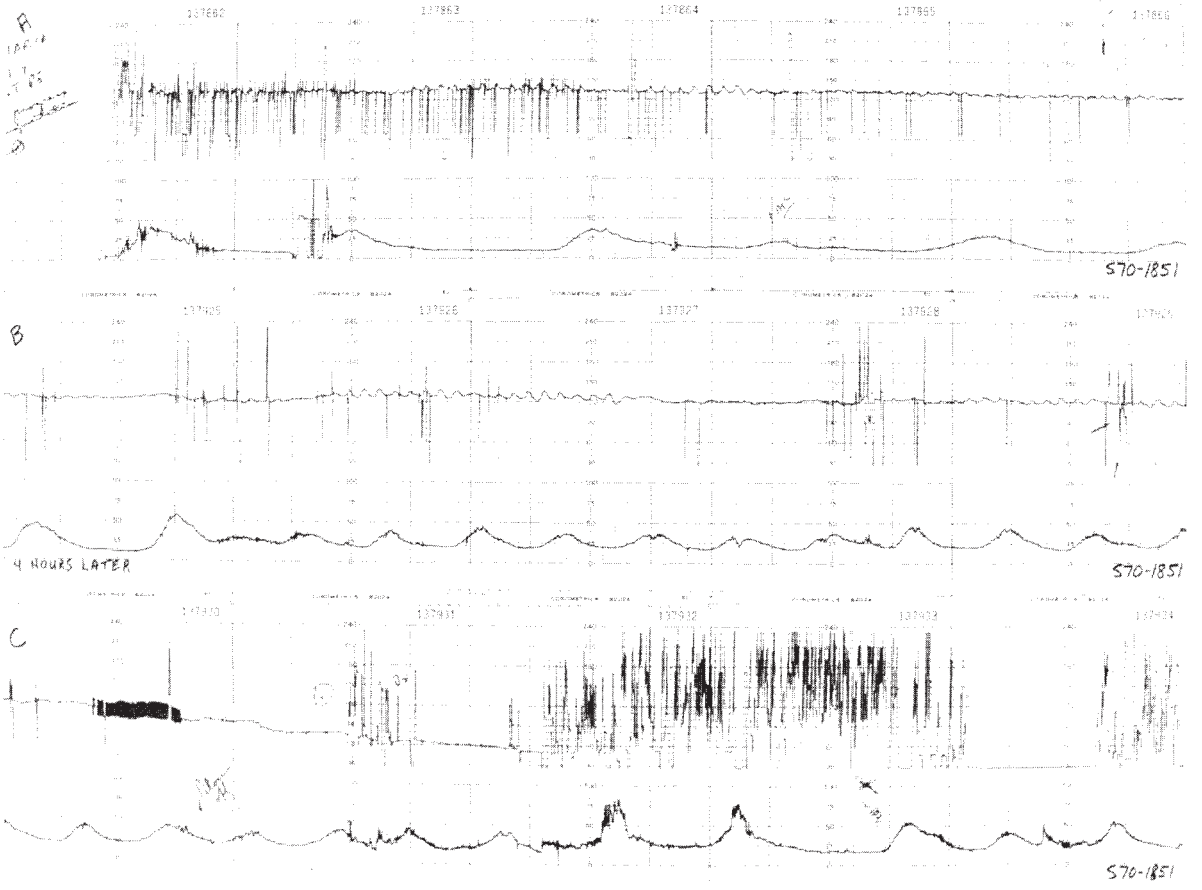

Figure 8.27. This tracing represents a terminal fetus. Note the absent short-term variability and intermittent sinusoidal pattern. Just before the final terminal bradycardia, there is an apparent arrhythmia.

Intermittent premature beats may be present, and a rapid beat-to-beat alternating atrial pacemaker has been reported in a severely hypoxic fetus just preceding death (Fig. 8.27). Importantly, one must be aware that these patterns may be seen in fetuses with major congenital anomalies and not necessarily be reflective of hypoxia or acidosis (Fig. 8.28)(38).

The implications for intervention become very difficult when dealing with one of these profoundly severe patterns because some interventions have been reported with salvage of apparently normal neonates. However, too often, the neonate is either severely damaged or does not survive. It is not possible using FHR patterns alone to be certain that prompt intervention will not result in a normal neonatal outcome. Thus, these patterns should be managed as other very abnormal patterns, and immediate intervention should be strongly considered.

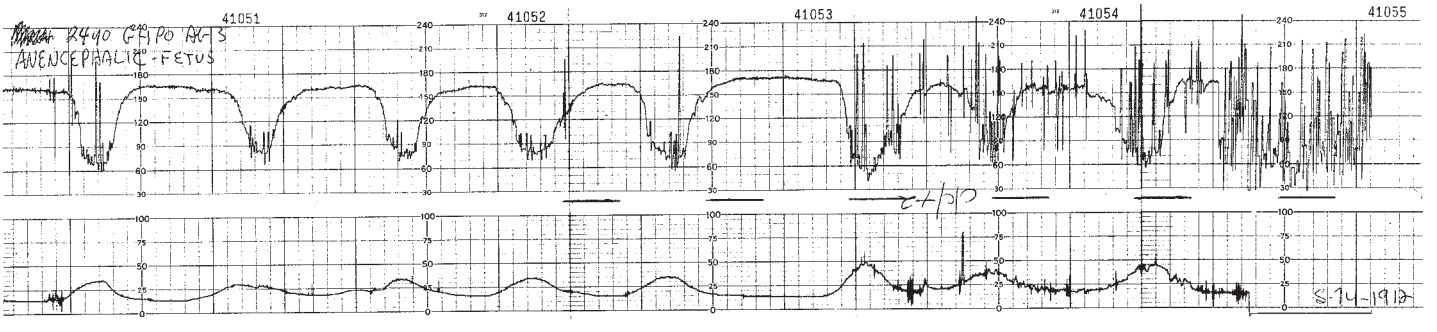

Figure 8.28. This pattern shows absent fetal heart rate variability, "blunted" variable deceleration, and fetal tachycardia. This was an anencephalic fetus with fetal death occurring at the end of the tracing, just before delivery.

Scalp and Acoustic Stimulation Tests

As has been mentioned often in this chapter, in both the antepartum and intrapartum periods, acceleration of the FHR (increase of FHR by 15 BPM lasting 15 seconds or more) is a reliable sign of fetal well-being. More specifically, accelerations in labor in the face of an abnormal FHR pattern have been shown to be a reliable sign of the absence of acidosis. Accelerations in response to fetal movement (spontaneous or following manual or auditory stimuli) provide virtually the same assurance. Clark et al. (39,40) originally described and subsequently prospectively demonstrated a strong positive correlation between acceleration of FHR in response to scalp stimulation and normal scalp pH (i.e., pH >7.19) (Fig. 8.29). In performing scalp stimulation, if acceleration of the FHR results, a normal fetal pH can be assumed. If no acceleration occurs, however, an abnormal pH is not necessarily present. Indeed, the majority of patients not responding to scalp stimulation still had a pH >7.20. Subsequent studies showed virtually identical predictive ability using a vibroacoustic stimulator placed on the maternal abdomen, similar to that described with antepartum testing (41). This simple test modality is a valuable tool for intrapartum evaluation of the fetus with an abnormal heart rate, and it markedly decreases the need for inappropriate delivery.

PHYSICIAN-NURSE COMMUNICATION AND TEAMWORK

Critical to the safe and successful delivery of obstetrical service to a woman and her fetus in labor is the close cooperation of the physicians and nurses responsible for providing her care. While most labor and delivery units operate very well with nurses managing the labors of women, it is vital that the physicians are appropriately informed of important information regarding their patient and updated with significant changes in either patient, the mother or the fetus. Interpretation of the FHR is primarily the responsibility of the nurse, but this is a shared responsibility, particularly when the tracing is not entirely normal. Using various electronic modalities, many physicians can now review FHR monitoring remotely using their computer or smart phone. At any time there is some concern regarding the monitor tracing, the nurse needs to communicate this concern to the physician and have the tracing reviewed either remotely or directly in the labor and delivery

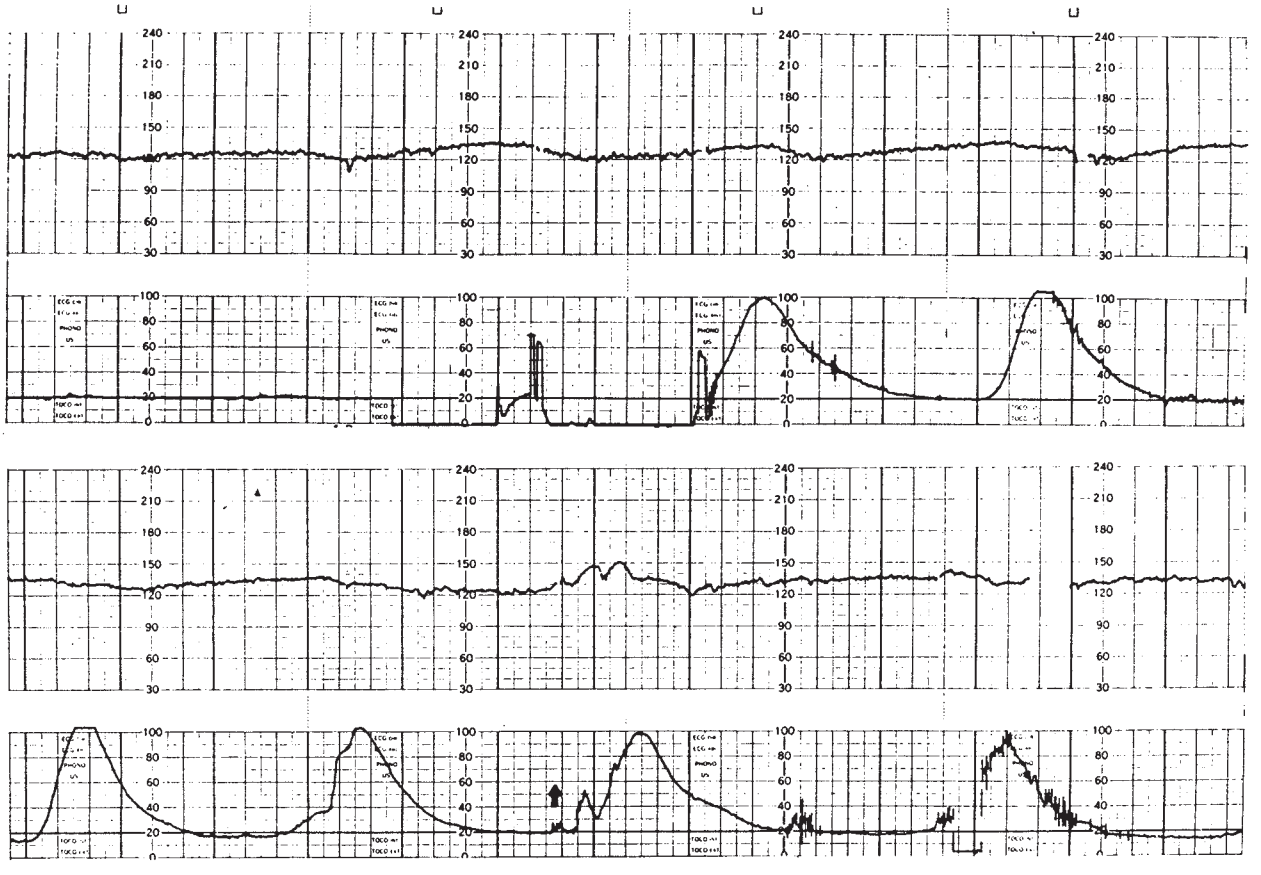

Figure 8.29. Decreased variability and late decelerations are present. Scalp stimulation (*arrow*) is accompanied by an acceleration of the fetal heart rate. Scalp pH is normal. (From Clark S, Gimovsky M, Miller F: The scalp stimulation test: a clinical alternative to fetal scalp blood sampling. *Am J Obstet Gynecol* 148:274, 1984, with permission.)

unit. If there is disagreement regarding the interpretation of the tracing, having someone else involved in the evaluation is recommended. If the nurse requests that the physician be in attendance in the hospital, such a request should be respected by either the physician or another designated responsible party coming to the bedside and evaluating the patient. When there is a responsible, privileged physician physically in attendance, it is the responsibility of that physician to dictate care decisions and to be ultimately responsible. If the physician is unresponsive to calls, appears to be incapable of clinical performance, or is clearly operating below the standard of care, the nurse and her supervisor have the responsibility to activate the chain of command to be certain that a responsible physician will assume the care of the patient. This is not easy to accomplish even in the most optimal of circumstances and often requires the recruitment of medical staff leadership in a potentially emergent situation. However, if the physician is in attendance, is the responsible physician, and does not appear to be compromised, generally speaking, there is no expectation to utilize the chain of command when there is some disagreement over a management decision or FHR interpretation between the physician and nurse. Such situations can be very stressful and challenging to all involved, and only with clear communication, professional deportment by everyone, and ongoing review of care processes can these clinical circumstances be sure to end safely and appropriately for the patient and her fetus.

Case 1

This case illustrates a nonreactive FHR leading to a suspected intrapartum diagnosis of a fetal CNS abnormality (Fig. 8.30). In the first panel, the patient is having her initial contraction stress test because of a gestation of 41½ weeks. Pregnancy was uncomplicated, and an ultrasound was reported as normal at 17 weeks' gestation. In Figure 8.30, the patient is having a breast stimulation contraction stress test that is nonreactive, but no late decelerations are seen. Because of the gestational age, the patient was admitted for induction of labor and repeat ultrasound. Figure 8.30B,C shows an intrapartum tracing with a persistent lack of reactivity and a possible wandering baseline. However, no late or variable decelerations are seen. Real-time ultrasound revealed massive hydrocephaly with a biparietal diameter of 13.5 cm. Because of the size of the fetal head, the patient underwent a primary cesarean section. She delivered a 4,950-g female with Apgar scores of 2 and 3 at 1 and 5 minutes, with normal umbilical artery and vein pH values. A computed tomography scan confirmed massive hydrocephaly, and the infant died 18 hours after delivery. This is an example of a nonreactive tracing not resulting from abnormal fetal oxygenation or pH but rather from a major CNS abnormality. The lack of late decelerations with a persistently nonreactive tracing raises the suspicion of previous neurologic injury, major CNS abnormality, or maternal use of depressive or narcotic drugs.

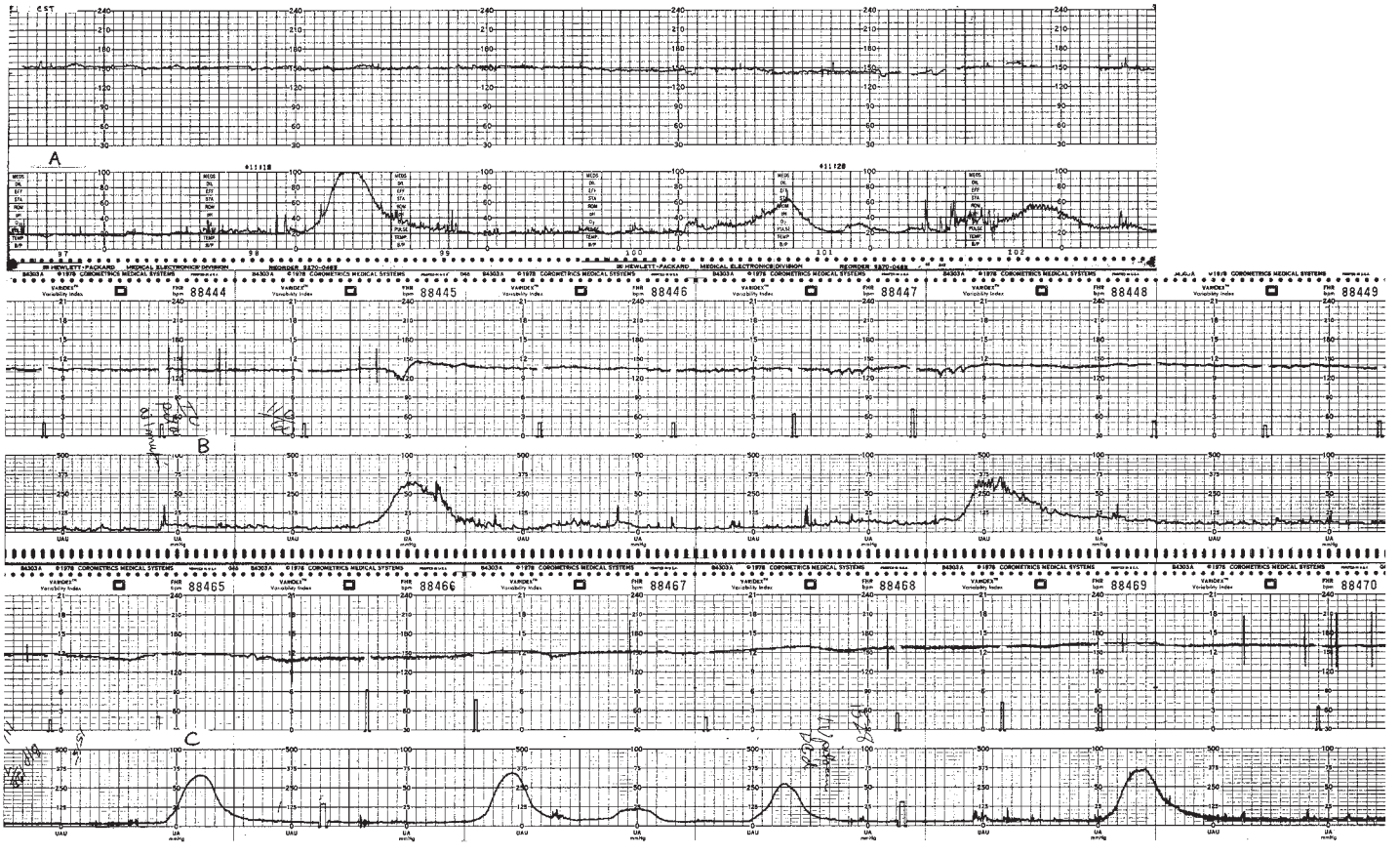

Figure 8.30. Case 1.

Intrapartum use of ultrasound is of value in identifying possible CNS abnormality or other fetal structural abnormalities.

Case 2

This case illustrates an unusual FHR pattern with a wandering baseline. The patient entered the hospital at 40 weeks' gestation for a labor check. Pregnancy had been apparently without complication. Figure 8.31A shows the FHR tracing during the time of evaluation for early labor. Note the smooth changes in baseline FHR without reactivity. These were interpreted as prolonged accelerations, and the patient was discharged home. Figure 8.31B shows the FHR 15 hours later when the patient returned in active labor. Persistent late decelerations were noted, and a cesarean section performed under general anesthesia resulted in the delivery of a 6-lb, 8-oz female. Apgar scores were 1 and 2 at 1 and 5 minutes, respectively. The infant began having seizures within 2 hours of delivery but had no respiratory distress. Problems with feeding and seizure control persisted for 2 weeks before the infant was discharged home. A computed tomography scan obtained before discharge revealed a 2-cm-diameter cystic mass in the right parietal area of the brain. At 1 year of age, the infant has achieved normal milestones with the exception of limited use of the left arm.

This most likely represents a case of a previous significant *in utero* asphyxial event. Although the initial evaluation was incorrect in interpreting the wandering baseline as accelerations, it is such an unusual pattern that it is easy to understand the misinterpretation. It was not until labor occurred that further hypoxia was present, as demonstrated by late decelerations. It is doubtful that earlier intervention would have had any significant impact on ultimate neurologic outcome, but it is difficult to establish that all the damage resulted from a preexisting condition.

Case 3

This case illustrates the FHR-uterine contraction pattern while a patient's uterus is rupturing. The patient entered into labor at 40 weeks' gestation with a known previous cesarean section. She was monitored with a scalp electrode and uterine pressure catheter. She was fully effaced, 6 cm dilated, and at 0 station when monitoring began. In Figure 8.32A, there is a mild tachycardia of 160 BPM with good variability and mild variable decelerations, and at the end of Figure 8.32A, there is a fetal tachycardia of 180 BPM. Figure 8.32B shows further progression of the late component mixed with moderate variable deceleration. A decision to perform a cesarean section was made in the last panel of Figure 8.32B, and the uterine catheter was removed. The severe decelerations can be seen to merge during the last half of Figure 8.32B, and the baseline FHR continued to increase. A cesarean section was performed soon after the end of this tracing, and a ruptured lower segment transverse incision was encountered. The fetus weighed 3,840 g and was moderately depressed but did well. The uterus was repaired.

This case indicates that there was no evidence of a loss of intrauterine pressure, even though the rupture likely occurred during monitoring. Severe variable and/or prolonged decelerations appear to be the most sensitive warning sign of such catastrophes. We encourage the use of internal uterine pressure catheters and fetal scalp electrodes in patients undergoing a trial of labor after a cesarean delivery.

Case 4

This case illustrates the effect of decreasing maternal blood pressure on the FHR and fetal pH (Fig. 8.33). The patient was a severe preeclamptic with blood pressure of 172/120

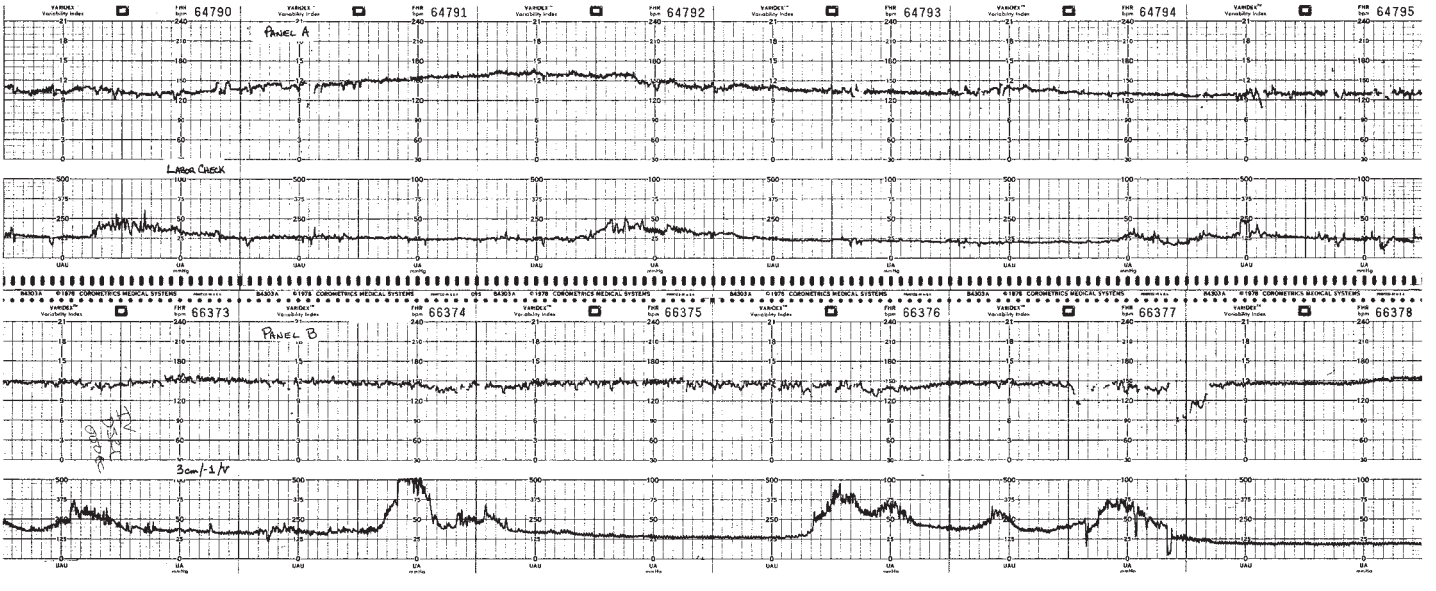

Figure 8.31. Case 2.

mm Hg, receiving magnesium sulfate. The tracing in the upper panel shows a normal FHR-uterine contraction tracing with good variability and a baseline rate of approximately 135 BPM. There are no periodic decelerations. The middle panel shows the FHR tracing after the maternal blood pressure decreased to 140/104 mm Hg following the parenteral administration of an antihypertensive medication. Note the decrease in variability, the increase in baseline FHR to approximately 160 BPM, and the presence of late decelerations. At the beginning of the bottom panel, supplemental oxygen is begun, the baseline FHR remains elevated, and late decelerations persist. At the end of the bottom tracing, a fetal scalp pH was 7.20. The FHR pattern persisted, and the patient was delivered by cesarean section of a baby with normal Apgar scores.

This case illustrates that even though blood pressure lowering may be indicated for maternal reasons, one must be careful to monitor the fetal response. Even though the maternal pressure was still increased after treatment, the decrease from 172/120 to 140/104 mm Hg represented relative hypotension from the fetal standpoint.

Case 5

This case illustrates the evolution of FHR changes to fetal death (Fig. 8.34). The patient was at 34 weeks' gestation with amnionitis and meconium staining. This is a case from many years ago but illustrates the natural course when no intervention occurred because of a perceived maternal risk of cesarean section in the face of an infected uterus and a fetus of only 34 weeks' gestation.

Figure 8.34A shows fetal tachycardia, possibly due to the maternal fever, with late decelerations beginning in the lower panel. The variability is moderately decreased by the end of the lower panel. Figure 8.34B shows persistence of late deceleration with progressive loss of variability and progressively shorter latent periods between the onset of the contractions and the onset of the late decelerations. Figure 8.34C

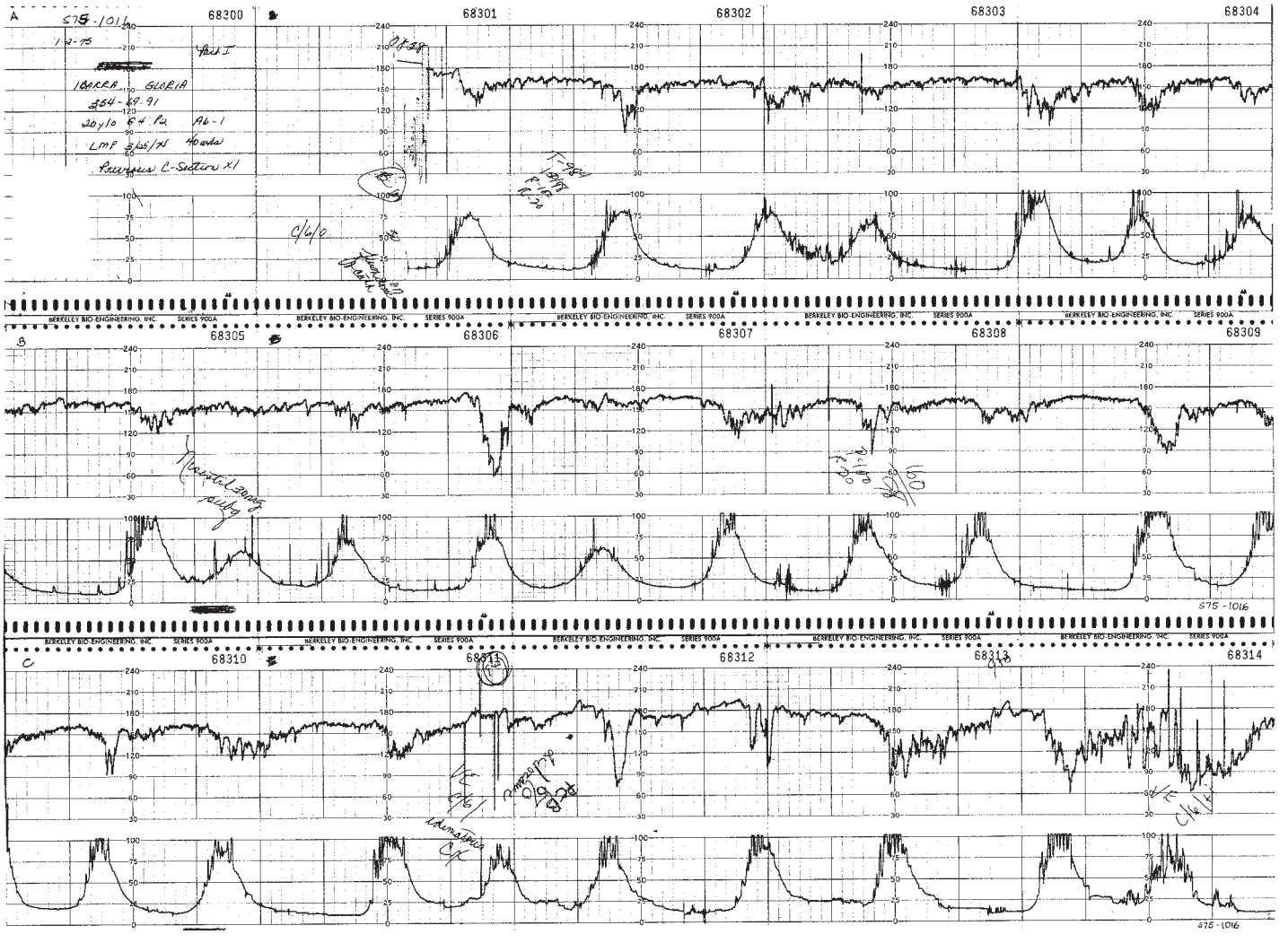

Figure 8.32. **A, B:** Case 3.

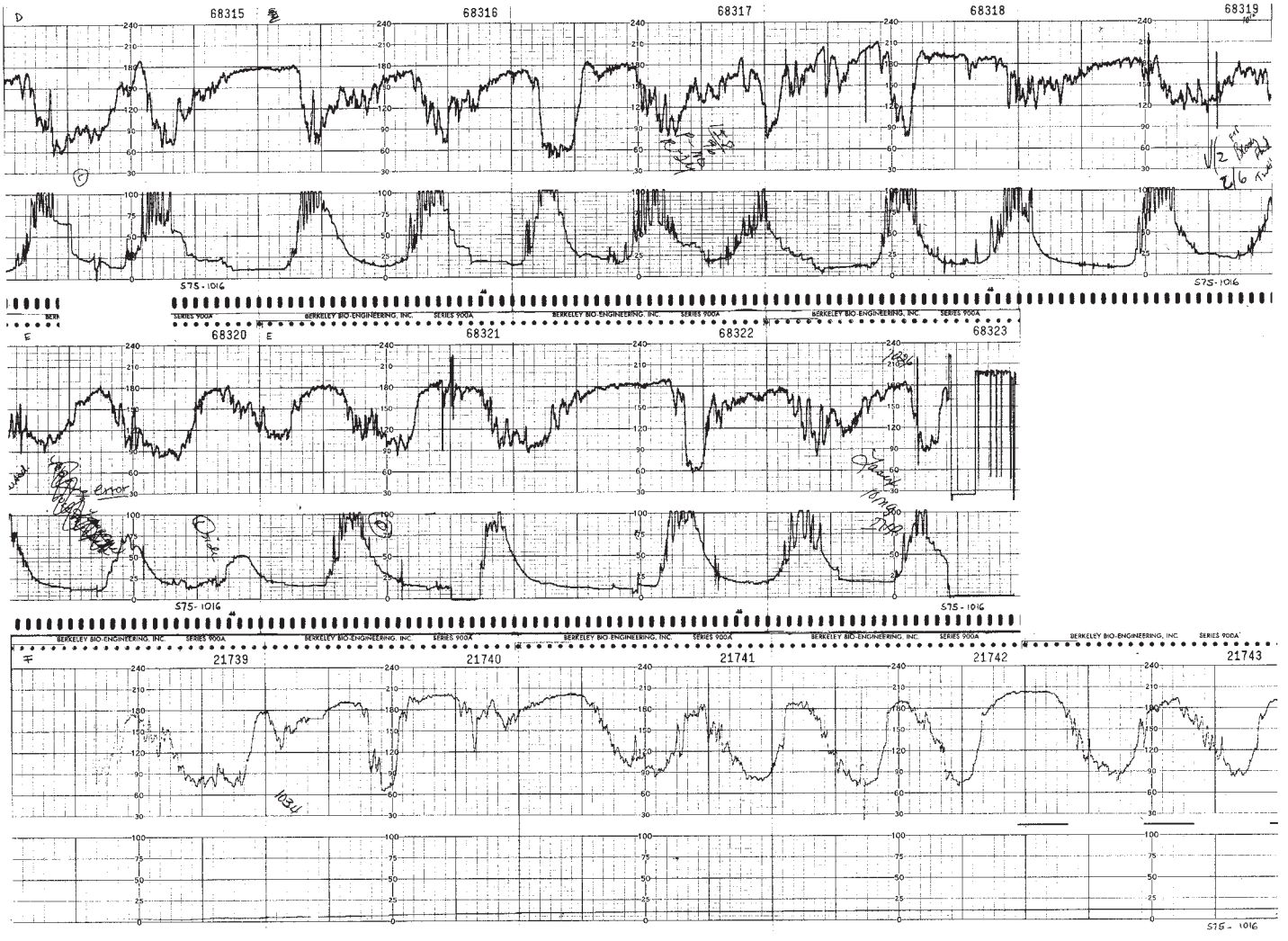

Figure 8.32. ***(continued)***

shows continuation of late decelerations, tachycardia, and no variability.

At the end of the middle panel, rather deep decelerations are seen that progress to a disorganized pattern in the lower panel and fetal death, which occurs at the end of the lower panel. This tracing represents 3 hours of monitoring, and it would be reasonable to expect that fetal salvage could have occurred with earlier intervention.

Case 6

This case illustrates the value of FHR monitoring during the initial evaluation of severe preeclampsia (Fig. 8.35). It also shows the difficulty sometimes encountered in detecting uterine activity. The patient was a severe preeclamptic admitted at 28 weeks' gestation with increased liver enzyme levels and thrombocytopenia. It was decided to effect delivery for maternal indications. Oxytocin was started, and the fetus was monitored externally. The upper panel of Figure 8.35 reveals no evidence of uterine activity despite several attempts to adjust the tocodynamometer. The FHR is smooth, and there are several decelerations characteristic in shape for late deceleration. The patient was moderately obese, and the uterus measured only 22 cm, but good dates with sonographic confirmation revealed a 28 weeks' gestation by biparietal diameter with probable fetal growth restriction and oligohydramnios. The FHR pattern was thought to represent late decelerations with decreased or absent variability, and a cesarean section was performed that resulted in a moderately depressed 740-g growth-retarded neonate who did well in the nursery. Even though contractions cannot always be recorded, the FHR changes should be observed closely, and if the clinical picture is that of a pattern of persistent late decelerations, the clinician should be willing to interpret such changes as abnormal.

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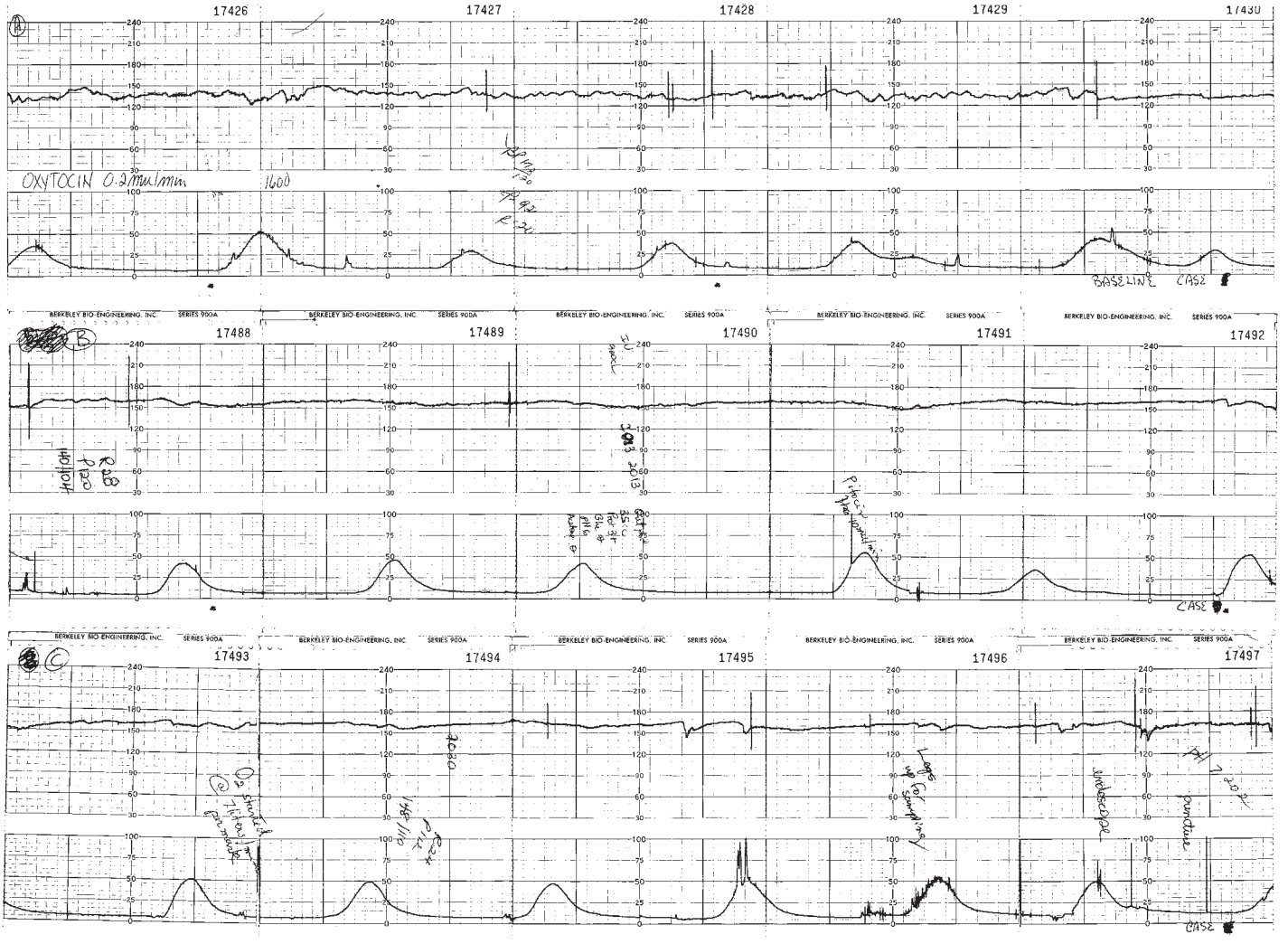

Figure 8.33. Case 4.

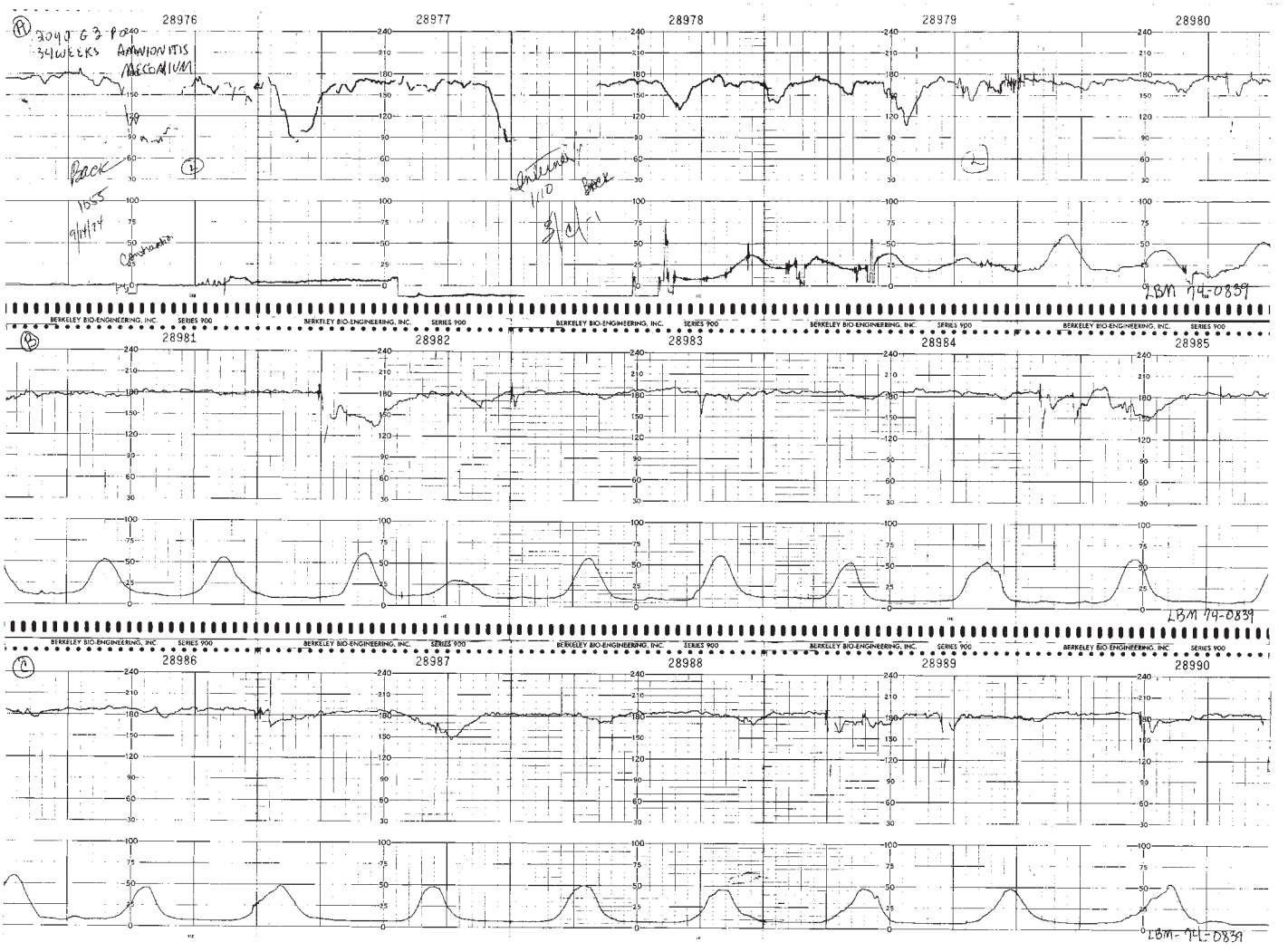

Figure 8.34. **A–C:** Case 5. *(Continued on next page)*

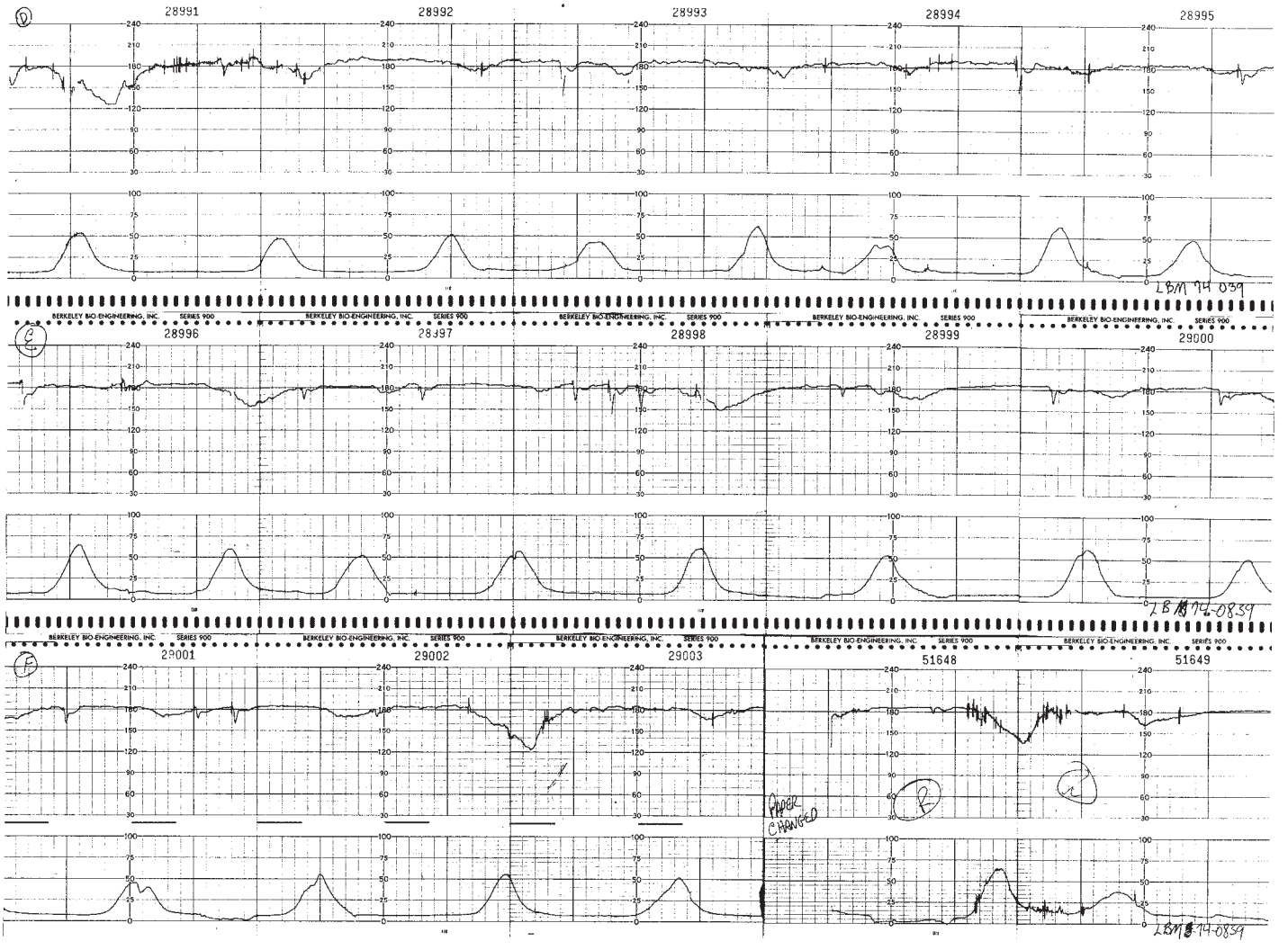

Figure 8.34. *(continued)*

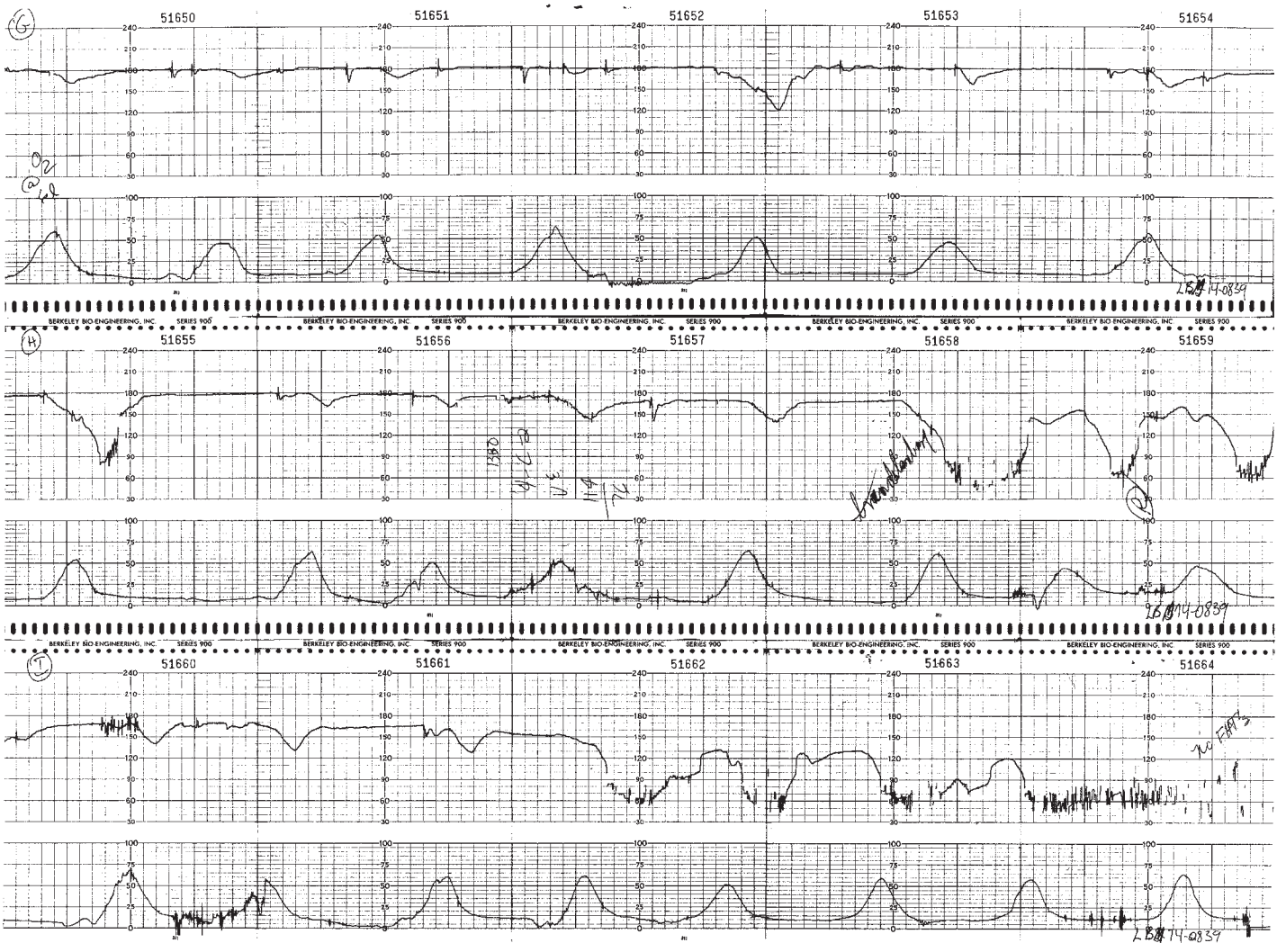

Figure 8.34. *(continued)*

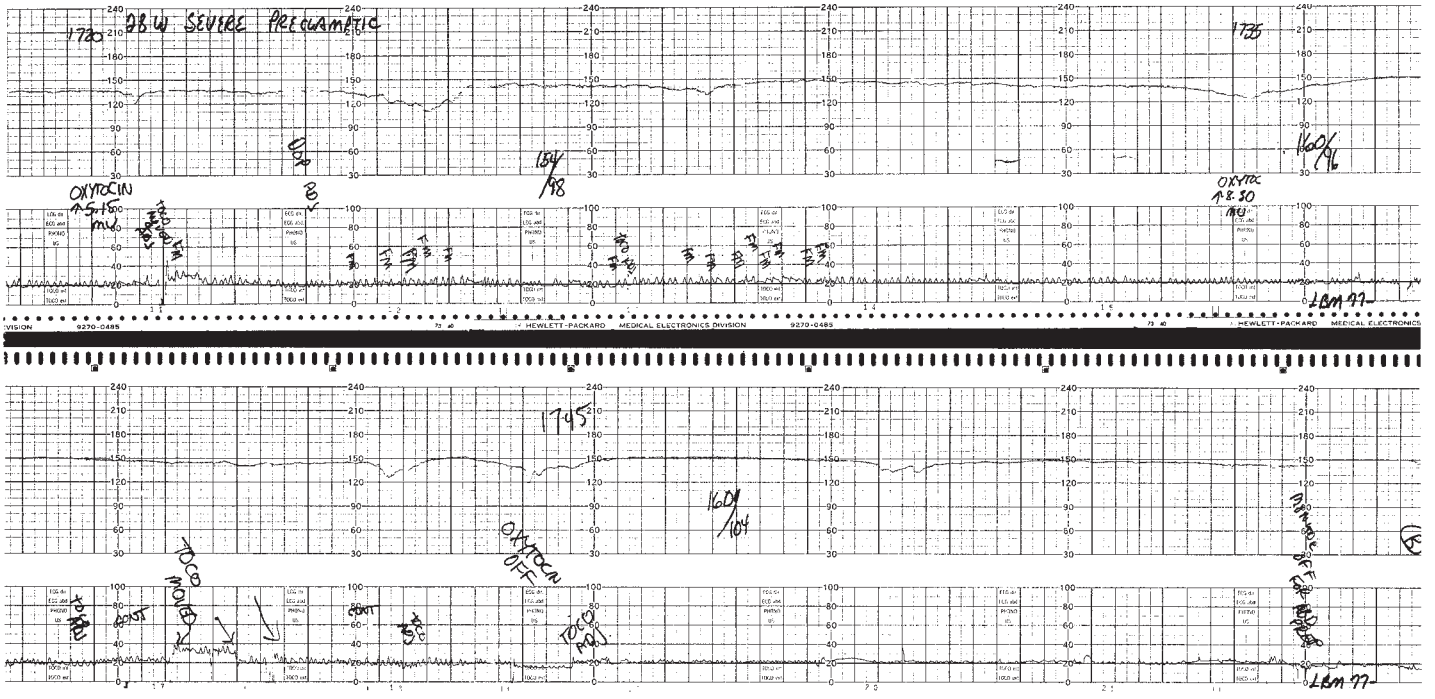

Figure 8.35. Case 6.

Case 7

This case represents an example of complete loss of variability without periodic changes (Fig. 8.36). The neonate was severely depressed but had a normal umbilical cord pH. Complete absence of variability without periodic changes may represent an already damaged fetus who is not currently hypoxic or who may have suffered brain damage from a previous insult. It also may represent a CNS malformation. This is one of the most difficult patterns to manage. An ultrasound is warranted to rule out major CNS anomalies, and a drug screen to rule out the use of depressant drugs. In the absence of another explanation, there are several alternatives. In early labor, a biophysical profile may be helpful. Fetal sleep cycles should also be considered but should not last beyond 60 minutes. Maternal repositioning, hydration, and use of supplemental oxygen should be employed as well as fetal acoustic or scalp stimulation, if possible. While uterine contractions unaccompanied by late decelerations would indicate the absence of hypoxia, without a specific identifiable cause for such a pattern requires very close monitoring and appropriate intervention.

Case 8

This case illustrates lack of variability and evolution of atypical variable decelerations during labor in a patient with marked metabolic alteration. The patient was a 27-year-old gravida 4 para 1 insulin-dependent diabetic (class D) with ketoacidosis and pregnancy-induced hypertension at 33 weeks' gestation. The patient was comatose with an arterial pH of 7.22 and a blood glucose concentration of 474 mg%. She had not taken insulin for 3 days because of persistent vomiting. As seen in Figure 8.37A, FHR is nonreactive but without late decelerations. Figure 8.37B (45 to 50) shows the appearance of blunted variable decelerations with overshoot. Her cervix was 6 cm dilated, and insulin was being infused continuously. Figure 8.37C shows deeper atypical variable decelerations with cervical dilatation of 8 to 9 cm. Figure 8.37D (84 to 89) is in the delivery room, where the patient spontaneously delivered a 3,010-g male with Apgar scores of 0 at 1 minute, 0 at 5 minutes, and 2 at 10 minutes. Cord umbilical vein pH was 7.03, and umbilical artery pH was 6.96. The infant died in the nursery at 1 hour of age.

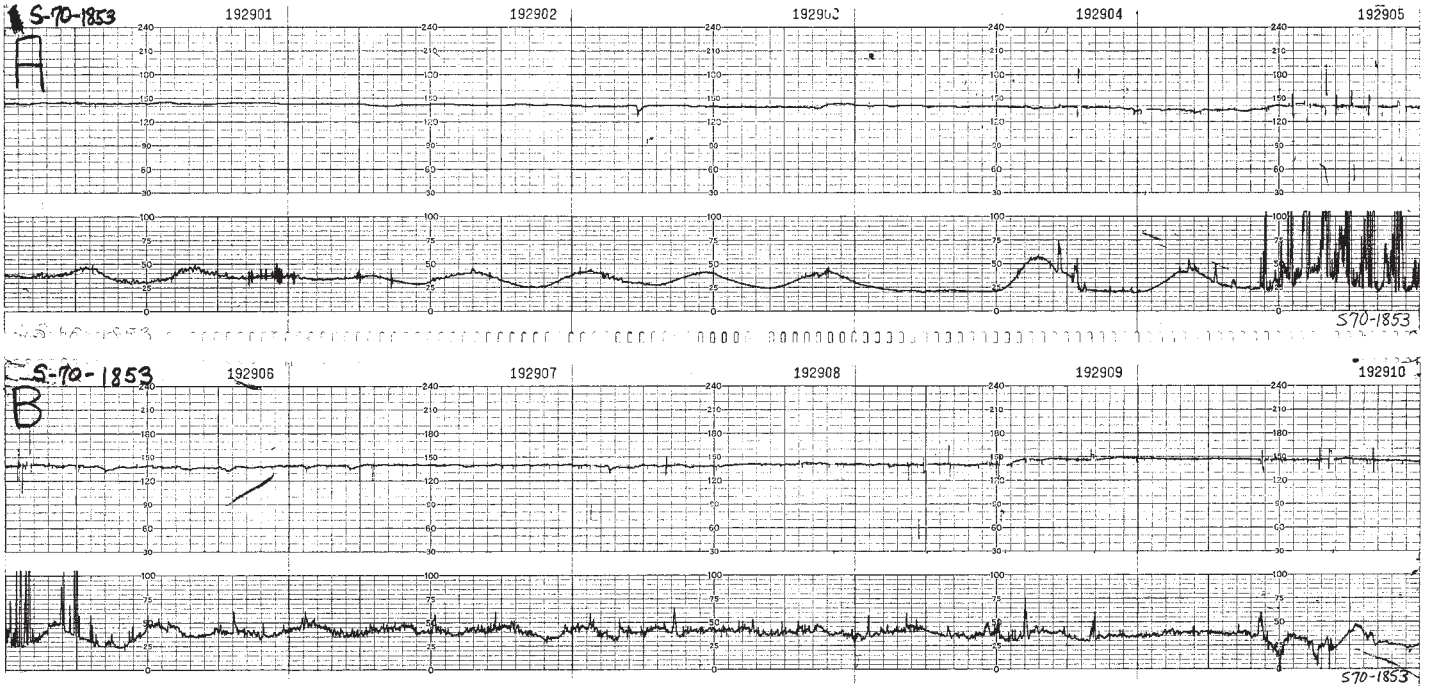

Figure 8.36. Case 7.

This is an example of preterminal FHR evolving in a very ill laboring woman without the option of cesarean section delivery because of the mother's compromised state. It is unclear what condition this infant was in on admission of the mother but at no point was there a normal FHR pattern.

Case 9

This case demonstrates markedly increased variability of a highly atypical nature. The patient was a 22-year-old gravida 1 at 41 weeks' gestation admitted in early labor (Fig. 8.38A). Note the "sawtooth" pattern. This should be distinguished from the more common pattern of marked increased variability, often referred to as a "saltatory" pattern, which usually is not associated with significant hypoxia or acidosis. This continues intermittently over the next 2 hours (Fig. 8.38B,C) and then becomes continuous (Fig. 8. 38D). Therefore, a primary cesarean section was performed with delivery of a depressed newborn with Apgar scores of 2 and 3 at 1 and 5 minutes. Umbilical cord pH was 6.92 (umbilical artery) and 6.96 (umbilical vein). The infant was hypotonic for 24 hours but showed no other signs of complications due to asphyxia. After 6 days, he was discharged home and was reportedly normal at 2 years of age.

Case 10

There is sudden decompensation of FHR following spontaneous rupture of membranes (Fig. 8.39). Only 12 minutes passed between decision and delivery, yet a stillbirth resulted. Examination of the placenta and membranes confirmed a velamentous umbilical cord insertion and ruptured vasa previa. This is a classic example of a fortunately very rare vasa previa with fetal exsanguination secondary to inadvertent laceration of an umbilical vessel within the fetal membranes.

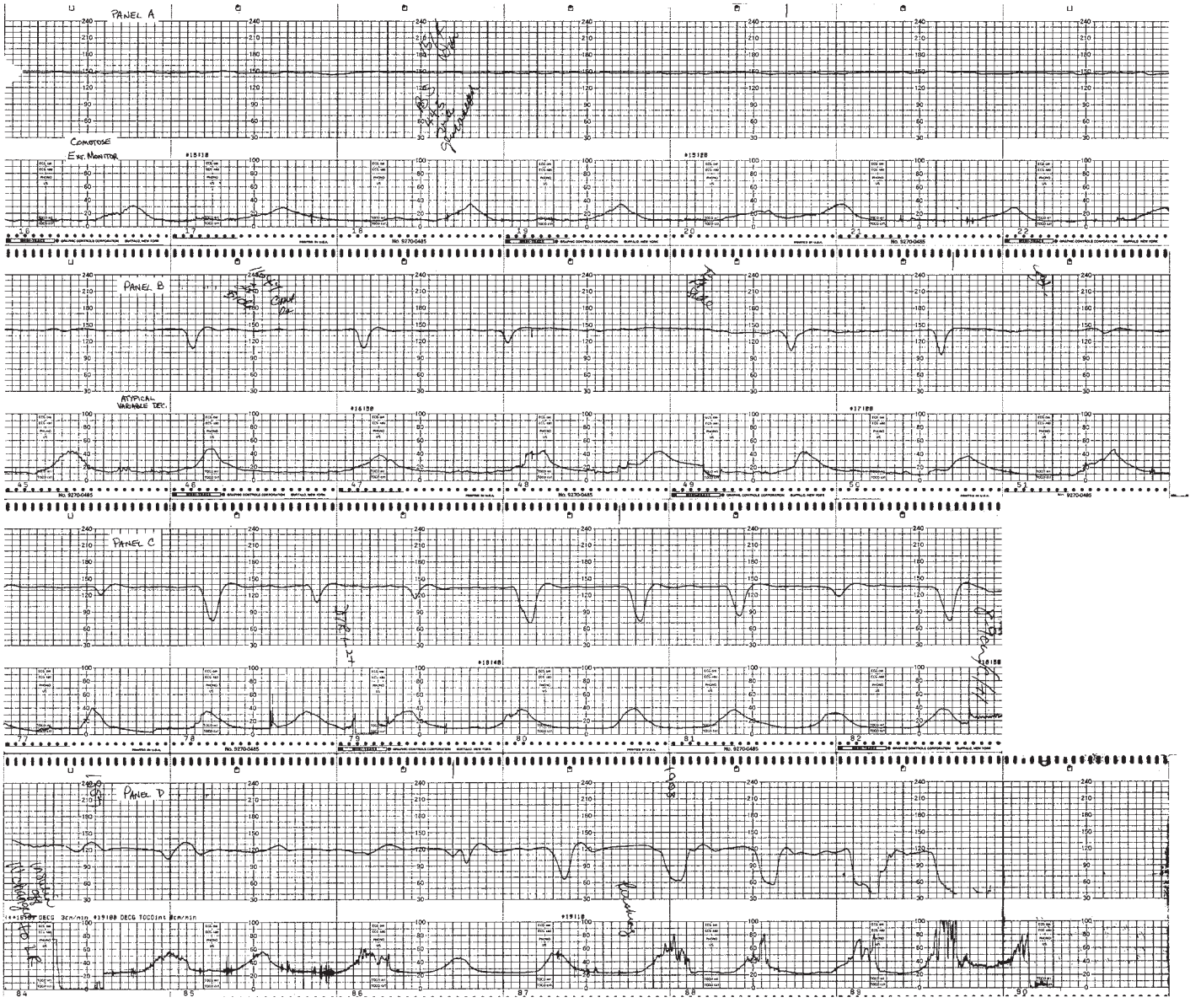

Figure 8.37. Case 8.

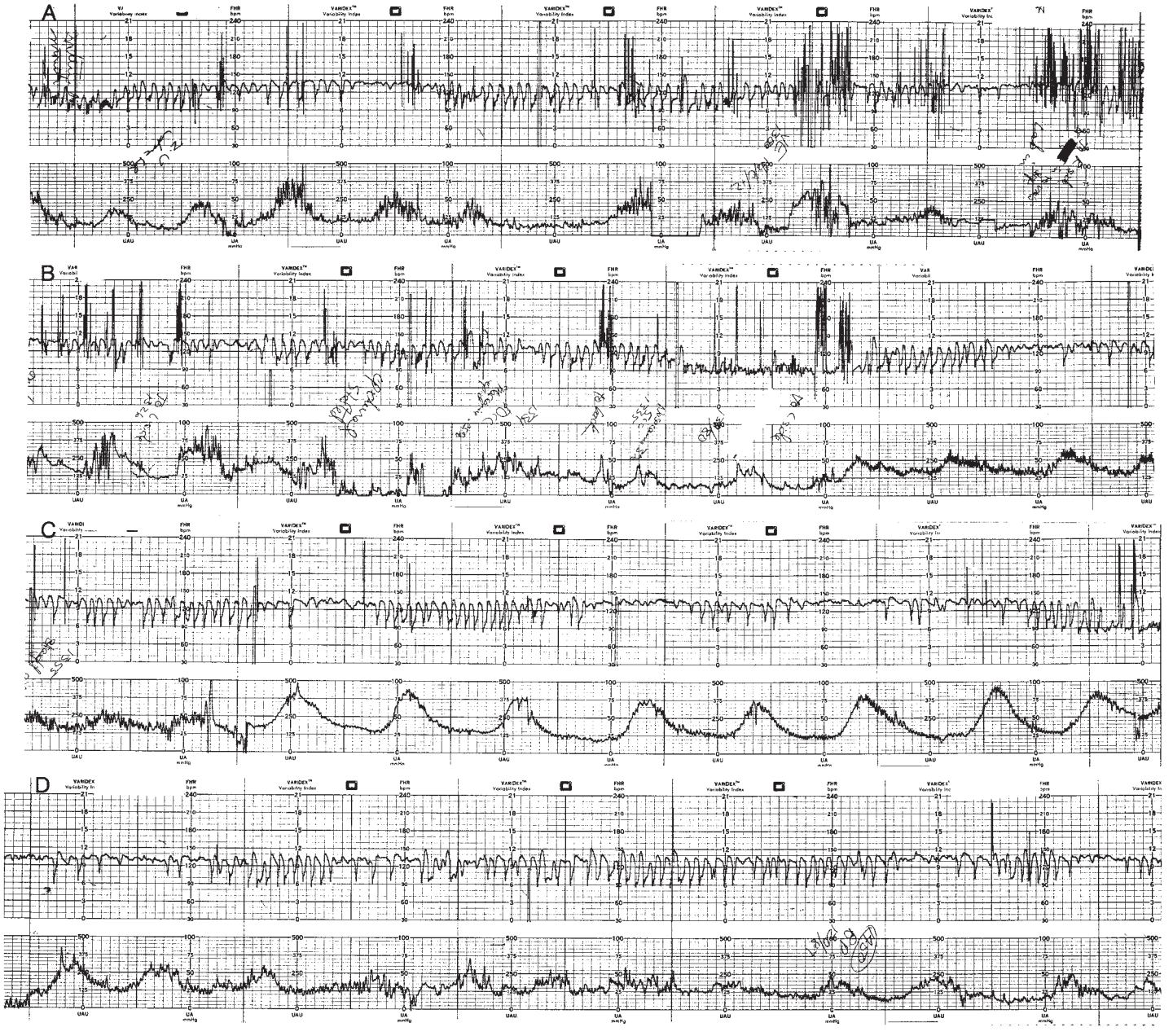

Figure 8.38. **A–D:** Case 9.

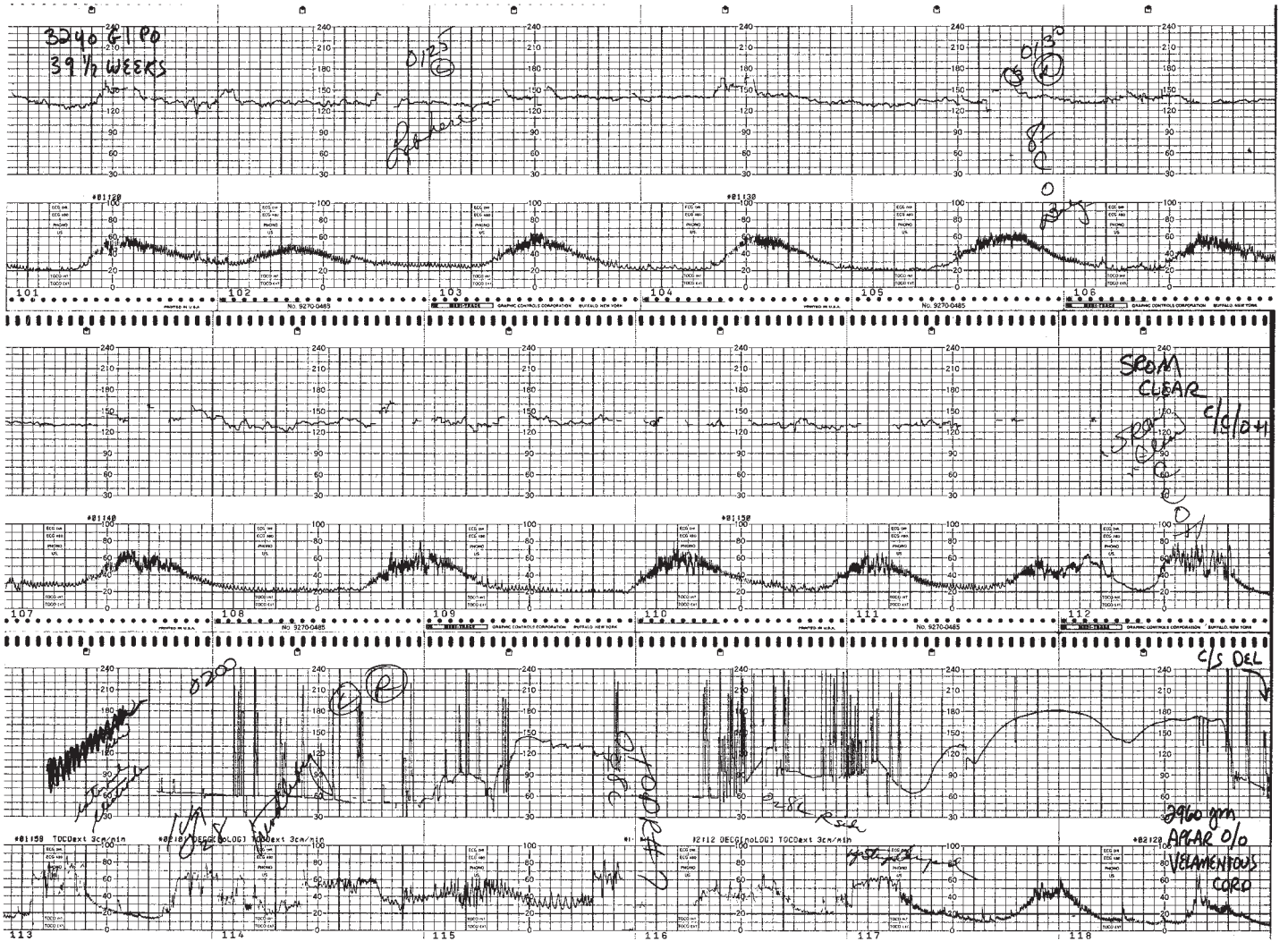

Figure 8.39. Case 10.

SUMMARY

The management of abnormal FHR patterns in labor is a complex process that must be viewed in the context of both the limitations of the modality and the goal of erring on the side of avoiding fetal death and damage (Table 8.4). Generally, this means that in many circumstances, one will intervene operatively only to deliver a vigorous baby without evidence of acidosis. The likelihood of a metabolic acidosis overall in labor is approximately 1% to 2%. Although there is a very high correlation between normal FHR patterns and normal Apgar scores, there is a poor correlation between abnormal patterns and low Apgar scores. Fewer than half of patients with abnormal FHR patterns will have newborns with 5-minute Apgar scores below 7. However, because we do not yet know where the point of permanent sequelae to intrapartum fetal hypoxia lies, the best approach should be one in which the endpoint is for the best outcome rather than for the highest correlation, provided the incidence of intervention is not excessive. In the presence of most concerning FHR patterns, the clinician should find additional ways to assess for the absence of a metabolic acidosis. Using modalities for this purpose, such as scalp and acoustic stimulation, will safely result in reducing unnecessary interventions.

Occasionally, a depressed baby will be born following a normal FHR pattern. The following is a list of factors that could explain low Apgar scores with a normal FHR pattern:

1. Birth trauma (breech, midforceps, shoulder dystocia)
2. Drugs (general anesthesia with prolonged induction delivery time)
3. Infection (chorioamnionitis, TORCH, group B streptococcus)

TABLE 8.4 Management of abnormal fetal heart rate patterns: a proposed protocol

The following algorithm for management is proposed:
1. When a pattern suggests the early development of hypoxia: a. Identify when possible the cause of the problem (e.g., hypotension from an epidural). b. Correct the cause (e.g., fluids and ephedrine to correct the hypotension). c. Give measures to maximize placental oxygen delivery and exchange: oxygen by face mask, lateral positioning, hydration, consider decreasing or discontinuing oxytocin.
2. If the pattern becomes or remains abnormal and the previous measures have been completed:
a. Attempt to identify other measures of fetal well-being to rule out metabolic acidosis.
Accelerations—spontaneous or elicited
b. If reassurance using one of the above methods can be provided, and the pattern persists, continuous or intermittent (every 30 min) evidence of absence of acidosis must be ascertained.
c. If fetal accelerations or a normal fetal pH cannot be provided, deliver expeditiously by the safest and most reasonable means (operative vaginal or cesarean section).
Patterns that cannot be corrected and therefore warrant evidence of possible metabolic acidosis include Persistent late decelerations (>50% of contractions) Variable decelerations With developing tachycardia and loss of variability With developing slow return to baseline Sinusoidal tracing Recurrent prolonged decelerations The patient comes in with a pattern of absent variability but without explanatory decelerations. An unusual or confusing pattern that does not fit into one of the categories defined

4. Meconium aspiration/resuscitation
5. Congenital malformations
6. Extreme prematurity
7. Acute fetal-maternal hemorrhage (monochorionic/diamniotic twins)
8. Upper airway obstruction

We have seen instances in which a physician indicated that the FHR pattern was completely normal, none of the above factors were present, and there was an unexplained low Apgar score. In this situation, it is common to find either that the FHR pattern was a poor quality external tracing without sufficient clarity to define the FHR pattern or that there were unrecognized decelerations present that were missed by the clinician. A common cause for poor correlation with outcome is when the monitoring is discontinued while the patient is moved to the operating room and never reestablished despite the passage of several minutes before surgery. Recently, we have seen several cases in which the electronic monitor is actually recording maternal and not FHR during extended periods of time at the end stages of labor using the external monitoring options. Care must be taken by all that the information generated by the monitor is in fact from the fetus and not from the mother who may be experiencing a significant increase in her heart rate with the efforts associated with managing her contractions and pushing in the second stage of labor.

Only by understanding the limitations of our present tools for intrapartum fetal evaluation and keeping in perspective the goals of avoiding damage as a result of intrapartum hypoxia and acidosis can one achieve the optimum balance between a good perinatal outcome and a minimum of unnecessary operative intervention.

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CHAPTER 9

Alternative and Backup Methods to Improve Interpretation of Concerning FHR Patterns

The majority of fetal heart rate (FHR) patterns seen in labor at some point will evolve into those that are neither clearly indicative of fetal hypoxia (Category III) nor clearly not associated with hypoxia (Category I) and thus fall into Category II. As clarified in the bulletin by the American Congress of Obstetricians and Gynecologists (ACOG), some of these patterns, such as milder degrees of variable decelerations with average variability, are unlikely to be indicative of a deteriorating or concerning status (1). And, while interventions such as oxygen, fluid, and position change may be warranted, these patterns can be watched to be sure they do not deteriorate into one that is more concerning. Others in this category, such as recurrent late decelerations with moderate or reduced variability, are indeed associated with some degree of hypoxia, and the question becomes, is the hypoxia severe enough that the fetus has become or is becoming acidotic as well? As a basic premise, the purpose of electronic fetal monitoring (EFM) is to detect fetal hypoxia and acidosis at a point where, if it cannot be reversed, intervention by cesarean section or operative vaginal delivery can occur before damage to fetal tissues and organs or fetal death occurs. As repeatedly pointed out elsewhere in this text, the limitation of EFM is primarily that many other variables affect the FHR and may mimic changes that occur when there are hypoxia and the resulting acidosis. Thus, in many situations, some alternative or "backup method" for more specifically assessing the degree of hypoxia and/or acidosis is needed.

When EFM was first coming into general use in the United States and still in many countries, particularly in Europe, this question is answered with scalp pH sampling. But in the United States and many other countries, scalp pH sampling has been largely abandoned due to its technical difficulty, its need for frequent repetition, and the problem with distinguishing between relatively innocuous and more common respiratory acidosis and far more serious metabolic acidosis. Several newer methods of assessing fetal status in the face of more concerning Class II patterns have evolved since the introduction of scalp pH sampling including the utility of spontaneous or evoked FHR accelerations, fetal pulse oximetry, fetal electrocardiogram (ECG) waveform analysis (STAN technology), and computer interpretations of FHR pattern. These backup methods, the reasoning behind them, and their strengths and weaknesses will be the subject of this chapter. A few other methods that showed promise for this purpose such as continuous pH monitoring and fetal pO_2 monitoring have been tried experimentally but never put into clinical practice and will not be discussed.

FETAL SCALP pH SAMPLING

The physiologic basis of the respiratory changes that occur with substantial and prolonged hypoxia, the conversion to anaerobic metabolism, and the resulting acidosis are discussed in Chapter 2. Ideally, the direct measurement of oxygen levels or pH would answer the question of the severity of hypoxia and/or the presence of acidosis and thus the logic behind fetal scalp pH sampling that came into use shortly after or concurrently with the introduction of EFM. While not a measurement of central or arterial pH as would be ideal, the fetal scalp is usually the only part that can be accessed in labor, and measurement of the pH from capillary blood obtained from the scalp in most situations accurately provides information that can be used to predict the central pH of the fetus at any given point in time.

Equipment Necessary for Fetal Scalp Blood Sampling

Before sampling fetal blood, the proper equipment must be available, and there must also be an assistant to help hold the patient in position, make notations on the monitor, connect the light source to the battery, accept the filled capillary tubes from the physician, and prepare them properly for the laboratory.

A sterile tray (now available with disposable items) should contain the following: (a) four or five 200-μL heparinized capillary tubes, (b) a conical endoscope with light source, (c) a 2-mm blade on a long handle, (d) silicone grease, (e) 10 or 15 sponges, and (f) a long-handled sponge holder. The physician should wear sterile gloves, and the patient should be prepped and draped in a sterile manner.

Technique of Fetal Scalp Blood Sampling

The optimum position of the patient for fetal scalp blood sampling is important. The lithotomy position, with a patient in stirrups and the maternal buttocks extending over the edge of the table, is preferred by most. This can be done in most convertible labor beds, but, if not available, it is easily accomplished in the delivery room. The lateral Sims' position is also satisfactory, requires less patient movement, and allows the patient to remain in the lateral position. When using the lateral Sims' position, it is important that the patient be well flexed at the hip with the lower leg extended. The upper leg is held by an assistant with the patient's buttocks extending well over the edge of the bed to allow the person taking the scalp sample to be positioned below the level of the maternal vagina. With both lithotomy and Sims' techniques, the most important factor is for the scalp sampler to be able to angle the cone toward the maternal spine.

With the patient in position, the cone (with light source) is inserted into the posterior fornix under direct visualization. Once the cone is inserted past the anterior lip of the cervix, the cone is angled anteriorly into the cervix and the presenting part is visualized. A sponge is used to wipe the scalp surface clean, and then silicone grease is applied to form a nonwettable surface that will allow the fetal scalp blood to form in easily accessible beads. A standard fetal scalp blade with a depth of 2 mm is then used with a quick "stab" to make a clean incision and blood will appear. A 200-μL heparinized capillary tube is then inserted to touch the drop of blood, and keeping the tube angled downward, the blood is allowed to flow by gravity. About one-fourth of a tube of blood without bubbles is needed for a pH, but for complete fetal scalp blood gases (pH, pCO_2, and base deficit), the tube should be about three-fourths full. After taking the sample, the capillary tube should be immediately handed to an assistant for proper sealing and mixing with a magnetic "flea." Pressure with a sponge should be kept on the scalp wound through the next two contractions, and it should then be observed during another contraction to be sure the bleeding has stopped. Sometimes, more pressure is required, and other times (rarely), it may be necessary to put a skin clip on the wound to stop the bleeding. Once fetal scalp blood sampling has been done, continued observation of the patient must be carried out, as even what appears to be "heavy show" during labor may be significant fetal hemorrhage from the scalp puncture site (2).

Indications for Fetal Scalp Blood Sampling

Total agreement on the indications for fetal scalp blood sampling does not exist. Certainly, if an institution cannot provide 24-hour ready access to accurate micro blood gas analysis with a 10- to 15-minute turnaround time, fetal scalp blood sampling should not be used. The ability to implement decisions for rapid operative intervention is also necessary to effectively use this technique.

Given the necessary logistical support, the indications for fetal scalp blood sampling should be limited to patients who are in labor with ruptured membranes and cervical dilatation sufficient to allow introduction of the cone (usually 2 to 3 cm) and with the fetal head at a station that is within 2 cm of the spines. Fetal scalp blood sampling for acid-base studies should be limited to patients who have electronic FHR tracings suggestive of hypoxia or to clarifying a confusing pattern such as absent variability without decelerations or accelerations. The better one understands FHR monitoring, the less necessary fetal scalp blood sampling will be. Examples of situations where fetal scalp sampling may remain a good choice in modern clinical practice include

1. A confusing FHR pattern is present with elements that suggest fetal hypoxia.
2. There is a sustained flat FHR, especially at the time of admission, without significant periodic changes.
3. Uncorrectable late deceleration with moderate or minimal variability is present in a patient for whom vaginal delivery is anticipated within 1 to 2 hours.

Correlation between Fetal Heart Rate Patterns, Fetal pH, and Outcome

A pH of 7.25 or greater indicates the absence of acidosis and the pattern can be safely watched unless it worsens, a value between 7.20 and 7.25 is equivocal and needs to be repeated immediately, and a value of <7.20 is consistent with acidosis and should warrant operative intervention unless delivery is imminent. It should be reemphasized that whenever possible the fetal pCO_2 should be simultaneously measured and taken into account. A respiratory acidosis alone is not an indication for delivery. Only if acidosis occurs and appears to be metabolic should a low pH be considered potentially harmful to the fetus.

Early studies on the correlation between FHR patterns and fetal scalp blood pH revealed that there was at least a general correlation (3–10). Kubli et al. (11) showed that it was indeed rare to have a fetal scalp blood pH value below 7.20 with an innocuous FHR pattern. However, many patterns of late deceleration and moderate-to-severe variable deceleration were often associated with fetal scalp blood pH values above 7.20. Furthermore, approximately 10% of fetal scalp blood pH samples obtained at the time of delivery were found to be below the values found in the umbilical artery (12).

The correlation between fetal scalp blood pH measurement and neonatal Apgar score increases as the sample is taken closer to the time of birth. With samples taken within 5 minutes of delivery, Hon and Khazin (5) and Modanlou et al. (6) showed that the correlation between low scalp pH and low Apgar scores at both 1 and 5 minutes was very high. However, there appears to be a rather poor correlation between fetal pH and Apgar scores between 7 and 10, with a relatively high incidence of falsely low values. This may be accounted for partially by local factors such as stasis that may make the fetal pH low at the scalp when the central fetal circulation is normal, especially at the time of delivery when caput formation is the greatest.

Therefore, scalp pH appears to be a reliable method of backing up a concerning FHR pattern. The limitations are however substantial primarily from a technical difficulty standpoint and the need to repeat the test every 20 to 30 minutes, and thus, the test is rarely used in the United States. Thus, there is a real need for an alternative method to clarify FHR patterns that are concerning for hypoxia and acidosis (Category II) but not more clear-cut as with Category III patterns.

ACCELERATIONS OF THE FHR AS A SUBSTITUTE FOR FETAL SCALP BLOOD SAMPLING

In 1982, Clark et al. (13) reported on 200 patients who had fetal scalp blood sampling and noted that none were acidotic if there was an FHR acceleration associated with the fetal scalp blood sampling. In a subsequent prospective study, they found that no fetus with a fetal scalp blood pH below 7.19 demonstrated acceleration at the time of scalp sampling. (14) In 1986, Rice and Benedetti (15) showed that, in patients with a concerning FHR pattern, 70 of 71 fetuses with acceleration had a pH above 7.20, whereas 7 of 32 with no acceleration had fetal scalp blood pH values below 7.20 (Figs. 9.1 and 9.2).

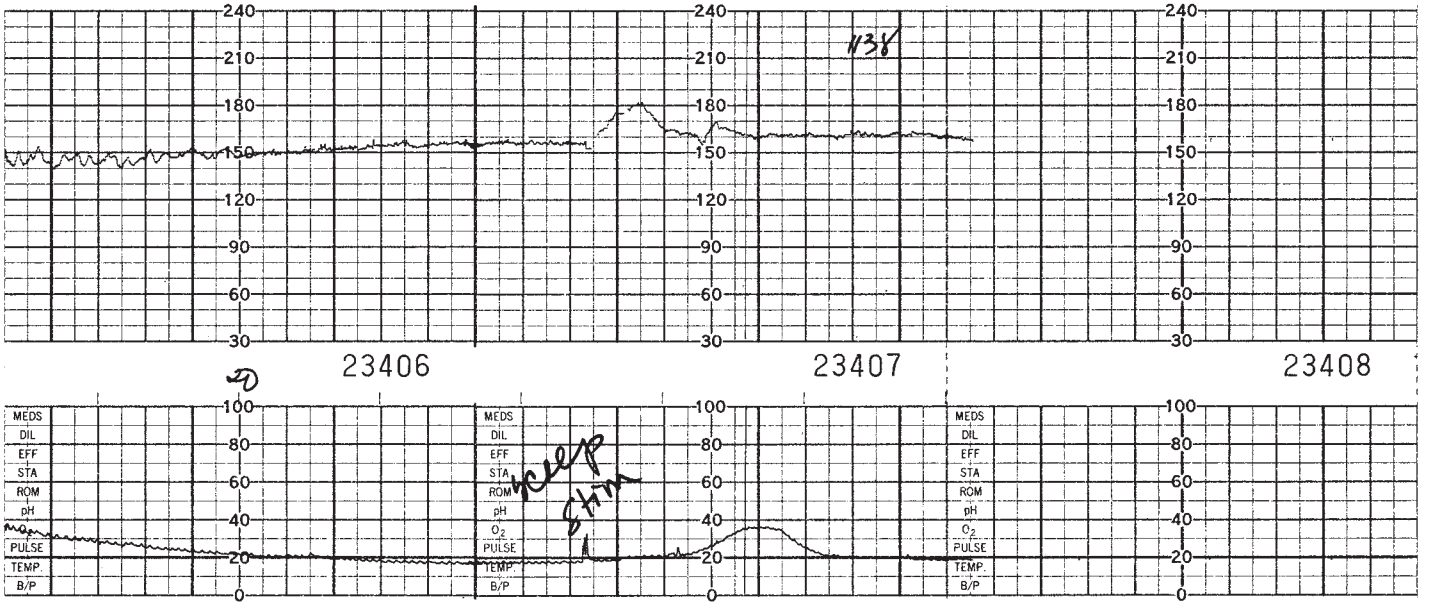

Figure 9.1. Example of fetal heart rate acceleration in response to fetal scalp stimulation.

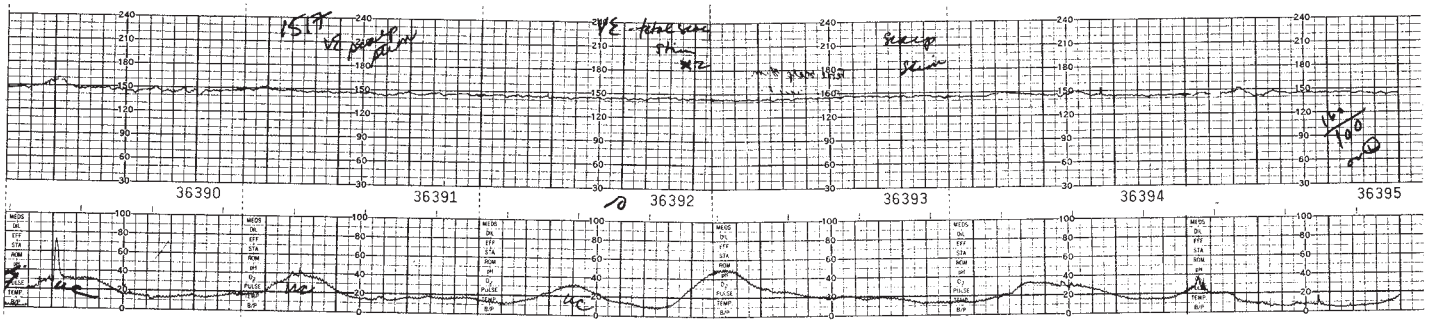

Figure 9.2. Example of no fetal heart rate acceleration in response to fetal scalp stimulation.

Smith et al. (16) reported on a similar correlation between fetal acceleration in response to sound stimulation with an artificial larynx (also known as a vibroacoustic stimulator). In their study of fetuses with abnormal FHR patterns, all 30 fetuses that showed one or more accelerations in response to sound had a pH above 7.25, while 18 of 34 that showed no acceleration with sound were acidemic. It has subsequently become clear that accelerations regardless of whether they are occurring spontaneously or elicited by scalp or acoustic stimulation have a similar meaning (i.e., the fetus is not acidemic). The converse however is not necessarily true. The fetus that is not moving will not have accelerations regardless of its acid-base status, and there will be no accelerations present. Thus, the primary place for judging whether the fetus is acidotic or not based on accelerations is in the situation where the remainder of the FHR pattern is concerning (certain types of Category II more likely to be associated with fetal hypoxia). In this setting, accelerations, either spontaneously occurring or elicited by fetal scalp stimulation or acoustic stimulation, indicate that the fetus fetal scalp blood pH at that point in time would unequivocally NOT be acidotic. If the fetus does not respond with acceleration to either of these stimuli, in the setting of an otherwise concerning FHR pattern, it would appear that approximately 50% will show acidosis on a simultaneously obtained fetal scalp blood sample. Because most hospitals with obstetrics units do not have fetal scalp sampling available (17), this approach has become the alternative to fetal scalp sampling. In the vast majority of institutions in the United States, fetal scalp stimulation and acoustic stimulation have virtually replaced fetal scalp blood sampling without an increase in cesarean section rates for nonreassuring fetal status on the EFM recording (18). It is inappropriate to use scalp or vibroacoustic stimulation during a deceleration. During an ongoing prolonged deceleration or bradycardia, first there are no data in the literature to help understand whether the presence or absence of an acceleration has the same meaning. More importantly, even if the acceleration did mean the absence of acidosis at that point, there is no assurance that, in the face of potentially rapidly deteriorating oxygenation, a few minutes later the fetus would not become acidotic. The value of these techniques is when the stimulation occurs during a time when the FHR is at its baseline rate and the acceleration evoked is above the baseline. Also, like fetal scalp sampling, if an acceleration does occur but the pattern remains the same or worsens, an acceleration must be seen every 20 to 30 minutes to again be reassured of the absence of acidosis.

It must be reemphasized that accelerations in these studies, and thus in clinical use, are used as a method of determining, only in the setting of a concerning pattern, whether the fetus may not be acidotic. Looking for the presence of accelerations in the first 30 to 60 minutes at the time the patient is admitted is also a valuable tool, since a hypoxic event at some time prior to admission could render the fetus acidotic. But, except for evaluating the fetus on admission, the absence of accelerations in the absence of an otherwise concerning pattern, is rarely indicative of acidosis, simply means the fetus in not moving, and should not be an indication for eliciting accelerations with tactile or vibroacoustic stimulation.

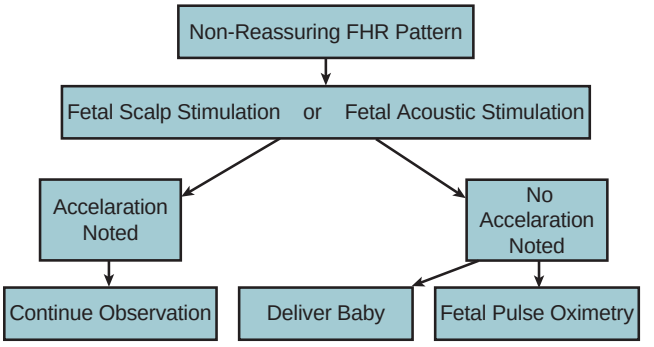

Figure 9.3. Algorithm for management of nonreassuring fetal heart rate patterns with fetal scalp stimulation and fetal scalp blood sampling.

It is thus left for the clinician to use electronic FHR monitoring, fetal scalp blood sampling, and the response to fetal scalp or sound stimulation as complementary methods that are still only parts of the whole clinical picture to be appreciated in making appropriate decisions for intervention or nonintervention (Fig. 9.3).

FETAL PULSE OXIMETRY

Since the early 1980s, monitoring of oxygen saturation using pulse oximetry has been available for "air-breathing" adults and children and is now used in virtually every operating room and intensive care unit. By 1985, its use had become so pervasive that 95% of all patients in operating rooms in the United States were monitored with this device. Oxygen is present in the blood in two forms. In the plasma, oxygen is dissolved and unbound. This unbound form accounts for approximately 1% of total blood oxygen, and it is the portion responsible for all oxygen diffusion. Dissolved oxygen is measured as partial pressure, or pO_2. The remaining 99% of oxygen in blood is bound to hemoglobin. This bound form is measured as oxygen saturation *in vitro* by co-oximetry and *in vivo* by pulse oximetry and are both expressed as percent saturation. The percent saturation is the percentage of hemoglobin binding sites occupied by oxygen molecules (saturated hemoglobin) divided by the total number of binding sites (saturated + unsaturated hemoglobin). The measurement of oxygen saturation using pulse oximetry involves two principles. The first is that red and near-infrared light are alternately shined into tissue, and the absorption of the transmitted or reflected light is measured. Deoxyhemoglobin absorbs red light better and oxyhemoglobin infrared; thus, using standard curves, the oximeter is able to calculate the relative amounts of saturated and unsaturated hemoglobin

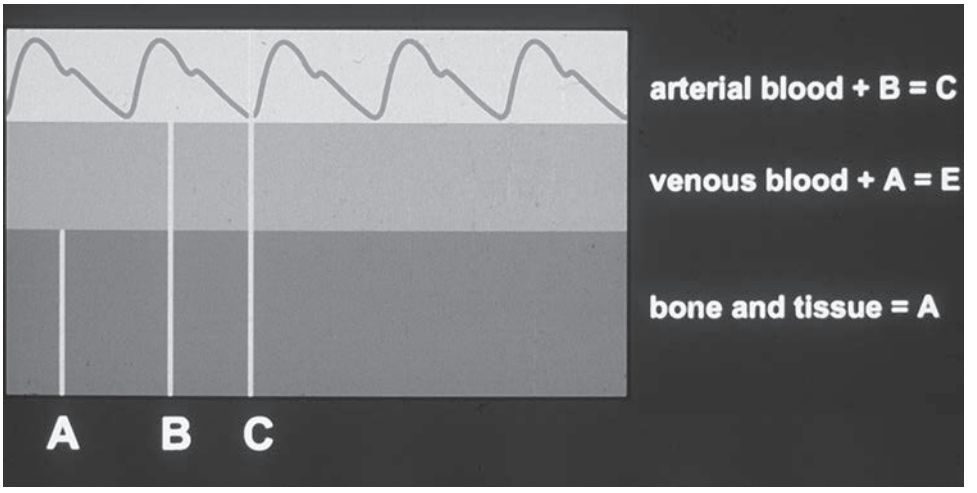

Figure 9.4. This figure illustrates how the pulse wave is used to separate the light absorption of arterial blood from that venous blood and surrounding tissue.

and determine percent oxygen saturation. To determine the light absorbed by arterial blood as opposed to venous blood and surrounding tissues, the oxygen saturation is determined at the peak of the pulse wave (equal to light absorbed by arterial and venous blood and surrounding tissue) and the nadir of the pulse wave (light absorbed only by venous blood and tissue) (Fig. 9.4). By subtracting the amount of light absorbed at the nadir from that absorbed at the peak, the amount remaining is that absorbed by arterial blood.

Fetal Pulse Oximetry

During labor, the only site that we have access to is usually the fetal scalp. The cervix must be sufficiently dilated and the membranes ruptured to have access. The scalp is covered by hair, and often mucous, blood, vernix, meconium, and vaginal secretions, all of which can interfere with light transmission and photodetection. The amplitude of the fetal pulse wave is small due to a low fetal pulse pressure, hindering accurate calculation of arterial saturation. In addition, when the scalp is used, there is often stasis within the developing caput, hindering access to the fetal pulse wave. The level of fetal oxygen saturation ($FSpO_2$) is normally only approximately 55%, and conventional oximeters do not work well at these low levels as they are optimally calibrated to work at levels in the range of 90% to 100%. Finally, fetal hemoglobin is different from that of the adult, altering light absorption characteristics. All of these complex factors and interactions have hindered the development of fetal pulse oximetry. Early efforts at trying to overcome these problems led to the testing of a number of rather creative sensors. Suction cups, glue, and clips were tried. Scalp electrodes with photoemitters and the photodetector placed at the tip of the spiral and base of the electrode were the dominant method tried for many years. None of these devices overcame the hurdles created by the scalp, the interfering fluids, and the caput.

A completely different approach to sensor placement was introduced that eliminated many of these hurdles. This device is inserted transcervically, much like an intrauterine pressure catheter, to ultimately lodge against the fetal cheek (Fig. 9.5). On the surface of the device are three gold electrodes to determine adequate electrical contact, a photoemitter, and a photodetector. The sensor is connected to a fetal pulse oximeter that processes the signal and calculates and displays the oxygen saturation. The pulse oximeter is connected to a conventional FHR monitor that displays the signal on the lower (contraction) channel of the fetal monitor tracing. This provides a continuous tracing of $FSpO_2$ that can be correlated with heart rate changes and uterine contractions (Fig. 9.6). A concept that is essential to the utility of $FSpO_2$ monitoring is termed the "critical threshold," which is defined as a level of fetal hypoxia below which and

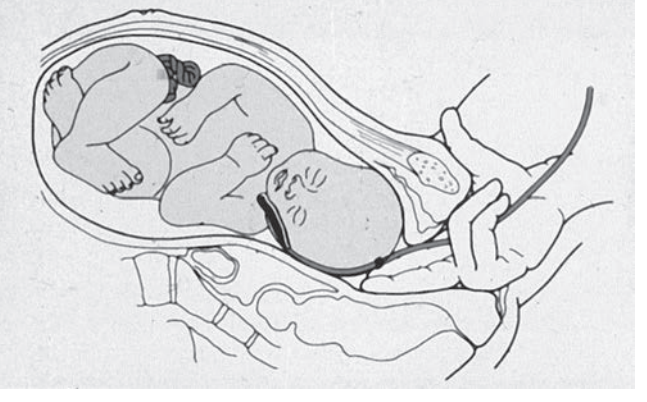

Figure 9.5. Picture of a pulse oximeter probe inserted transcervically to ultimately lodge against the fetal cheek. The catheter is inserted much like intrauterine pressure catheter. The sensor is held against the fetal cheek by the pressure provided from the uterine wall and pelvic sidewall.

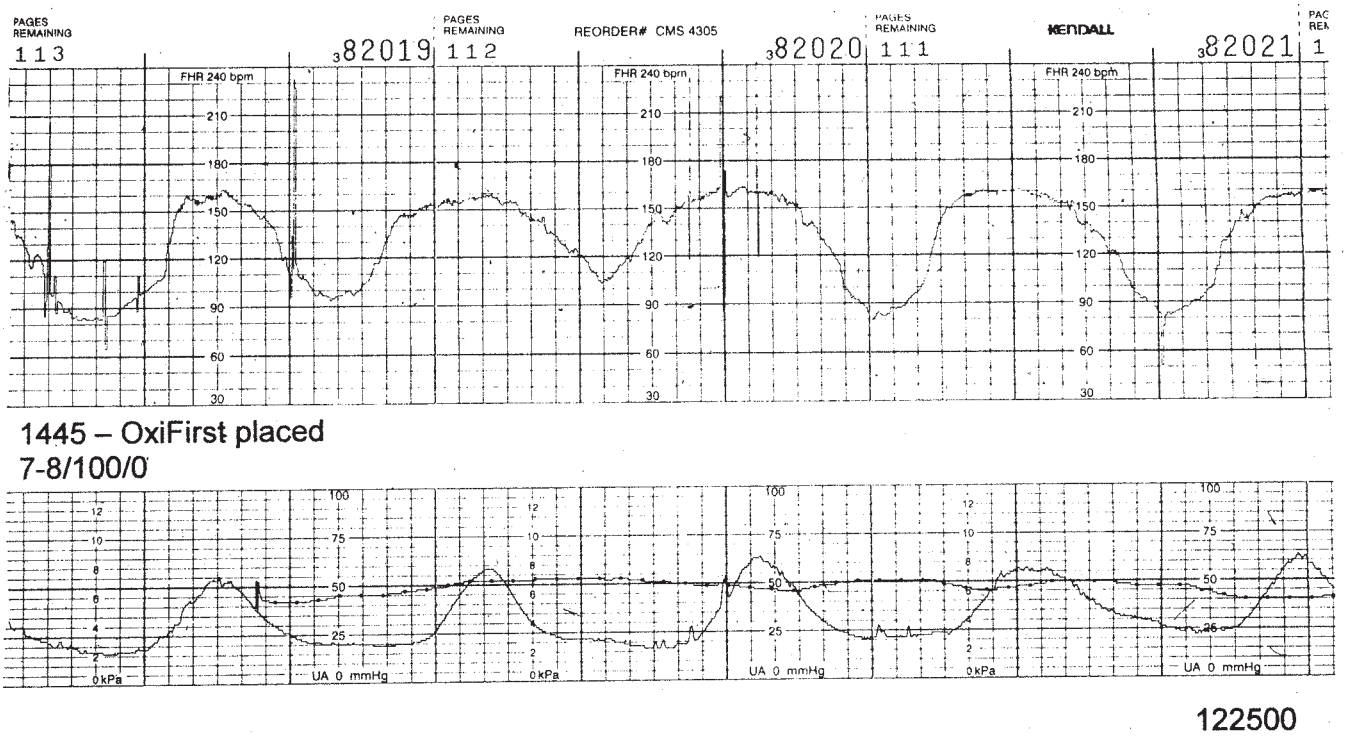

Figure 9.6. This fetal heart rate tracing with a typical heart rate on the upper channel and contraction on the lower channel and is also continuously displaying the fetal oxygen saturation on the lower channel superimposed on the contractions.

if sustained for a defined duration would likely be associated with a metabolic acidosis and above which there would be virtually no risk of acidosis. Animal studies have confirmed that the fetus will not become hypoxic unless the oxygen saturation falls below 30% (19,20). Human studies (21–24) have demonstrated normal oxygen saturation values for the human fetus in labor (usually between 40 and 55%), validated the 30% critical threshold, and compared outcomes of fetuses with normal and abnormal values (21,24,25). Studies have confirmed that in fetuses with oxygen saturation values that do not fall below a level of 30% for longer than 10 minutes have a very low risk of fetal metabolic acidosis. Thus, it appears, using this concept of the critical threshold, that in the presence of a nonreassuring FHR pattern, fetal pulse oximetry is equivalent to scalp pH in evaluating the question of whether the hypoxia has progressed to a metabolic acidosis. Furthermore, an $FSpO_2$ value below 30% must be present continuously for some minutes before there is a likelihood of significant fetal acidosis.

While this modality showed great promise as a backup method for many Category II potentially concerning FHR patterns, two large randomized clinical studies were not able to prove that using this modality in clinical practice could substantially improve our ability to avoid unnecessary cesarean sections or operative vaginal deliveries done for concerning FHR patterns but not associated with depressed or acidotic babies (26,27), although at least two studies did demonstrate a reduction in metabolic acidosis at delivery (26,28). While the modality was indeed safe, it did add expense and without clear proof of efficacy could not be endorsed (29). Despite the fact that many centers had begun to use fetal pulse oximetry in clinical practice and that it had been approved for clinical use by the FDA, it was abandoned and removed from the market by its manufacturer. Nonetheless, given the impact this technology has made in adult and pediatric medicine and the logic behind its application with EFM, it is very possible that with improvements in the engineering and software technology behind it, we may see its reemergence for this purpose sometime in the future.

FETAL ECG ANALYSIS

One of the most recent developments in technologies to assist in managing and more accurately detecting fetal hypoxia and acidosis in labor has been the introduction of fetal ECG analysis. Marketed by Neoventa of Sweden and named the STAN technology, this method is a combination of FHR interpretation and ST analysis (Fig. 9.7). The ST analysis is based on changes in the fetal ECG waveform and the way it is affected by the myocardial adaptation to oxygen deficiency using the computerized real-time analysis of the QRS and

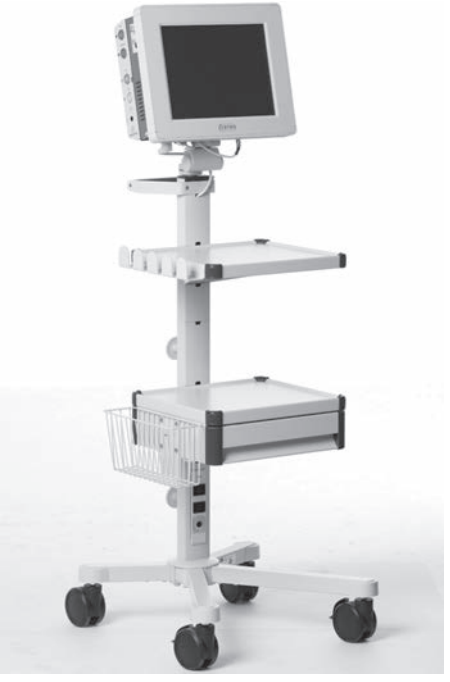

Figure 9.7. This is the device marketed by Neoventa of Sweden that contains an FHR monitor and a computer that analyzes the fetal ECG and alerts the clinician to various changes of the ECG waveform that suggest fetal hypoxia.

T-wave patterns and intervals of the fetal ECG. Specifically two types of ECG changes correlate with fetal myocardial hypoxia/acidosis. These include an elevation in the T wave as well as a downward slope in the ST segment expressed as the ratio of T/QRS. The computer program recognizes these changes and displays them on the monitor strip as "ST events" (Fig. 9.8). There are three types of ST events. Two are related to the elevation in the T wave, and one is related to the slope in the ST segment. These ST events alert the clinician to these changes in the fetal ECG, and then the clinician correlates these with the FHR pattern; if both are concerning at the same time, the likelihood of significant hypoxia and developing acidosis may be high enough to warrant intervention. If the FHR is normal or similar to the U.S. Category I, no intervention is indicated. But if the FHR is any more concerning than this and the ST events persist, the clinician is advised to use interventions to improve the pattern, and if these fail, then operative delivery is warranted.

Since its introduction, there have been four large randomized controlled trials including almost 10,000 subjects comparing STAN plus EFM and scalp sampling to EFM and scalp sampling without STAN (30–33). These trials to date have been done in Europe and Scandinavia in locations where scalp sampling is generally part of the evaluation of the fetus with concerning FHR patterns. The Cochrane review by Neilson (34) in 2006 included these four trials. This review concluded that with the addition of the STAN monitor, there were fewer babies with severe metabolic acidosis (pH <7.05 + BD >12, RR.64), fewer babies with neonatal encephalopathy (RR .33), fewer operative vaginal deliveries (RR .87), and a lower need for scalp sampling (RR .76). There were no differences in babies with Apgar scores <7 at 5 minutes nor cesarean deliveries for concern of fetal hypoxia.

While little data have been published on its use in the United States, there is currently an ongoing large multicenter trial comparing EFM plus STAN to EFM alone by the NICHD MFMU Network.

Therefore, while there are certainly strong data in favor of the use of the STAN technology, strictly speaking, it cannot truly be used as a backup method as it requires a normal FHR pattern prior to implementing the ECG analysis because the computer requires a normal fetal baseline to then be compared to the ECG after subsequent FHR changes

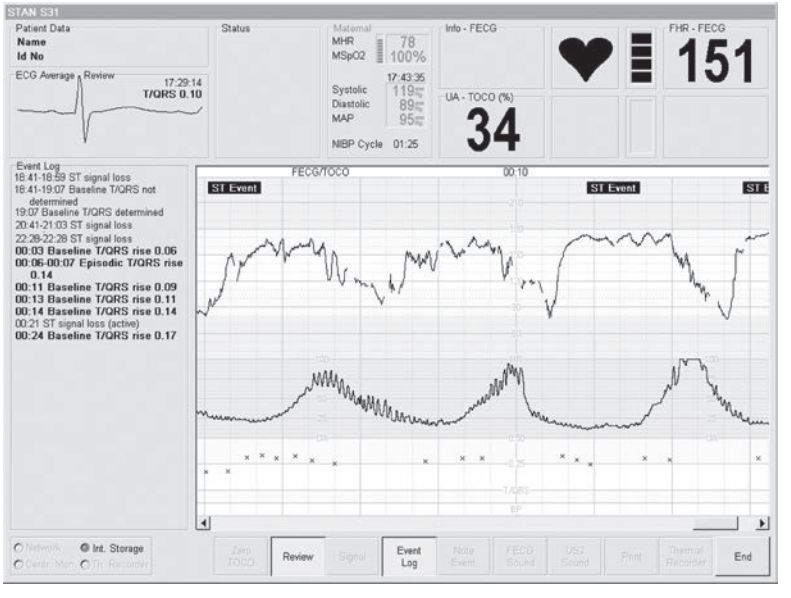

Figure 9.8. This is the tracing produced by the STAN monitor. Deep decelerations late in timing possibly suggestive of hypoxia is confirmed by several "ST events," which the ECG waveform analysis has concluded as also suggestive of fetal hypoxia.

occur. Thus, it would have to be used in conjunction with EFM on all patients in labor to be effective. Other limitations include that it has currently only been evaluated in conjunction with fetal scalp sampling, although the Network trial should clarify this issue, and that it requires an internal electrode specifically designed for the technology.

Computerized Interpretation of EFM

Since EFM is really a pattern recognition and interpretation technology that is highly complex, has a reasonable set of clear definitions at this point, and has a problem with consistency of interobserver interpretation, the use of computer algorithms to interpret the EFM and suggest interventions would seem a logical solution. For many years, investigators have made efforts in this direction and concluded that such technology added little to human interpretation. Although almost since the inception of EFM companies have included the options of activating alarms on their monitors for such things as bradycardia or tachycardia, true computer interpretation that could assist with patient management and use categorization of patterns similar to the NIH three category system have only recently been developed and are in use in a limited number of settings. Generally speaking, the methods for comparing these new systems with interpretation of the patterns by clinician experts have been reported as either sensitivity (i.e., ability to not miss concerning decelerations) or proportion of agreement (i.e., number of decelerations where computer and clinician experts agree). However, even these carefully chosen statistical comparisons can be misleading. Since the goal of EFM as a diagnostic test is to avoid injury and death, an intervention for an appropriately concerning pattern, which results in a good outcome may not be classified correctly when evaluating the ability to correctly detect concerning patterns based on outcomes. So at present, the best known available method is to compare the ability of the interpretation system to agree in interpretation of the tracing with recognized experts. For the most part, rate is accurately reported by any system, variability is in fair agreement (although little has been reported in the literature for some time and newer systems have likely improved on this), and decelerations and accelerations had proportion of agreement in the range of 45% to 70% (35,36).

More recently, systems that use neural network or artificial intelligence technology (LMS/Perigen, Princeton, New Jersey, OxSys, Oxford, UK) have been described and have demonstrated accuracies far in excess of those previously reported, with sensitivities and performance accuracy for decelerations, types of decelerations, and accelerations in the range of 80% to 95% (37–39). It should be noted that this level of performance is equal to or exceeds the level of agreement between typical clinicians and clinician experts. By combining this computer interpretation of EFM with the assessment of the FHR by the clinician at the bedside, the potential for improved interpretation of EFM patterns is great. However, this latter speculation has not yet been validated in prospective studies.

SUMMARY

Given the lack of specificity of FHR monitoring because of the influence of factors other than hypoxia that affect FHR patterns and cause confusion, and given the frequent lack of agreement over interpretation of FHR patterns, it would be ideal to employ other methods that could more specifically answer the question of whether hypoxia and/or acidosis exists with many Category II tracings. Currently, the only method commonly used, at least in the United States, is the elicitation of accelerations, either spontaneous or elicited by scalp or vibroacoustic stimulation to rule out the presence of acidemia. Other newer methods, such as fetal pulse oximetry, have been introduced but not stood the test of rigorous scientific validation. Newer methods just coming into use including ST waveform analysis and computer interpretation of FHR patterns show promise but must ultimately be subjected to more rigorous testing before they are likely to be widely accepted.

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CHAPTER 10

Evaluation and Management of Fetal Heart Rate Patterns in Premature Gestation

The potential for the beneficial effects of fetal heart rate (FHR) monitoring in the patient delivering prematurely is perhaps greater than in any other risk group. Continued dramatic improvements in the neonatal survival rates at progressively earlier gestational ages increase the necessity for evaluating fetal well-being in early pregnancies and for intervention for fetal indications. Delivering prematurely is, of course, an abnormal event, and as opposed to normal labor and delivery in the term pregnancy, the various pathologic causes of premature delivery are often scenarios in which the likelihood of fetal hypoxia is increased. The premature fetus is more likely not only to be exposed to hypoxia but also to develop, and die from, the most common and serious complications of prematurity if born depressed and/or acidotic. FHR patterns in the premature fetus, both normal tracings and those indicative of hypoxia, differ from those at term. It therefore seems appropriate to emphasize the evaluation and management of fetal hypoxia separately in the premature fetus.

EFFECTS OF HYPOXIA IN THE PREMATURE FETUS

It is clear that neonatal care is the major contributor to the ongoing improvements in survival and decreasing permanent morbidities in premature babies. An impressive body of evidence indicates that the condition in which the premature newborn is delivered has an important impact on both its likelihood to survive and its odds of developing serious complications of prematurity.

Respiratory Distress Syndrome

In the early 1970s, when electronic FHR monitoring was initiated, Hobel et al. (1) and Martin et al. (2) showed that fetal hypoxia had an impact on the outcome of the premature newborn. Both investigations demonstrated that premature babies with low Apgar scores, abnormal FHR patterns in labor, or both had higher chances of developing respiratory distress syndrome (RDS), the most common serious complication of premature neonates, and had higher chances of dying of this complication. In the series of Martin et al., abnormal FHR patterns were more predictive of RDS development than were low Apgar scores. Subsequent research has confirmed these findings. Physiologically, this makes sense as those enzyme systems responsible for the production of surfactant, which keeps the term baby's lungs open at birth, are very sensitive to acidosis in the preterm baby. Donald et al. (3) have shown that even those babies with amniotic fluid lecithin-to-sphingomyelin ratios that are indicative of fetal lung maturity may develop RDS if fetal hypoxia and/or acidosis precede delivery.

Intraventricular Hemorrhage

With improved outcomes in even smaller babies, and as the treatment of RDS has become more successful, intraventricular hemorrhage (IVH) has assumed greater importance in the morbidity of the very premature newborn. Initially, intracranial compression forces were thought to be causative, but as data have accumulated, it has become apparent that route of delivery, *per se*, has little to do with IVH (4–6). The most important risk factor for IVH is the degree of prematurity, with newborns in the range of 500 to 1,000 g being at highest risk. Ventilator therapy probably ranks next in importance due to the combination of RDS and patent ductus arteriosus in this very-low-birth-weight group (7,8). FHR patterns in labor indicative of hypoxia rank next. Babies with no RDS and normal Apgar scores at birth are less likely to develop the more severe degrees of IVH (grades 3 and 4), regardless of birth weight (8). Because fetal hypoxia and RDS are integrally related, it becomes apparent that the labor process is of critical importance in determining whether the very-low-birth-weight fetus will develop and possibly die of these two serious complications (IVH and RDS).

Necrotizing Enterocolitis

Necrotizing enterocolitis (NEC) is another important and potentially lethal complication in the premature infant. Although the cause is unknown, risk factors include infection, early feeding, umbilical arterial lines, exchange transfusion, and polycythemia (9). NEC is most likely to occur in premature infants with a history of perinatal asphyxia. The proposed mechanism is diversion of gastrointestinal blood flow to more critical organs with asphyxic conditions, resulting in ischemic injury to the bowel (9,10). In a review of FHR tracings of babies who developed NEC, Braly et al. (11) found that only 1 of 16 babies had a normal tracing, and 11 had severe FHR changes.

Death and Long-Term Injury

As one would expect, premature babies with hypoxia in labor and the above complications are more likely to die in the neonatal period (1–15). Furthermore, the abnormal FHR patterns in labor that are indicative of potential compromise have also been found to correlate with immediate and long-term neurologic sequelae. Westgren et al. (12,13) found that in premature fetuses abnormal FHR patterns to be more highly predictive of fetal acidosis and that in acidotic babies with abnormal FHR patterns, neurologic problems in the neonatal period were more frequent than those in premature controls. They also found a relationship between abnormal FHR, acidosis, and death from asphyxia in the first 2 years of life.

In general, the earlier in pregnancy that delivery occurs, the more profound the correlation between abnormal FHR patterns, neonatal depression and acidosis, and immediate and long-term complications. Most studies find these relationships to be most persistent and most profound in babies of <33 weeks' gestation with birth weights of <1,500 g (12,13,15). For complications such as severe IVH, earlier gestations, such as those of <28 weeks and with birth weights of <1,000 g, are more important.

RELATIONSHIP BETWEEN CAUSE OF PREMATURE BIRTH AND FETAL COMPROMISE

Many of the causes of prematurity are also situations that increase the probability that the fetus will be subjected to antepartum and/or intrapartum hypoxia; thus, the premature baby not only is more likely to suffer complications if it becomes asphyxiated but also is more likely to be subjected to asphyxia. The implications for management relate to the reasons for this increased exposure as it pertains to the various causes.

Abruptio Placentae

Abruption is an uncommon complication, occurring in only about 0.5% of term infants but in 5% to 10% of premature deliveries (16–18). Abruptio placentae increases hypoxic insults through two mechanisms. The separation of the placenta from the maternal decidual surface and thus the maternal blood supply, depending on degree, decreases placental surface area and thus oxygen exchange. In addition, there is often a marked increase in the frequency and, occasionally, duration of contractions, which prolongs the interval during which oxygen delivery to the intervillous space is interrupted. Because placental insufficiency is the mechanism of the fetal hypoxia, late decelerations will be the characteristic FHR pattern, and prolonged decelerations may occur in severe cases. One particular benefit of fetal monitoring is that patients sometimes present with preterm labor with minimal or no bleeding, and the contraction pattern and/or late decelerations are the earliest and only indication that abruptio placentae is the etiology of the premature labor (Fig. 10.1).

Preeclampsia

Preeclampsia is not an unusual cause of premature delivery resulting from associated premature labor, abruption, or induction of labor at an early gestational age because of the clinician's concern over the severity of the disease. Often, the onset of preeclampsia at very early gestational ages is associated with intrauterine growth restriction (IUGR) and chronic placental insufficiency. In such cases, FHR patterns may exhibit late decelerations if there is insufficient placental perfusion (Fig. 10.2) and/or variable decelerations if there are growth restriction and oligohydramnios leading to umbilical cord compression (Fig. 10.3). Clinical decisions regarding delivery of a very premature infant in a setting of abnormal FHR patterns are complex and involve the integration of both maternal and fetal issues and prognoses.

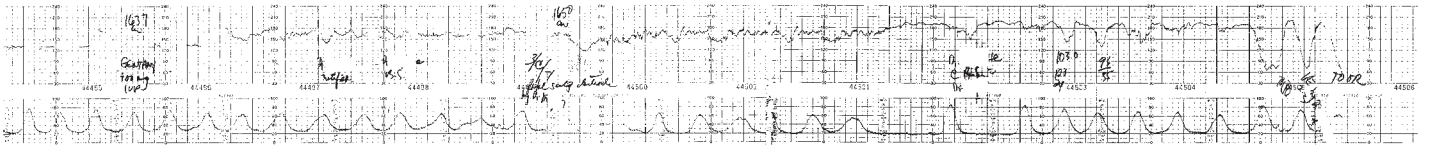

Figure 10.1. This patient presented in active labor with a maternal fever, no bleeding, and a gestational age of 32 weeks. The rise in baseline might be secondary to the maternal fever, but the onset of frequent ominous decelerations led to emergency cesarean section. A large retroplacental hematoma was present at surgery.

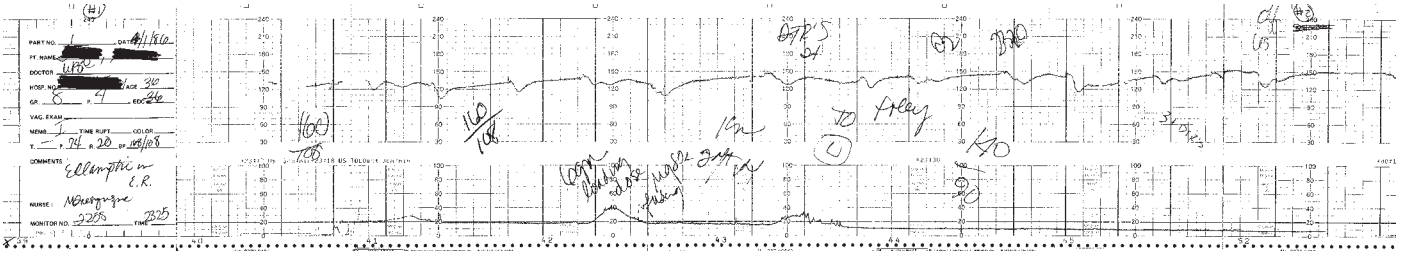

Figure 10.2. This patient presented with severe preeclampsia, and while receiving a loading dose of magnesium sulfate, she experienced an eclamptic seizure. Persistent late decelerations continued, and a cesarean section was performed following maternal stabilization. A severely growth-restricted infant with Apgar scores of 3 and 6 at 1 and 5 minutes, respectively, was delivered with evidence of moderate respiratory acidosis. Newborn course was benign.

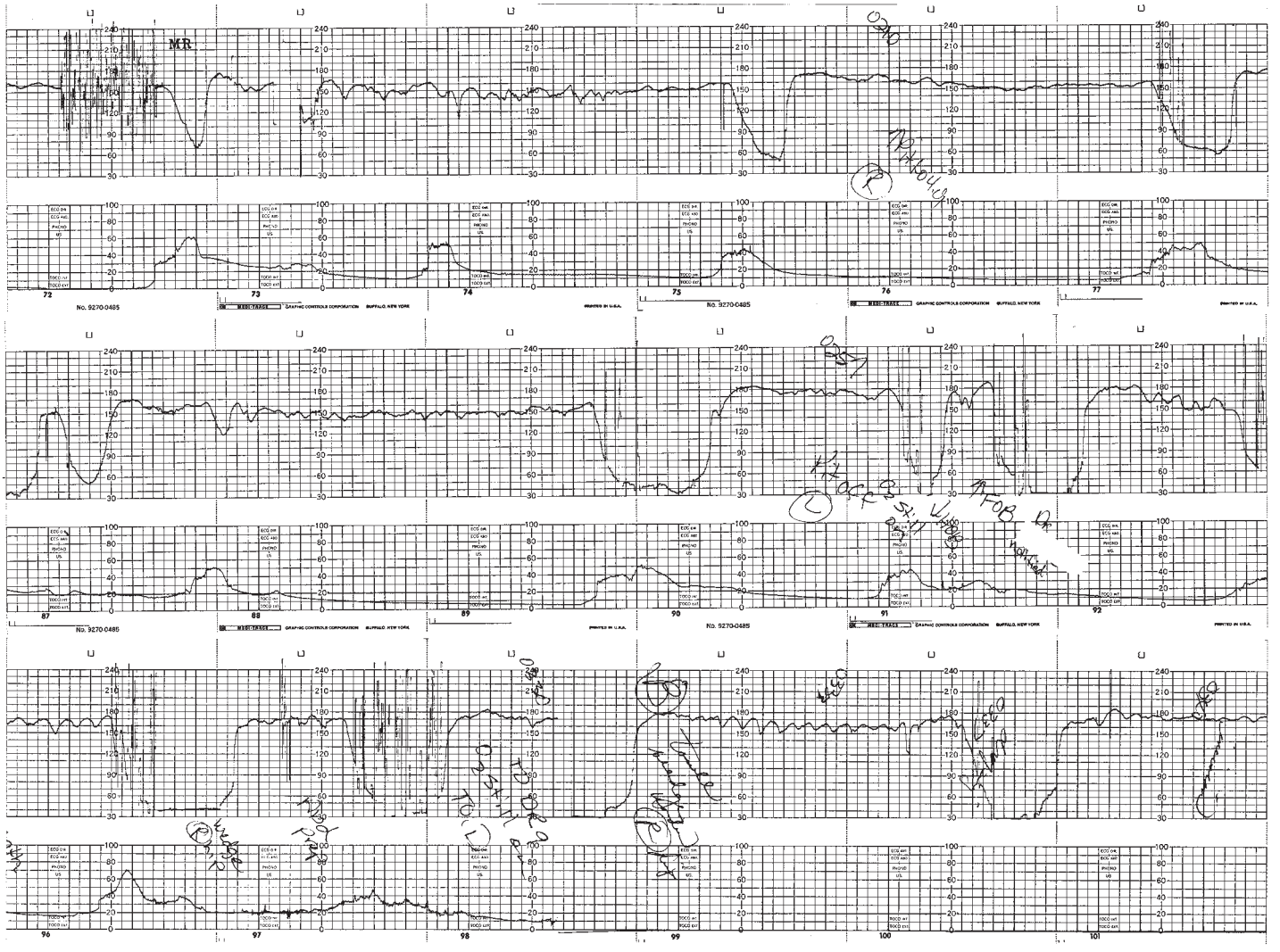

Figure 10.3. This patient with severe preeclampsia at 27 weeks illustrates the rapid development of progressively severe variable decelerations associated with developing fetal tachycardia. These patterns are similar to those seen with oligohydramnios secondary to premature rupture of membranes, but in this case, the oligohydramnios was present with intact membranes. Cesarean section yielded an 860-g baby with Apgar scores of 1 and 4 and marked respiratory acidosis.

Preterm Premature Rupture of Membranes

Preterm premature rupture of membranes (PPROM) is the most common single circumstance leading to premature delivery and accounts for approximately 35% of preterm deliveries. Patients with PPROM in preterm labor have a sevenfold greater risk of fetal compromise requiring cesarean section than patients with preterm labor with intact membranes (19). The vast majority of such patients have variable or recurrent prolonged decelerative patterns consistent with umbilical cord compression secondary to oligohydramnios (Figs. 10.4 and 10.5). Prophylactic use of amnioinfusion has been demonstrated to decrease the frequency and severity of such decelerations and increase the likelihood of allowing vaginal delivery in such patients (20).

Intrauterine Growth Restriction

IUGR may result in preterm delivery either because the problem that caused the growth restriction also leads to preterm labor, as in preeclampsia or thyrotoxicosis, or because the physician detecting the IUGR decides to intervene prematurely. In such cases, fetal hypoxia in labor is more likely due to the associated inadequate delivery or exchange of oxygen resulting in late decelerations or to the associated oligohydramnios leading to umbilical cord compression patterns (Fig. 10.6). Fetuses with IUGR are even more susceptible to damage from asphyxia than their premature counterparts of similar gestational ages.

Infections

Premature rupture of membranes (PROM) or premature labor is commonly associated with clinical or occult chorioamnionitis. FHR patterns are affected by factors such as maternal fever or fetal sepsis. A variety of FHR changes may be seen. If there is maternal fever, the fetal temperature will increase, and fetal tachycardia will develop. This tachycardia may be associated with a loss of variability from the rising FHR. The increased oxygen demands with the higher heart and metabolic rates may result in late decelerations. In septic fetuses, there may be tachycardia, loss of variability, and loss of accelerations (reactivity) (Fig. 10.7). Often, this cannot be distinguished from changes secondary to maternal fever, and the use of the FHR to identify fetal infection is a problem.

A substantial proportion of premature infants are exposed to amniotic fluid infection and an associated proinflammatory response and cytokine production. Neonatal mortality is increased by prematurity, amniotic fluid infection, and associated maternal and fetal inflammatory responses. Morbidities such as RDS, bronchopulmonary dysplasia, periventricular leukomalacia, and cerebral palsy are increased in the setting of amniotic fluid infection over and above those attributable to prematurity alone (21–25).

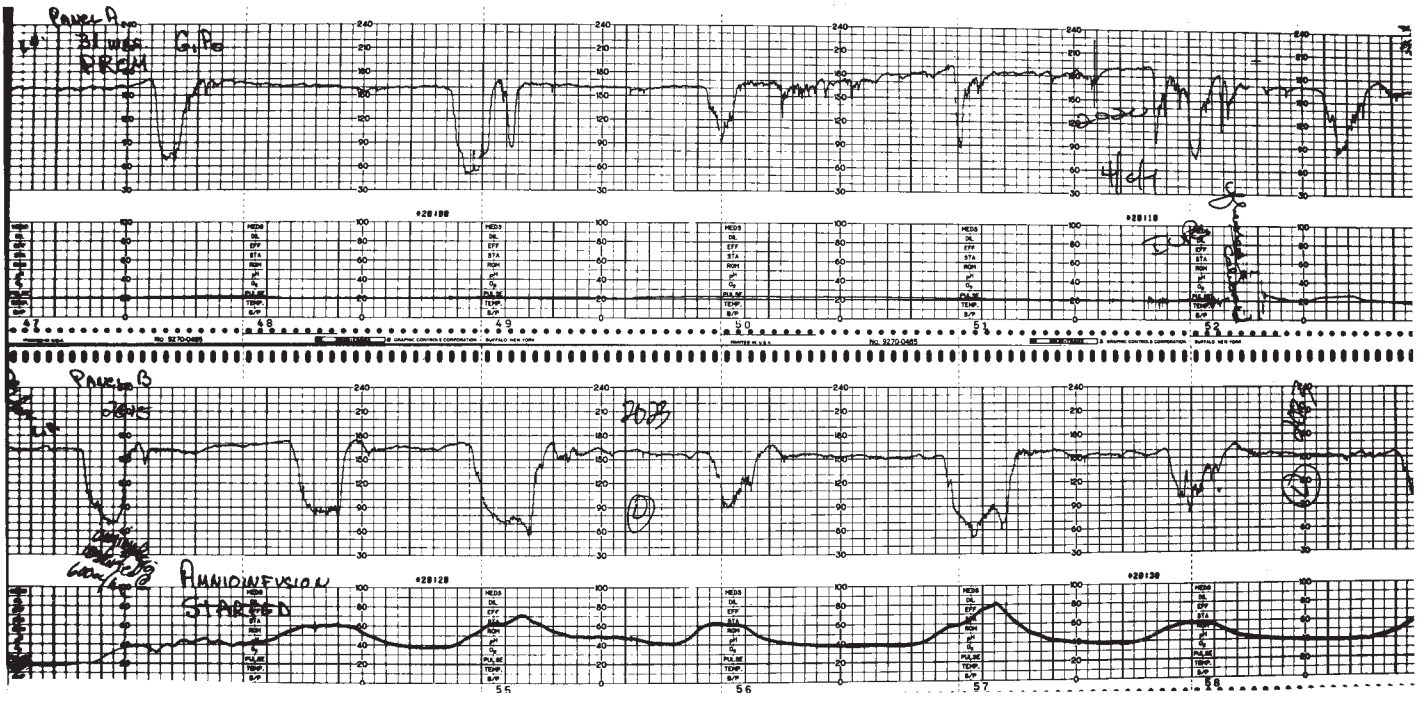

Figure 10.4. Typical variable decelerations seen in early labor (4-cm dilation) in a patient with premature rupture of membranes and oligohydramnios at 31 weeks. An attempt at amnioinfusion (seen on the **lower panel**) failed to relieve the cord compression, and the patient required cesarean section for a worsening pattern.

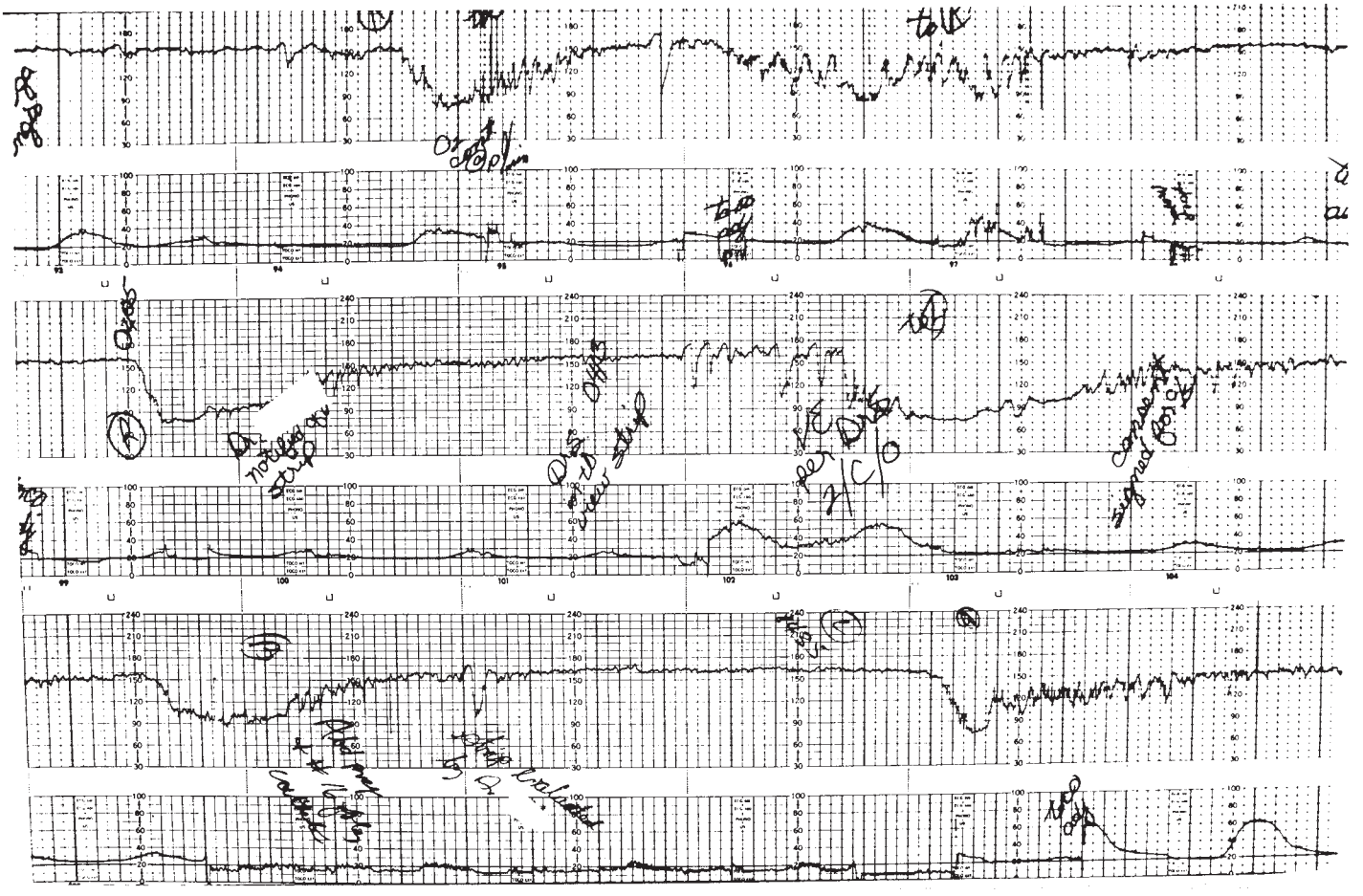

Figure 10.5. This patient was being treated expectantly in the hospital with premature rupture of membranes (PROM) at 34 weeks. She noticed mild contractions and was immediately monitored. Note the prolonged and variable decelerations that are occurring even though the patient is in early labor (irregular contractions, 2 cm dilated). With the presence of oligohydramnios secondary to PROM, the early development of such patterns is common.

Consequently, close attention needs to be directed to those at greatest risk to be complicated by amniotic fluid infection. There is currently no evidence to suggest that intrapartum intervention can alter these outcomes.

Fetal Isoimmunization

Fetal isoimmunization and subsequent hydrops may be identified in the preterm pregnancy. In an anemic fetus, tachycardia, late decelerations, and sinusoidal patterns may occur (Fig. 10.8).

Malpresentations

Premature fetuses are more likely than those at term to present in labor with breech or other malpresentations. Similarly, fetuses allowed to proceed to labor and delivery with nonvertex presentations are more likely than those with vertex presentations to have cord compression and cord prolapse (Figs. 10.9 and 10.10).

Multiple Gestation

Multiple gestations deliver prematurely at a higher rate than any other risk group. Twins are more likely to exhibit abnormal FHR patterns in labor for a number of reasons. More malpresentations, abruptions, cord prolapses, IUGR, preeclampsia, and PROM are exhibited in this group than in singleton gestations, and as explained previously, each of these risks has associated increases in fetal compromise (Fig. 10.11). In addition, monochorionic multifetal gestations with discordance of fetal growth, regardless of the cause of the discordance, exhibit more fetal compromise patterns. Therefore, the effects are additive. Twins are more likely to deliver prematurely and are more likely to have these associated complications.

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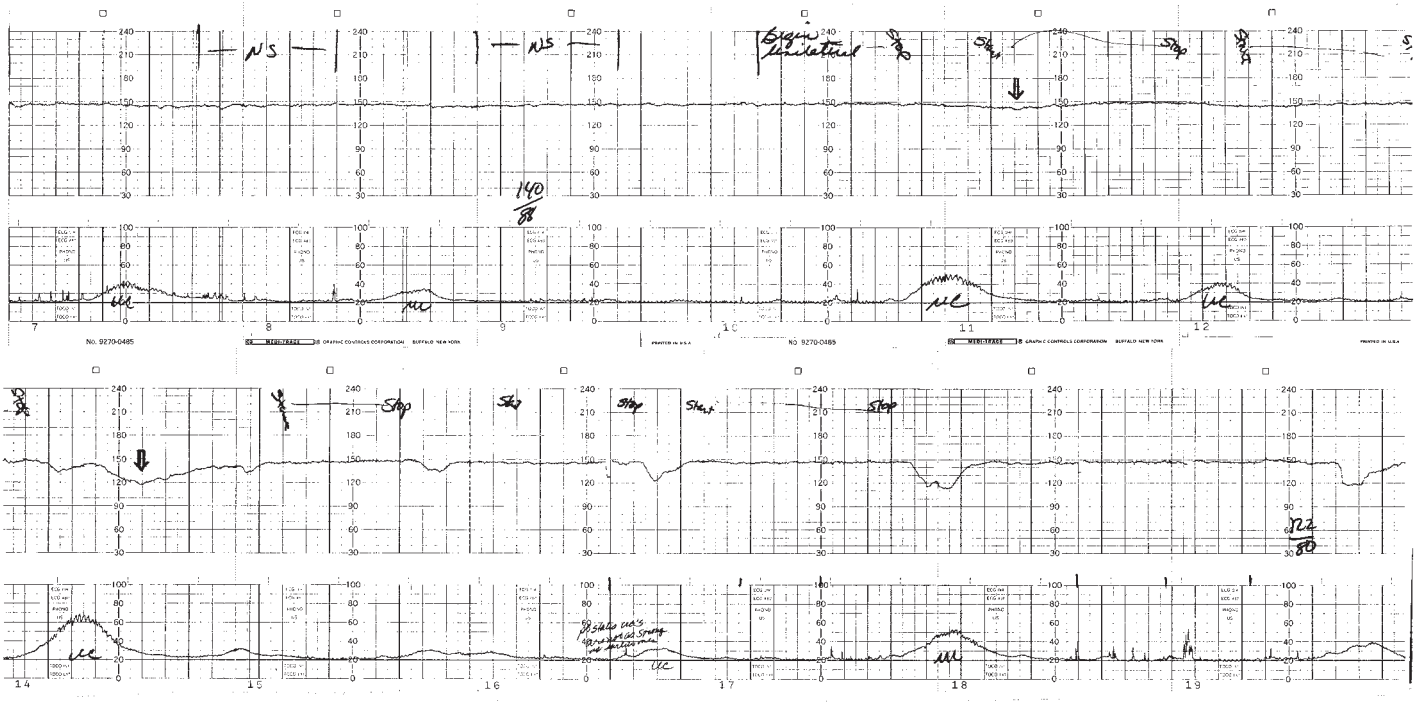
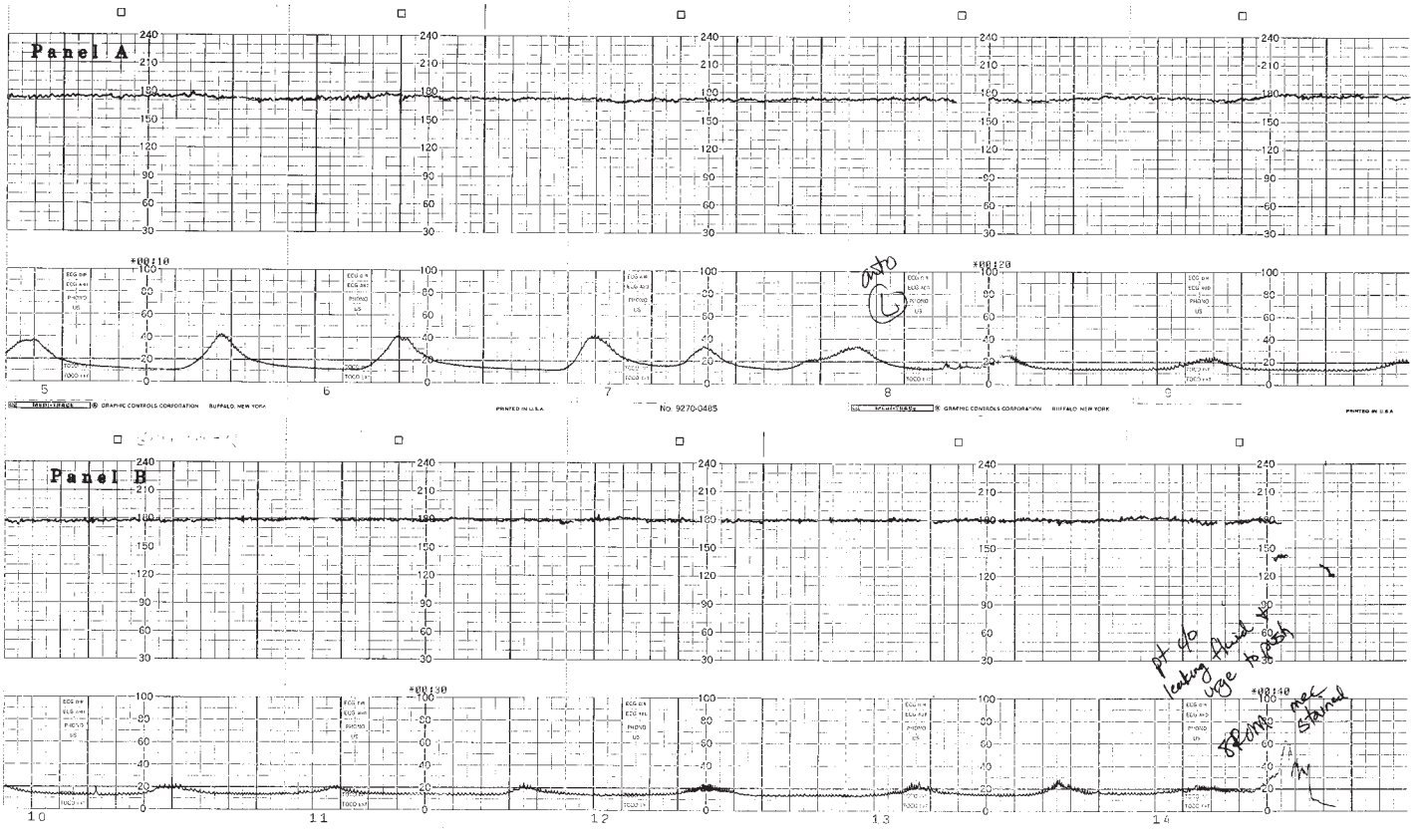


Figure 10.6. A 27-week fetus with suspected intrauterine growth retardation underwent a nonstress test, which was nonreactive. A contraction stress test was performed by nipple stimulation. Two late decelerations are seen with the stronger contractions (*arrows*) presumably secondary to the uteroplacental insufficiency, and multiple variable decelerations secondary to the oligohydramnios are also present.



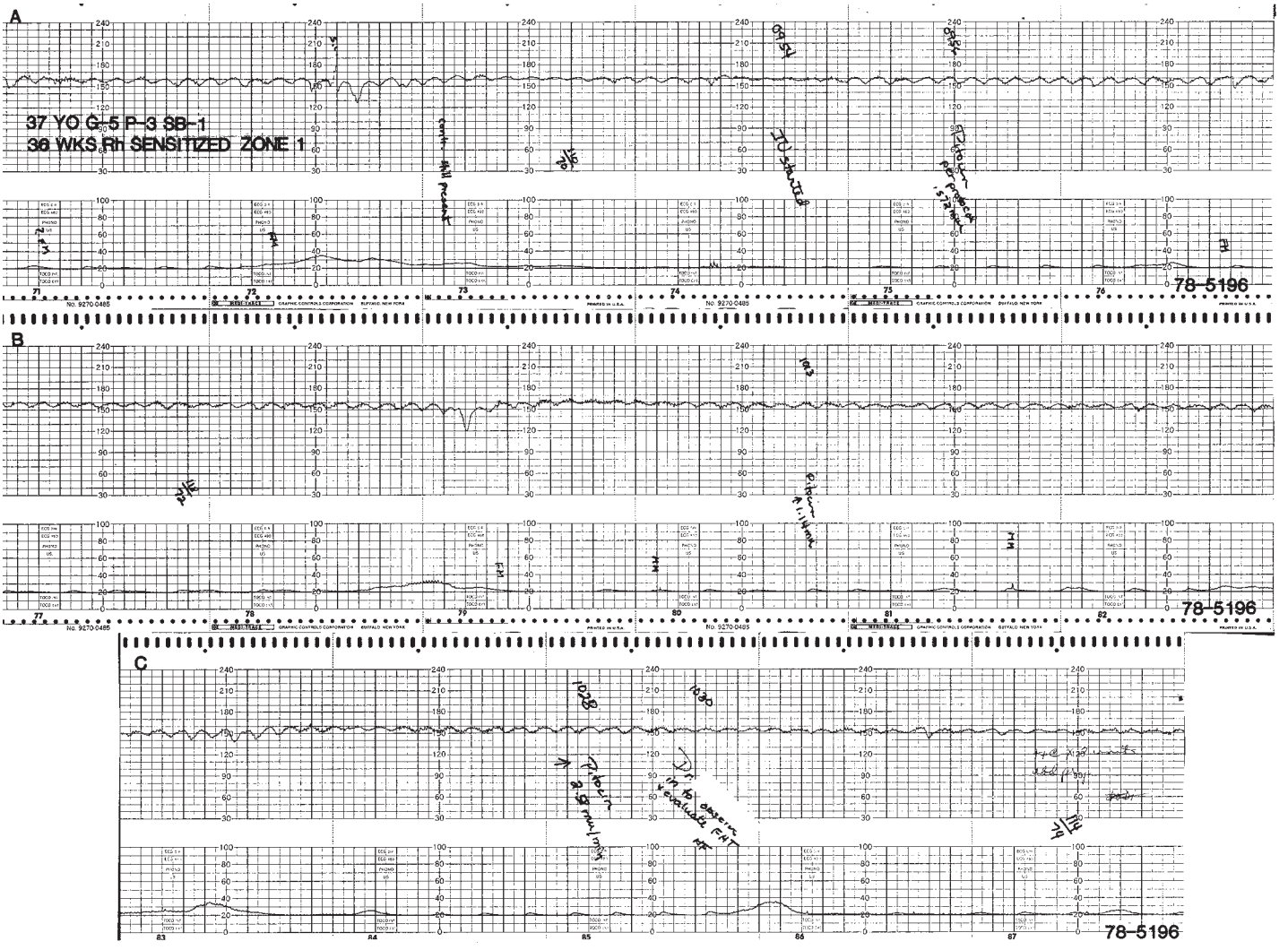


Figure 10.8. This Rh-sensitized patient at 36 weeks had an amniocentesis 2 weeks previously with a normal (Liley Zone I) delta OD450. On routine nonstress testing, this classic sinusoidal pattern was found. Note the absence of reactivity and the probable late deceleration on **panel B.** Immediate delivery resulted in a severely anemic, nonhydropic baby with a central hematocrit of 18%.

Figure 10.7. External monitoring of this patient, being evaluated at 29 weeks for possible premature labor, revealed tachycardia (170 beats per minute) and no accelerations, although no decelerations were seen. The end of **panel B** notes where spontaneous rupture of membranes occurred. Labor progressed, and in view of a breech presentation, cesarean section was performed. The patient was delivered of a depressed baby (Apgar scores 1 and 3) with normal umbilical cord pH values. Clinically, the baby was septic, and blood culture specimens revealed *Listeria monocytogenes*. The combination of depressed Apgar scores and normal cord blood gas values is characteristic of the septic baby.

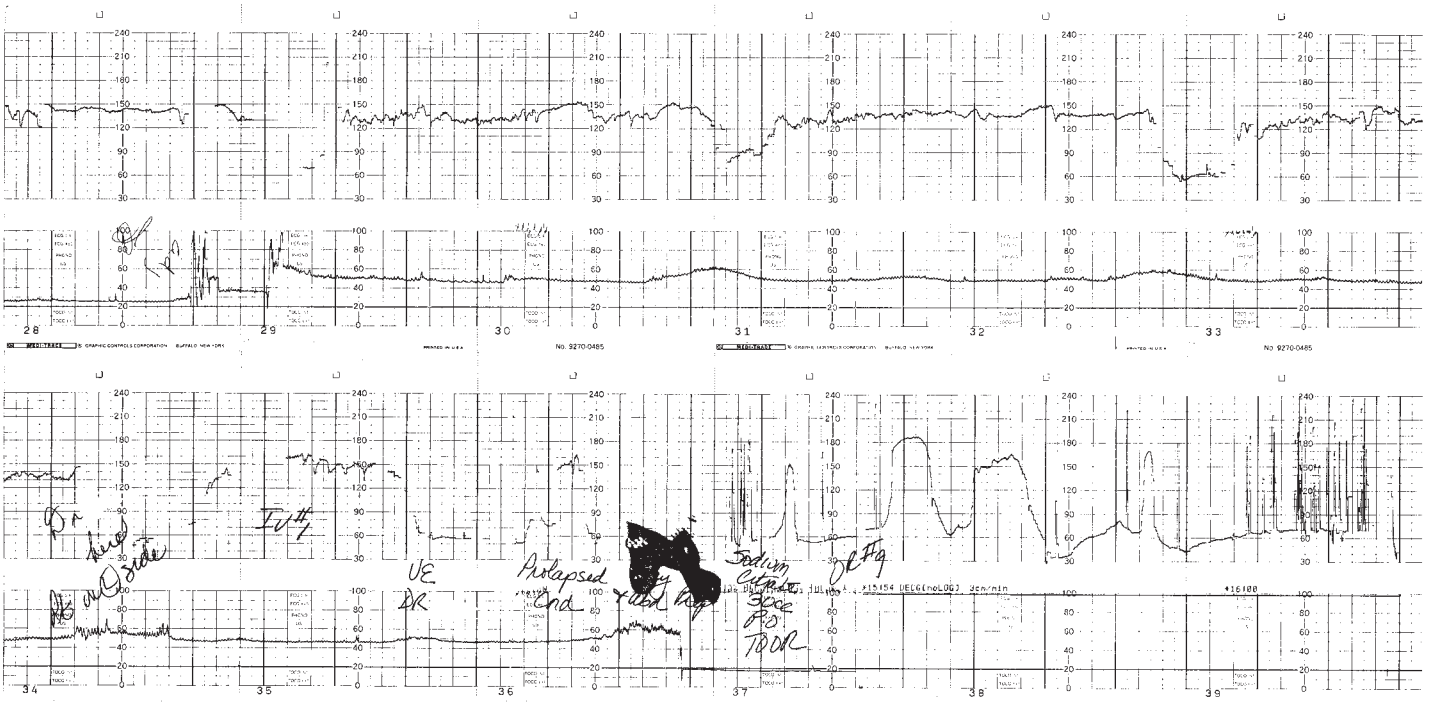


Figure 10.9. A patient with a breech presentation and premature rupture of membranes at 31 weeks was being managed expectantly: On noting some mild contractions, she was brought to the labor area for monitoring. The variable decelerations seen on the **upper panel** suggest significant umbilical cord compression. On the **lower panel** these have progressed rapidly, and pelvic examination revealed a cord prolapse. Elevation of the buttock, followed by immediate cesarean section, produced a 1,340-g baby with Apgar scores of 4 and 7 and a mild respiratory acidosis.

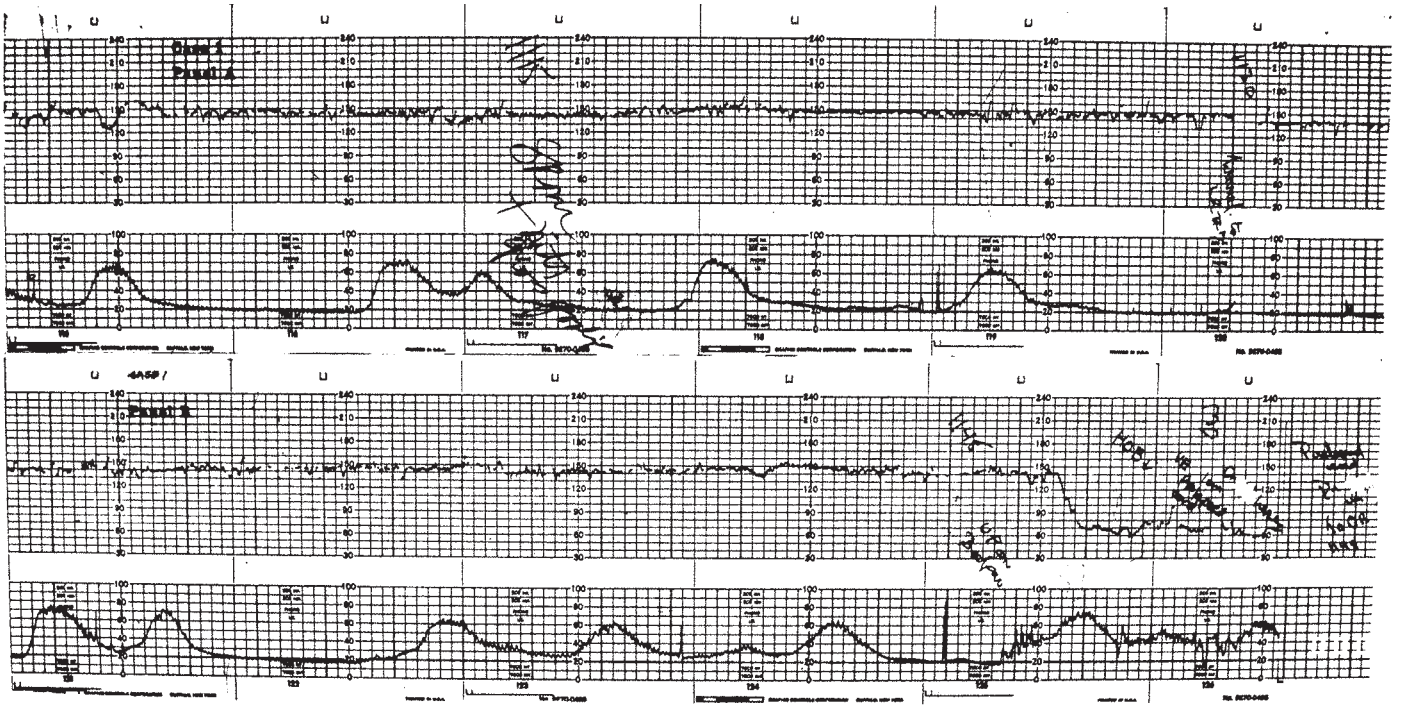


Figure 10.10. This patient is in active labor with a vertex presentation. Following a sudden gush of fluid from the vagina, there was a deceleration of the fetal heart rate. Immediate pelvic examination revealed a small loop of umbilical cord between the cervix and fetal head. Immediate cesarean section ensued with delivery of a healthy newborn.

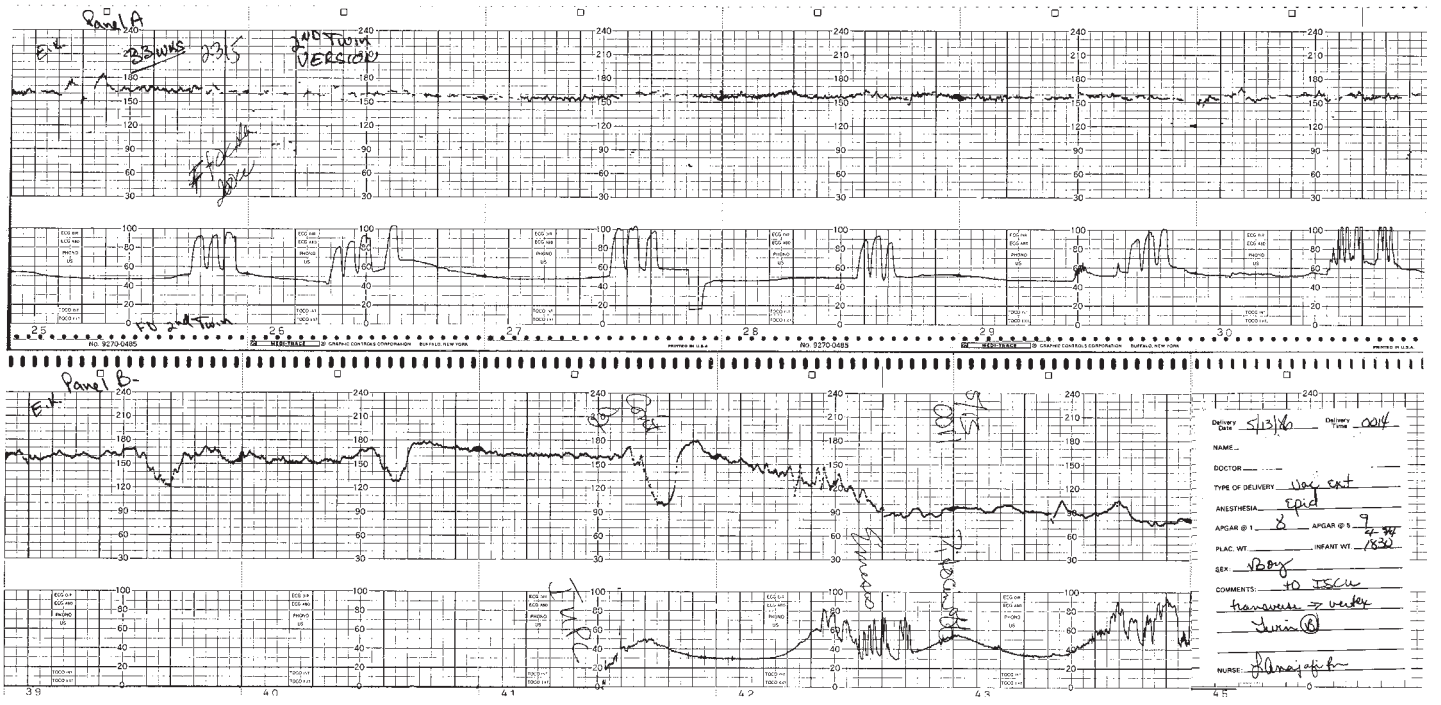


Figure 10.11. At 33 weeks, a patient with a twin gestation presenting vertex (twin A) and breech (twin B) was admitted in advanced premature labor. Following delivery of twin A, an external cephalic version of twin B was performed. As the vertex descended into the pelvis, an amniotomy was done and an internal electrode placed (**panel B**). A prolonged deceleration developed with the vertex at +1 station, and a vacuum extraction yielded a 1,830-g baby boy with Apgar scores of 8 and 9 with the cord adjacent to the baby's head.

DIFFERENCES IN FETAL HEART RATE PATTERNS IN PRETERM PREGNANCIES

In addition to being more susceptible to the effects of hypoxia and more likely to be subjected to these insults, preterm fetuses manifest FHR patterns in response to hypoxic insults that differ significantly from those of fetuses at term.

Antepartum Fetal Heart Rate Patterns

The normal preterm FHR and FHR patterns in the antepartum period differ somewhat from those of later gestations. The average FHR will be higher (see Fig. 2.6); up to 160 beats per minute (BPM) may be normal in the preterm fetus, whereas the normal-term FHR will infrequently range above 155 BPM. Accelerations are generally of lower amplitude and less frequency in the preterm fetus (Fig. 10.12A); however, even at 24 to 26 weeks and beyond, the majority of FHRs meet the criteria for reactivity (>15 BPM of amplitude lasting at least 15 seconds) (Fig. 10.12B) (26,27). Similarly, FHR variability is somewhat less in the very premature fetus, although this has not been quantified. Sorokin et al. (28) have reported that variable decelerations commonly occur in fetuses at 20 to 30 weeks, even in the absence of contractions. These decelerations are, however, generally minimal in depth and duration, and deeper and longer decelerations appear to have a significance similar to that in term pregnancies (Fig. 10.13).

Intrapartum Fetal Heart Rate Patterns

The intrapartum differences in the normal FHR, in both baseline and periodic changes, are the same as those described previously for the antepartum period. However, there are marked differences in the appearance and progression of abnormal patterns.

During labor, variable decelerations are more common in premature gestations (11,12). These decelerations may progress rapidly from reassuring to nonreassuring patterns in early gestations (11,13,29) and are more likely to be associated with loss of FHR variability (12,14). The combination of variable decelerations and loss of variability is strongly associated with low Apgar scores and fetal acidosis at birth in the preterm fetus (Fig. 10.14) (11,13,29–31).

Decreased or absent variability occurs more commonly in the premature fetus, and the progression of normal to absent variability is more rapid (12,14). Most important, loss of variability in the early gestation is much more predictive of acidosis and depressed Apgar scores than at term (11,13,29–31). Whereas at term only approximately 20% of babies with abnormal patterns will be depressed, in gestations of <33 weeks, approximately 70% to 80% of babies with abnormal FHR patterns will be depressed and/or acidotic.

Tachycardia is also more common in the premature neonate. Westgren et al. (13) found an FHR of >160 BPM in 78% of babies under 33 weeks' gestation, as opposed to

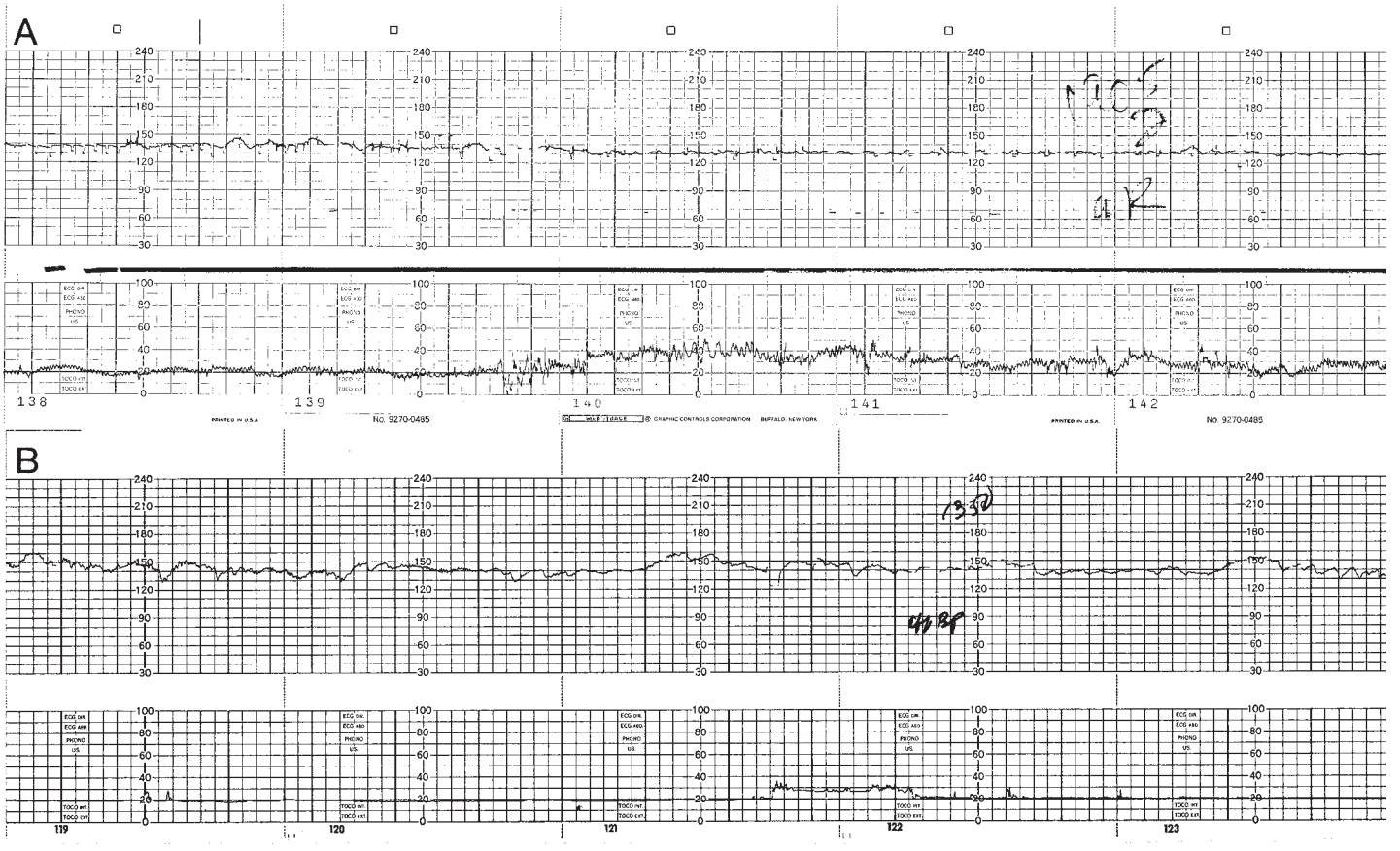


Figure 10.12. These external tracings of two different fetuses, both at 27 weeks, show the great variation in the amplitude and duration of accelerations that can be seen in healthy babies at this early gestational age. In the first part of **panel A**, three small accelerations of about 5 beats per minute (BPM) appear. Seen in **panel B** are numerous accelerations of 10 to 20 BPM above the baseline and lasting up to 1½ minutes.

20% of those beyond 33 weeks. Although maternal fever and tachycardia-inducing drugs are possible explanations in these earlier pregnancies, they do not adequately account for such a variation. Thus, the development of tachycardia and loss of variability, and their correlation with low Apgar scores and acidosis, suggest that the early fetus develops residual hypoxia and acidosis more rapidly than at term and that these FHR changes are indicative of changes in the metabolic state.

Late decelerations do not appear to be more or less common in premature pregnancies in labor *per se* (12,29); nonetheless, conditions that are more likely to provoke late decelerations—IUGR, abruptio placentae, and preeclampsia—are more frequent in premature deliveries.

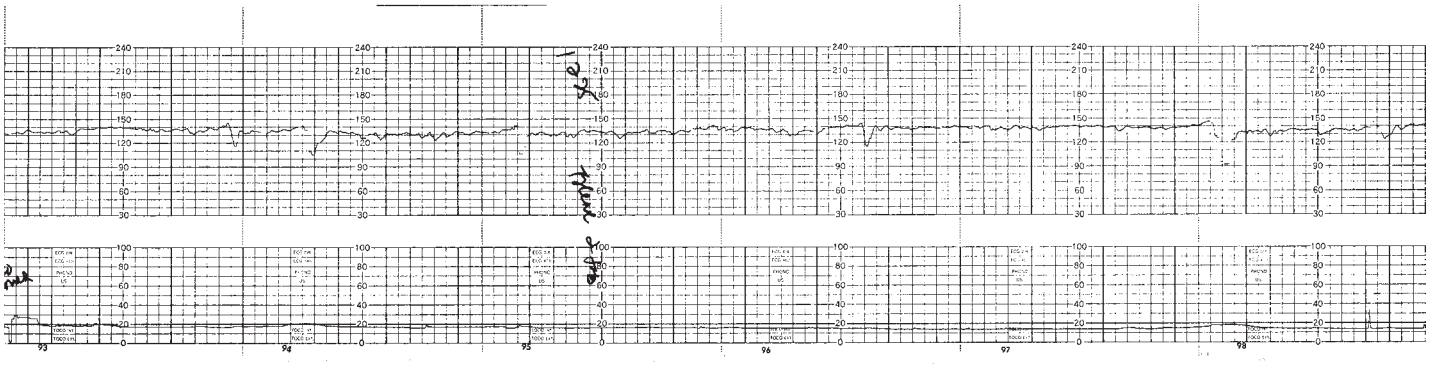


Figure 10.13. Frequent small decelerations, characteristic of the very premature fetal heart rate, are seen in this external tracing at 25 weeks.

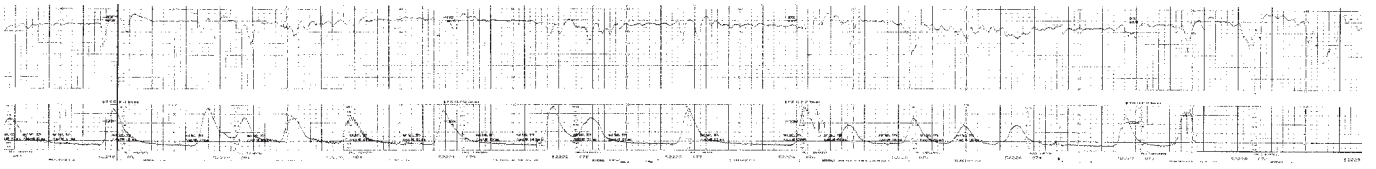


Figure 10.14. This tracing is that of a patient at 30 weeks' gestation with premature rupture of membranes for at least 2 days. She had no prenatal care, and maternal temperature was 101°F on admission. Note the significant baseline tachycardia and rapidly progressing and worsening decelerations. Emergency cesarean section resulted in delivery of a depressed 1,200-g premature infant with Apgar scores of 1 and 2 at 1 and 5 minutes, respectively. Cord gases revealed moderate to severe mixed metabolic and respiratory acidosis. The infant died secondary to overwhelming sepsis.

Prolonged decelerations appear to occur with frequencies similar to those in term pregnancies.

Finally, accelerations of the FHR are generally less frequent and of lower amplitude in premature gestations, especially those of <30 to 32 weeks. The NIH Consensus Conference (32) has suggested that the definition of an acceleration at <32 weeks be ≥10 BPM as opposed to ≥15 BPM in the fetus after 32 weeks. However, there are no controlled trials that indicate 10 × 10 accelerations in the premature fetus are indicative of pH >7.20 as with 15 × 15 BPM accelerations in the fetus >32 weeks' gestation

An additional factor needs to be considered. Because of threatened preterm delivery or pathologic conditions that are associated with preterm labor, drugs are often administered to the mother that have significant effects on the FHR. Beta-sympathomimetics such as ritodrine or terbutaline commonly increase the FHR to tachycardic levels (Fig. 10.15). This increase in FHR may itself diminish variability. The effects of other tocolytics such as magnesium sulfate, Indocin, and calcium channel blockers on FHR variability are minimal (Figs. 10.16 and 10.17A,B) (33).

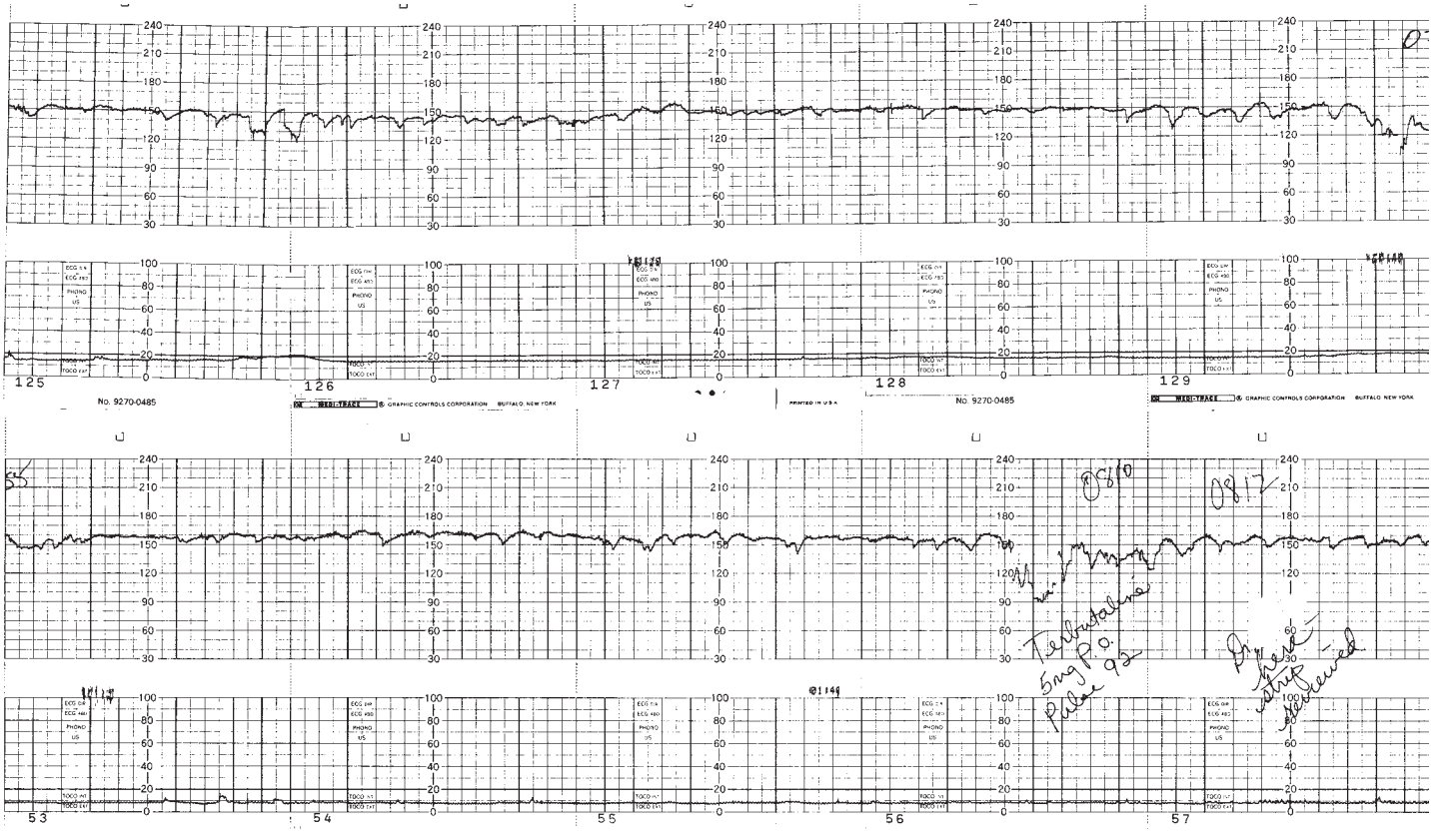


Figure 10.15. The patient was admitted at 28 weeks with premature rupture of membranes and without evidence of infection or labor. The baseline fetal heart rate (FHR) on the **upper panel** is 140 to 150 beats per minute (BPM). The patient was subsequently treated prophylactically with oral terbutaline. Five hours later (**lower panel**), the FHR rose somewhat to 155 to 160 BPM, presumably due to the passage of the beta-sympathomimetic into the fetal circulation.

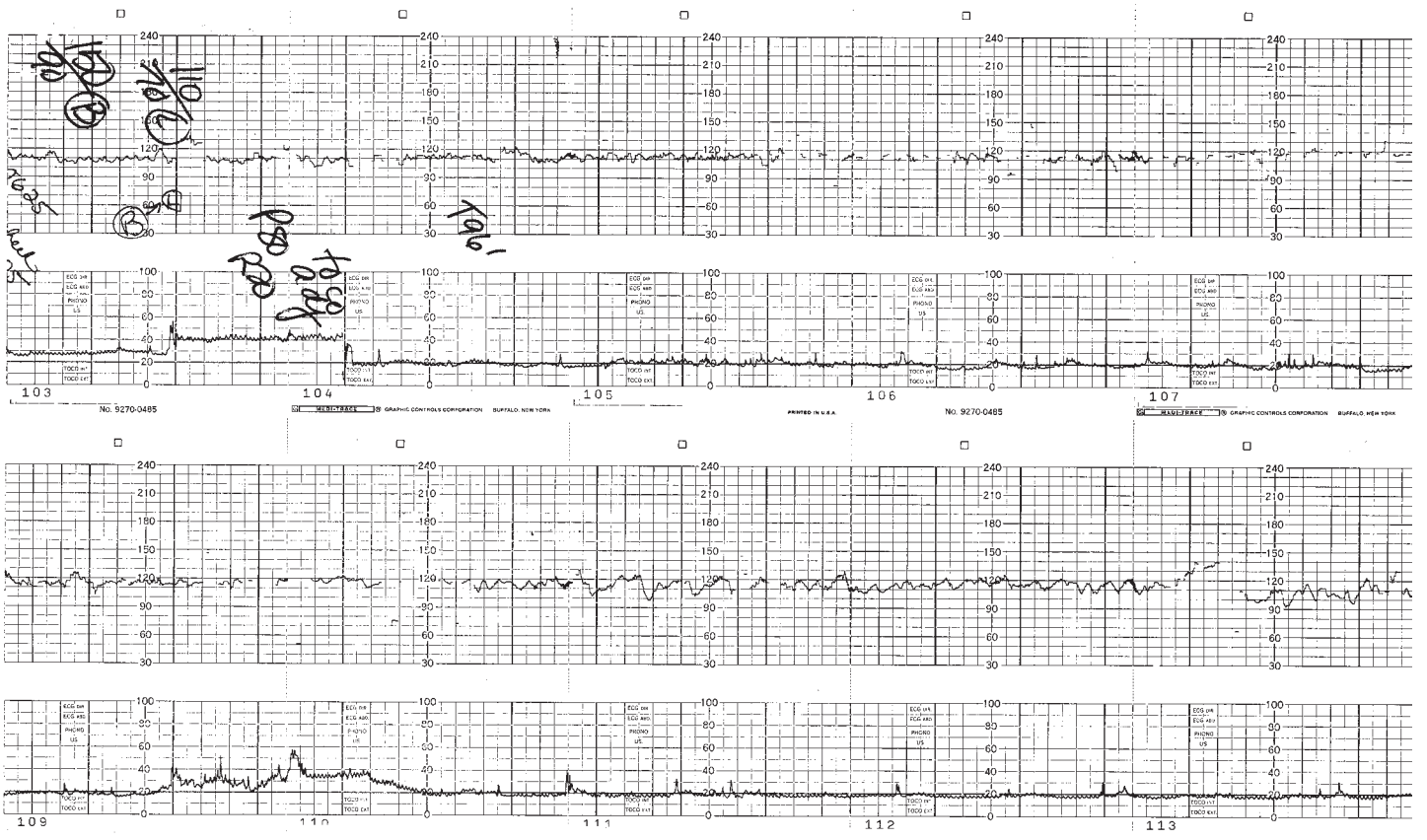


Figure 10.16. A patient in premature labor was successfully tocolyzed with intravenous $MgSO_4$. The FHR, before infusing the $MgSO_4$, was 140 beats per minute (BPM). Six hours later, the FHR baseline was showing mild bradycardia at 110 BPM. Note the accelerations on the **lower panel**, making this an overall reassuring tracing. The patient's temperature, as seen on the **upper panel**, was 35.6°C. The fetal bradycardia in this case was due to maternal (and thus fetal) hypothermia secondary to the $MgSO_4$.

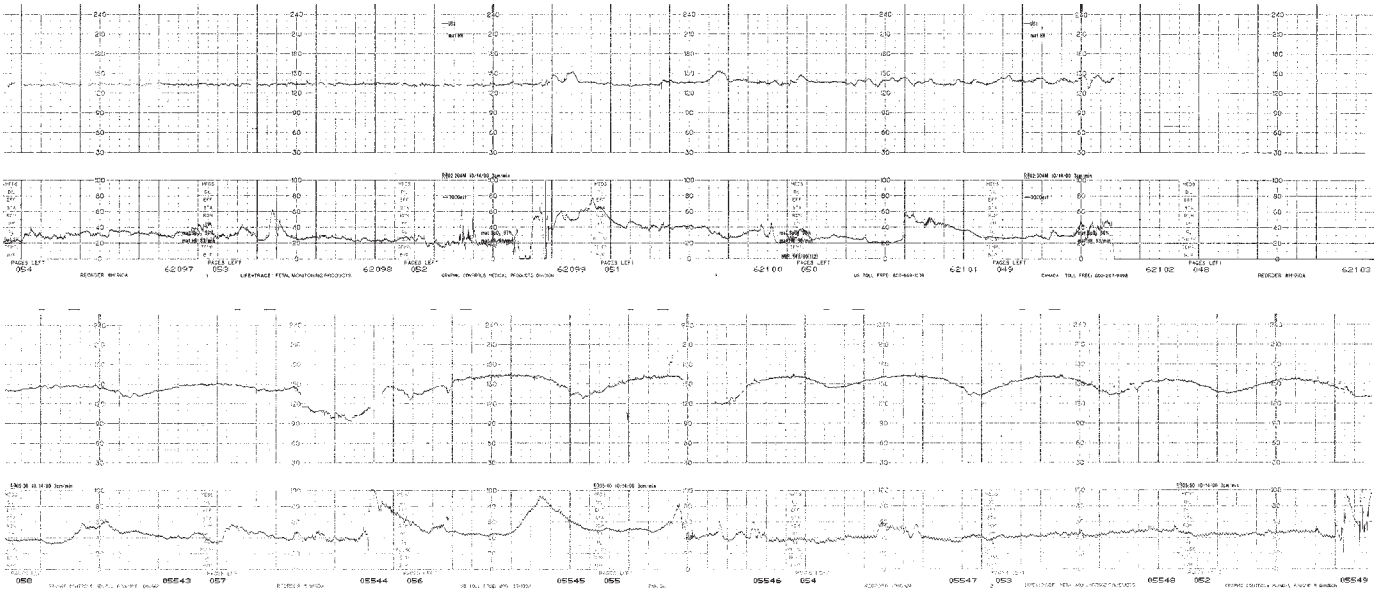


Figure 10.17. **A, B:** A patient in premature labor was admitted to the hospital for tocolysis. **Panel A** 54-49 demonstrates a fetus with a reassuring fetal heart rate (FHR) tracing. Despite tocolysis, the patient progresses in labor, and 3 hours later, the FHR tracing has become nonreassuring with late decelerations and a loss of long-term variability (**panel B**). This patient went on to deliver a viable female infant with Apgar scores of 5 and 7 at 1 minute and 5 minutes, respectively.

APGAR SCORES IN THE PRETERM NEWBORN

When the Apgar score was introduced as a tool for immediate neonatal assessment, it was described in the term fetus. Despite the initial absence of data for the utility of this method in premature infants, it has become applied to describing the condition of all newborns, regardless of birth weight or gestational age. Studies have shown that the premature newborn is more likely to have a depressed Apgar score even in the absence of acidosis (15,34). In the series of Goldenberg et al. (34), only 50% of babies at 28 weeks with a 1-minute Apgar score <7 had an umbilical arterial pH of <7.25. In the series of Bowes et al. (15), consisting of premature newborns with birth weights of <1,500 g, 58% with Apgar scores of <4 and 69% with scores of <7 at 1 minute had normal umbilical artery pH values. This is in contrast to the very good correlation between neonatal depression and low cord pH values in the term newborn. It appears that prematurity alone is an explanation of depression at birth, and the Apgar score alone may not be a reliable indicator of metabolic abnormality in the premature baby.

Should Premature Babies All Be Delivered by Cesarean Section?

Since babies delivering prematurely, especially very-low-birth-weight babies, are more often delivered due to conditions that more often are associated with hypoxia, and since these same babies are more likely to die or have serious complications associated with prematurity if hypoxia and/or acidosis occurs in labor, a logical question would be: why not deliver all these babies by cesarean section? For babies <1,000 to 1,250 g, current data would suggest that already 40% to 50% are already delivered by cesarean section, the most common reasons for which include IUGR, antepartum nonreassuring fetal status, placenta previa, malpresentation, and placental abruption. Although there are no prospective randomized controlled studies evaluating this question, a substantial body of retrospective data has addressed this issue. Although not all studies demonstrated improved survival or reduced morbidity with cesarean section (35,36), the majority of studies (37–42) have shown either improved morbidity and/or improved survival. In the largest study by Lee et al., using national birth certificate data on babies <1,500 g, those delivered by cesarean section showed improved survival up to 1,300 g. For the majority of studies such benefit was limited to babies <1,000 to 1,250 g, which is also consistent with the majority of studies demonstrating the differences in FHR patterns and adverse outcome previously described in this chapter.

SUMMARY

There appear to be substantial differences in normal FHR patterns and changes in these patterns in the preterm fetus. Tachycardia, loss of variability, and variable decelerations are more common. Compared to the term fetus, the progression of patterns where hypoxia appears to be involved occurs more frequently and more rapidly. Once patterns indicating hypoxia develop in the preterm fetus, the correlation with neonatal depression, acidosis, neonatal complications, death, and long-term sequelae is stronger than at term. Hence, there is a much greater potential in the prematurely delivering baby for improving outcome with electronic FHR monitoring and an aggressive and rapid approach to intervention. Bowes et al. (15) attempted such an approach in pregnancies delivering at <33 weeks, and their results suggested a significant improvement in outcome. The only prospective randomized trial is by Luthy et al. (43), who could not demonstrate any difference in outcome when comparing electronic FHR monitoring to auscultation. These authors, however, required documentation of fetal acidosis by scalp pH before intervening. A relatively high incidence of more severe degrees of IVH was seen in this study, and since acidosis is known to correlate with IVH, it may be that waiting for acidosis to develop in the premature fetus before intervening for fetal hypoxic patterns may be waiting too long. Based on the available information, it is apparent that fetal compromise develops rapidly in the preterm fetus and correlates with adverse outcome. Although based on current evidence routine cesarean section as the primary route of delivery at or below any given gestational age cannot be recommended, it is clear that even more than in the term gestation, the ability to perform a rapid and prompt operative delivery when the FHR suggests a high likelihood of rapidly progressing fetal hypoxia may be even more critical than in the term gestation when a trial of labor is being allowed.

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CHAPTER 11

Antepartum Fetal Monitoring

Because more than two-thirds of fetal deaths occur before the onset of labor (1), it would be natural to extend the principles of intrapartum fetal heart rate (FHR) monitoring to the antepartum period in an effort to prevent these fetal deaths. A substantial number of antepartum deaths occur in women who have risk factors for uteroplacental insufficiency (UPI) (2). Other causes include hydrops fetalis, intrauterine infections, cord accidents, congenital anomalies, and a number of unknowns. An ideal test for assessing the antepartum fetus would allow intervention before fetal death or asphyxial damage. Before the availability of such tests, the only method for attacking this problem was to prematurely deliver such fetuses based on empirical risk data, as in the method proposed by Priscilla White for managing diabetics (3). The problem with such an approach is twofold: The majority of such prematurely delivered fetuses were not in jeopardy, and the morbidity and mortality from premature intervention often exceed those of the original risk factor. It would be preferable to treat the disease process and allow the fetus to go to term; however, we have made few advances in treating UPI.

Several biochemical tests have been proposed to evaluate the antepartum fetus. Historically, these include maternal estriol, human placental lactogen, diamine oxidase, and heat-stable alkaline phosphatase. Since these biochemical tests are no longer used, they will not be discussed here. Currently available methods for antepartum fetal assessment include

1. Fetal movement (FM) counting
2. Assessment of uterine growth
3. Antepartum fetal heart rate (AFHR) testing
4. Biophysical profile (BPP) testing
5. Doppler velocimetry

PHYSIOLOGY AND PATHOPHYSIOLOGY

UPI implies inadequate delivery of nutritive and/or respiratory substances to appropriate fetal tissues. The term UPI may be applied specifically to inadequate exchange within the placenta due to decreased blood flow, decreased surface area, or increased membrane thickness. The term may also be applied more generally to problems of inadequate maternal delivery of nutrients or oxygen to the placenta, as in starvation or cyanotic cardiac disease, or to problems of inadequate fetal uptake (e.g., fetal anemia). Kubli et al. (4) suggested that UPI be divided into nutritive and respiratory components: nutritive deficiency leading to intrauterine growth retardation (IUGR) and respiratory insufficiency leading to asphyxial damage and subsequent fetal death. Parer (5) suggested that fetal nutritive function generally precedes fetal respiratory compromise (except in diabetics). Figure 11.1 is a theoretical scheme of the stages through which a fetus with declining placental function might pass. The rapidity with which this occurs may vary, being gradual in such cases as chronic hypertension or happening very suddenly as in abruption. Other conditions, perhaps including diabetes, might bypass the stage of nutritive insufficiency completely.

RISK IDENTIFICATION

To apply this knowledge to patient treatment, one must first identify the patients at risk who need evaluation. This risk identification must include data from the patient's history, physical examination, ongoing patient assessment (including uterine growth and blood pressure), and laboratory data. Those conditions that place the patient at risk for UPI are listed in Table 11.1. In addition, some obstetric/fetal conditions apparently unrelated to maternal disease may also be associated with UPI. The most common reasons for AFHR testing are postdate pregnancy, hypertension, diabetes, clinical IUGR, and the history of a stillbirth. However, distribution or indications vary depending upon the reported testing protocol (Table 11.2) (6–8). Similarly, the rate of abnormal test result varies depending on the primary testing modality used (Table 11.3).

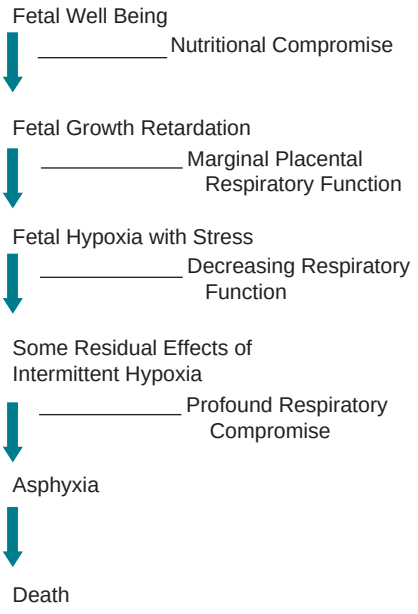

Figure 11.1. Theoretical scheme of fetal deterioration with progressive uteroplacental insufficiency.

TABLE 11.1 Conditions placing the fetus at risk for UPI

Preeclampsia
Chronic hypertension
Collagen vascular disease
Diabetes mellitus
Renal disease
Fetal or maternal anemia
Blood group sensitization
Hyperthyroidism
Thrombophilia
Cyanotic heart disease
Postdate pregnancy
Fetal growth restriction

UPI, uteroplacental insufficiency.

TABLE 11.2 Indications for antepartum testing by primary surveillance test

Indication	CST (%)	MBPP (%)	BPP (%)
Postdate	39	44	12
Hypertension	31	8	18
Diabetes	—	—	—
Gestational	5	6	7
Insulin dependent	10	—	2
Intrauterine growth restriction	9	24	21
Previous stillbirth	4	2	4
Other	5	6	38
Totals are >100% because some patients had more than one testing indication.			

BPP, biophysical profile; CST, contraction stress test; MBPP, modified biophysical profile.
From Freeman R, Anderson G, Dorchester W: A prospective multiinstitutional study monitoring. I. Risk of perinatal mortality according to antepartum fetal heart rate test results. *Am J Obstet Gynecol* 143:771, 1982; Manning F, Morrison I, Lange I, et al.: Fetal assessment based on fetal biophysical profile scoring: experience in 12,620 referred high-risk pregnancies. *Am J Obstet Gynecol* 151:343, 1985; Nageotte M, Towers C, Asrat T, et al.: Perinatal outcome with the modified biophysical profile. *Am J Obstet Gynecol* 170:1672, 1994, with permission.

FETAL MOVEMENT COUNTING

Long before electronic monitoring devices were available, clinicians recognized that maternal perception of a decrease in FMs may be a sign of impending fetal death. In a prospectively randomized antepartum fetal surveillance study from Denmark, Neldam (9) had over 1,000 patients followed by a FM counting protocol. In those women randomized to the protocol of daily monitoring of FM, there was a significant reduction in the relative risk of stillbirth to 0.25 (0.07 to 0.88) and of avoidable stillbirth to 0.27 (0.08 to 0.93). This method of fetal surveillance costs nothing and, when done in a systematic fashion, especially in low-risk populations, may contribute significantly to the detection of otherwise unsuspected fetal jeopardy.

Sadovsky (10) has used FM monitoring in developing a systematized approach to the assessment of fetal well-being. If there are more than three movements in 30 minutes, the fetus is considered to be in good condition. Less than three movements in 30 minutes is either indicative of a fetal sleep state or reason for concern, and further counting should continue. We instruct our patients to count for another 30 minutes, and if there are still less than three movements in the second counting period, we ask the patient to come to the hospital for a nonstress test (NST). If the NST is nonreactive, subsequent management is according to antepartum FHR testing protocols. If the NST is reactive (which is usually the case), the patient is reassured and asked to continue her daily counting schedule. At the time of the NST, the patient

TABLE 11.3 Distribution of test results by primary surveillance test

Result	CST (%)	MBPP (%)	BPP (%)
Negative	67	92	97
Equivocal	23	8	2
Positive	10	3[a]	1

[a]The positive test results for patients receiving MBPP were all inpatients receiving backup testing for an equivocal MBPP.
BPP, biophysical profile; CST, contraction stress test; MBPP, modified biophysical profile.
From Freeman R, Anderson G, Dorchester W: A prospective multiinstitutional study monitoring. I. Risk of perinatal mortality according to antepartum fetal heart rate test results. *Am J Obstet Gynecol* 143:771, 1982; Manning F, Morrison I, Lange I, et al.: Fetal assessment based on fetal biophysical profile scoring: experience in 12,620 referred high-risk pregnancies. *Am J Obstet Gynecol* 151:343, 1985; Nageotte M, Towers C, Asrat T, et al.: Perinatal outcome with the modified biophysical profile. *Am J Obstet Gynecol* 170:1672, 1994, with permission.

is usually taught how to count movement, because the reason for her perceived decreased count is often her nonrecognition of movement that is there. When simultaneous real-time ultrasound scanning has been done with patients asked to note the perceived movements, more movements are observed by ultrasound than are perceived by the patient (11). Most patients will feel three movements in just a few minutes, so very little time is actually required for the patient. A FM count that drops below three in 12 hours or that ceases for 12 hours is termed the "movement alarm signal" by Sadovsky, which correlates with impending fetal death (12–14). Moore and Piacquadio (15) performed a pilot study in which all patients were instructed to monitor the elapsed time it took every day from 28 weeks to register ten FMs. An NST was performed if 2 hours elapsed without 10 movements. They report a fourfold reduction in fetal mortality associated with complaints of decreased FM using this simple protocol. However, while awareness of FMs correlates with improved fetal and neonatal outcomes, the ability to identify a specific quantitative alarm for decreased FMs has not been successful (16). As an added point, the patient presenting with a complaint of absent or markedly decreased FM who has a nonreactive NST should be considered to be at increased risk for acute fetal-maternal hemorrhage (see Chapter 12).

ASSESSMENT OF UTERINE GROWTH

During the third trimester, assessment of uterine growth should be done on all patients at the time of their routine prenatal visits. As a general rule, the fundal height in centimeters as measured with a tape measure will equal the weeks of gestation. There are several things that may negate this relationship, including maternal obesity, multiple gestation, polyhydramnios, abnormal fetal lie, oligohydramnios, low fetal station, and fetal growth retardation. Except for maternal obesity and myomas, the other causes are all things about which the clinician should be interested. Specifically, abnormalities in the amniotic fluid volume may lead to the diagnosis of a fetal malformation or IUGR. Thus, abnormalities in the gross uterine size or abnormal growth rates of the fundal height should lead to further investigation, specifically sonography and/or FHR testing. Unfortunately, the accuracy of clinical assessment leaves something to be desired. The diagnosis of IUGR by clinical estimates is a poor predictor of a subsequent growth retarded neonate. Generally speaking, whenever the uterine size is significantly larger than gestational age, the patient should be advised to have a sonogram. Likewise, if the uterine size is significantly below gestational age or if there is a lack of uterine growth or a decreased growth rate, the patient should be evaluated by serial sonography. If the findings suggest UPI, some form of fetal surveillance should be performed.

WHEN TO BEGIN TESTING

Indications for testing and the gestational age for beginning testing are listed in Table 11.4. Many factors go into the decision as to when to begin testing (17–23). Single factors with minimal to moderate increased risk for antepartum death nearly all warrant surveillance starting at about 32 weeks (e.g., chronic hypertension, type I diabetes, previous stillbirth) (20,22). The greatest maternal risk factors are chronic hypertension with superimposed preeclampsia and the more severe White's classes of diabetes mellitus (D, F, and R). In diabetics with hypertension, proteinuria, IUGR, or proliferative retinopathy, the risk of fetal deterioration before 34 weeks is high, and fetal testing may begin as early as 26 weeks (21). In the patient without diabetes or hypertension, when the clinical diagnosis of IUGR is suspected by ultrasound, fetal testing should begin in some cases as early as it is reasonable to expect a chance of neonatal viability (about 26 weeks). As a general rule, AFHR testing should not begin until estimated fetal maturity is sufficient to expect a reasonable chance of survival should intervention (delivery) be necessary. This is especially difficult with twin gestations because intervention for a compromised twin may adversely affect an apparently normal twin before maturity. Testing for patients exceeding their due date is a common indication. In most cases, testing in some form should begin between 41 and 42 weeks' gestation (23).

TABLE 11.4 Indications and gestational age for AFHR testing

Indication	Gestational age to start testing (wk)
Diabetes	
Class A-1	40
Class A-2	32
Class B, C, D	32
Class F, R	26–30
Preeclampsia	At diagnosis >25–26
Chronic hypertension	32
Severe Rh disease	At diagnosis >26
Previous stillbirth	Gestational age of previous stillbirth
IUGR at diagnosis	26
Post date pregnancy	41–42
Cyanotic heart disease	32
Hyperthyroidism	32
Oligohydramnios	At diagnosis >25–26

AFHR, antepartum fetal heart rate; IUGR, intrauterine growth retardation; Rh, rhesus isoimmunization.
From references Froen JF, Heazell AEP, Tveit JVH, et al.: Fetal movement assessment. *Semin Perinatol* 32:243–246, 2008; Freeman RK: The use of the oxytocin challenge test for antepartum clinical evaluation of uteroplacental respiratory function. *Am J Obstet Gynecol* 121:481, 1975; Gabbe SG, Freeman RK, Mestman JH: Management and outcome of the class "A" diabetic. *Am J Obstet Gynecol* 127:465, 1977; Gabbe SG, Mestman JH, Freeman RK, et al.: Management and outcome of diabetes mellitus, classes *Br Am J Obstet Gynecol* 129(7):723, 1977; Pircon RN, Lagrew DC, Towers CV, et al.: Antepartum testing in the hypertensive patient: when to begin? *Am J Obstet Gynecol* 164:1563, 1991; Lagrew DC, Pircon RA, Towers CV, et al.: Antepartum surveillance in the diabetic: when to start? *Am J Obstet Gynecol* 168:1820, 1993; Weeks JW, Asrat T, Morgan MA, et al.: Antepartum surveillance for a history of stillbirth: when to begin? *Am J Obstet Gynecol* 172:486, 1995.

PERFORMING THE ANTEPARTUM TEST

Experience is the key to obtaining quality tests in the shortest time. It is most desirable for nurses or technicians to specialize in antepartum testing. When testing is done in a hospital setting, an area separate from the labor and delivery suite is preferable to resist the temptation to have nurses cross-cover the antepartum testing area and not be able to devote enough attention to the patient being tested. Alternatively, provision of testing either in a hospital or in an office setting should be done in a quiet and stress-free environment.

The patient is placed in the semi-Fowler's position to avoid supine hypotension syndrome. Baseline blood pressure is recorded and repeated throughout the test, again to be sure that supine hypotension does not occur, as this may be a cause of decreased uteroplacental perfusion and false-positive test results. Baseline contractions and FHR are recorded for approximately 20 minutes. The baseline heart rate and reactivity are noted, as is the background uterine activity. Following this evaluation, if results are not reassuring, continued monitoring of FHR may be necessary. Alternatively, initiation of a contraction stress test (CST) or use of real-time ultrasound to measure amniotic fluid volume and assess various fetal parameters should ensue.

WHICH TEST TO USE

In the previous editions of this text, we have presented the CST as the "gold standard" of antepartum fetal surveillance. It has been reported that using the CST as the primary means of fetal surveillance results in a very impressive and remarkably low incidence of unexpected fetal death within 7 days of a negative test result (24). However, significant problems accompany the use of contraction stress testing as primary fetal surveillance. These include the increase in time, cost, and inconvenience of the CST compared with other forms of fetal testing. In addition, the high frequency of equivocal test results and lack of consensus over test interpretation makes the CST an often impractical if not inappropriate choice of testing for many caregivers. As a result, the CST has essentially been replaced by either the NST, the BPP, or the modified biophysical profile (MBPP) for primary antepartum testing. In our centers, we now use the MBPP test as the primary means of antepartum fetal testing with the exception being the continued use of a CST alternating with a MBPP schedule every 3 to 4 days for insulin-requiring diabetics. Other forms of fetal assessment include the use of ultrasound and Doppler velocimetry of various fetal vessels.

What follows is a description of the various testing options available with particular emphasis on the evolution and reported application of the CST, NST, BPP, and the MBPP. There continues to be argument and controversy regarding the relative value and efficacy of these various types of fetal assessment despite their well-established place in the clinical practice of obstetrics (25).

CONTRACTION STRESS TEST

It can be surmised that, given a condition of borderline fetal oxygenation, a test that further stresses the fetus in terms of oxygen deprivation might produce some biophysical sign of such compromise, and that these data could be of prognostic importance. Early tests that attempted to accomplish this utilized the maternal exercise stress test and breathing gas mixtures with decreased oxygen concentrations (26). Animal data suggest that uterine contractions producing an intraamniotic pressure in excess of approximately 30 mm Hg create an intramyometrial pressure that exceeds mean intraarterial pressure, thereby temporarily halting uterine blood flow (27). A well-oxygenated fetus tolerates this limited period of intervillous stasis well; however, a hypoxic fetus will manifest late decelerations. It was therefore suggested that by inducing such contractions in the antepartum period, one might be able to detect the compromised fetus before death (and possible damage) occurred. In 1966, Hammacher (28) studied 207 pregnancies in the antepartum period and found that late decelerations correlated with lower Apgar scores at subsequent delivery and that 17 of 23 that resulted in stillbirth had manifested such late decelerations with spontaneous contractions in the antepartum period. Subsequently, Pose and Escarcena (29), Kubli et al. (4), and Spurrett (30) found late decelerations in the antepartum period to correlate with stillbirth, IUGR, and low Apgar scores. Sanchez-Ramos et al. (31) found no fetal deaths within a week of testing when no late decelerations were seen.

The first systematic trial of stress testing in this country was performed by Ray et al. (32) in 1972. They performed a prospective blinded trial on 66 patients and defined criteria for adequate testing, frequency of testing, and results, all of which are in common use in this country today. Of the 66 patients, 15 had positive test results, and of these, 3 had fetal deaths and 6 had low Apgar scores. Furthermore, there were no deaths within a week of a negative test result. Ray et al. called the test the oxytocin challenge test (OCT). Because the test can use either spontaneously occurring contractions or contractions induced by breast stimulation, it has become known more properly as the CST.

How to Perform the Contraction Stress Test

External monitors for contraction and FHR measurement are placed on the patient. With the patient in semi-Fowler's position or left lateral tilt to minimize supine hypotension, an initial monitoring period lasting from 20 to 30 minutes is obtained to assess the FHR baseline, to identify the presence or absence of periodic changes, and to determine if there is evidence of spontaneous uterine activity. If there are three adequate spontaneously occurring contractions within a 10-minute period and the FHR recording is of sufficient quality, the test is concluded (Fig. 11.2). If the contractions are absent or of insufficient frequency, they must be stimulated. Historically, oxytocin infusion intravenously has been used to elicit contractions of the uterus. This is accomplished by beginning oxytocin through an infusion pump at a rate of 1.0 mU/minute. The infusion rate is initially doubled every 15 minutes until the appearance of contractions. Smaller increments for oxytocin increase are then used until three contractions lasting 40 to 60 seconds occur in 10 minutes. Patience and experience are valuable in obtaining an adequate CST and avoiding overstimulation of uterine activity (Fig. 11.3).

A widely used alternative to intravenous oxytocin infusion is that of breast stimulation. Oxytocin is released from the posterior pituitary following breast stimulation, and this technique has been used for both initiation of labor as well as for initiation of the CST (32–37). When used for fetal testing, breast stimulation has similar efficacy to oxytocin infusion with a shorter testing time with less expense, discomfort, and inconvenience. Breast stimulation has been

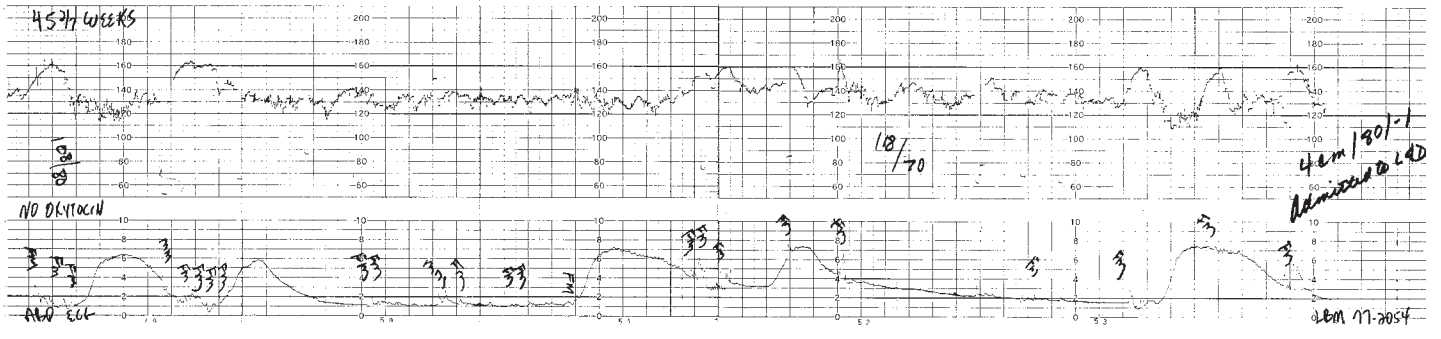

Figure 11.2. Spontaneous negative contraction stress test (no oxytocin needed). The patient is actually found to be in labor.

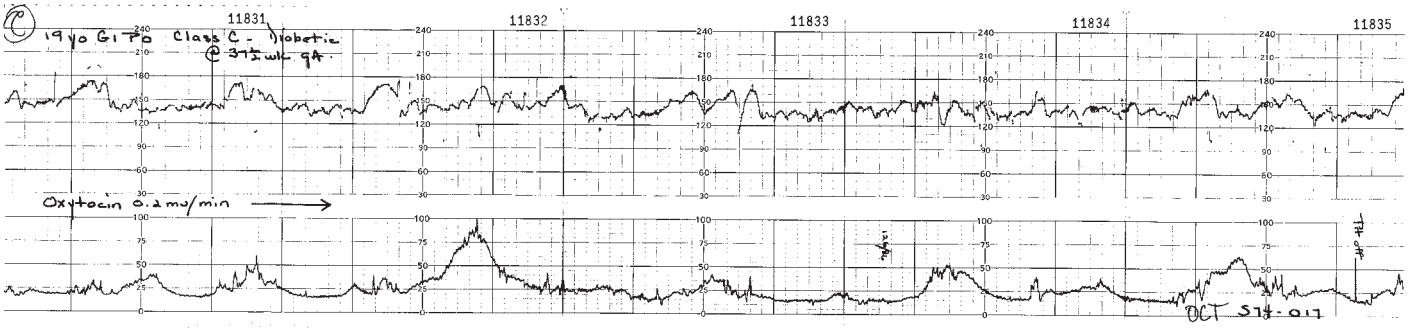

Figure 11.3. Negative contraction stress test result. Three contractions are obtained in 10 minutes, lasting more than 60 seconds each, with adequate quality fetal heart rate recording.

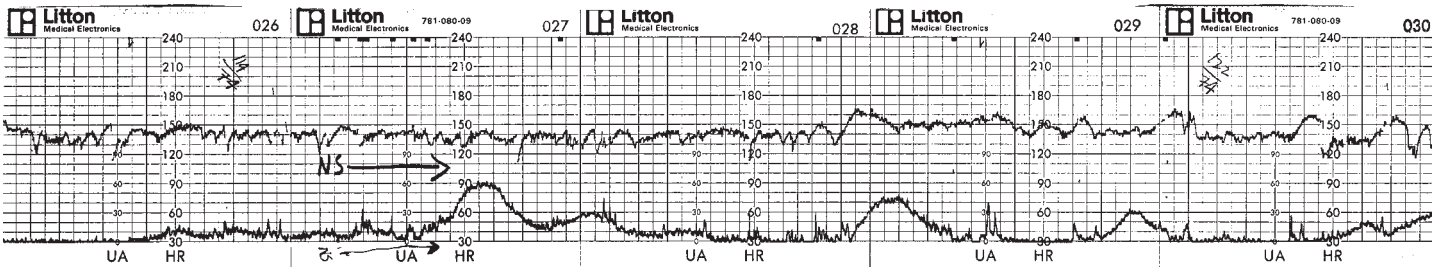

Figure 11.4. Reactive negative contraction stress test using breast stimulation.

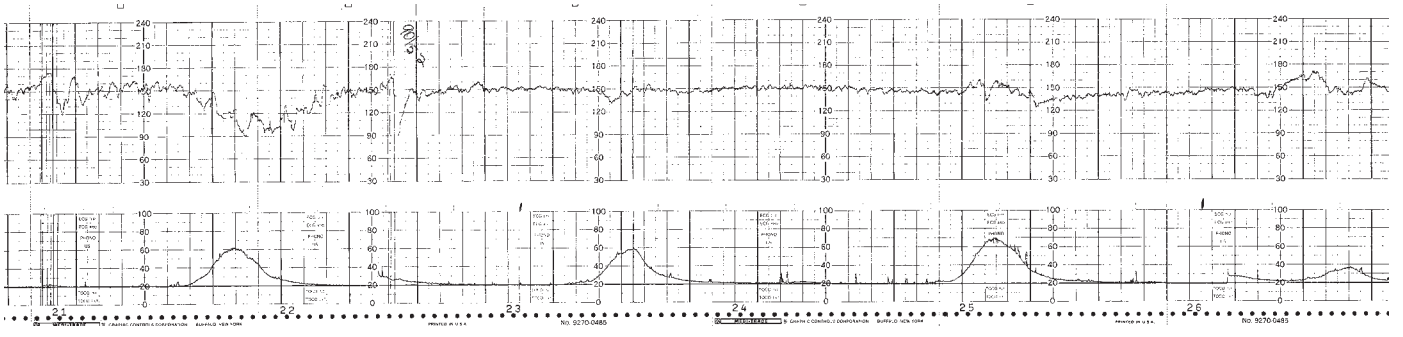

Figure 11.5. Reactive positive contraction stress test using breast stimulation.

associated with uterine tachysystole and fails to achieve adequate uterine activity in approximately 20% of tests (38–41). The test is best performed with the patient initially rolling or tugging on one nipple through her clothing until a contraction occurs. If no contraction results following 2 to 3 minutes, the patient is asked to perform bilateral stimulation following a 5-minute rest period. This cycle of stimulation is then repeated until adequate uterine activity is documented. Figure 11.4 is an example of a reactive negative CST done with nipple stimulation. Following the appearance of adequate uterine activity, oxytocin infusion or breast stimulation is stopped and the patient continued to be monitored until activity significantly diminishes or disappears (42).

If late decelerations are present with every spontaneous contraction, yet the contraction frequency is less than three in 10 minutes, initiation of further uterine activity is not indicated, and the CST result is positive.

Interpretation of the Contraction Stress Test

Negative

No late decelerations appearing anywhere on the strip, adequate contraction frequency (three in 10 minutes), and adequate FHR recording must be obtained (Figs. 11.2–11.4) for the interpretation to be negative.

Positive

In a positive CST result, late decelerations are present with the majority (greater than one-half) of contractions during the period of maximum contraction stress without excessive uterine activity (see "Hyperstimulation"). If persistent late decelerations are present before the contraction frequency is adequate, this is a positive test result and may be concluded. Figure 11.5 shows a reactive positive CST result, Figure 11.6 a minimally reactive positive CST result, and Figure 11.7 a nonreactive positive CST result.

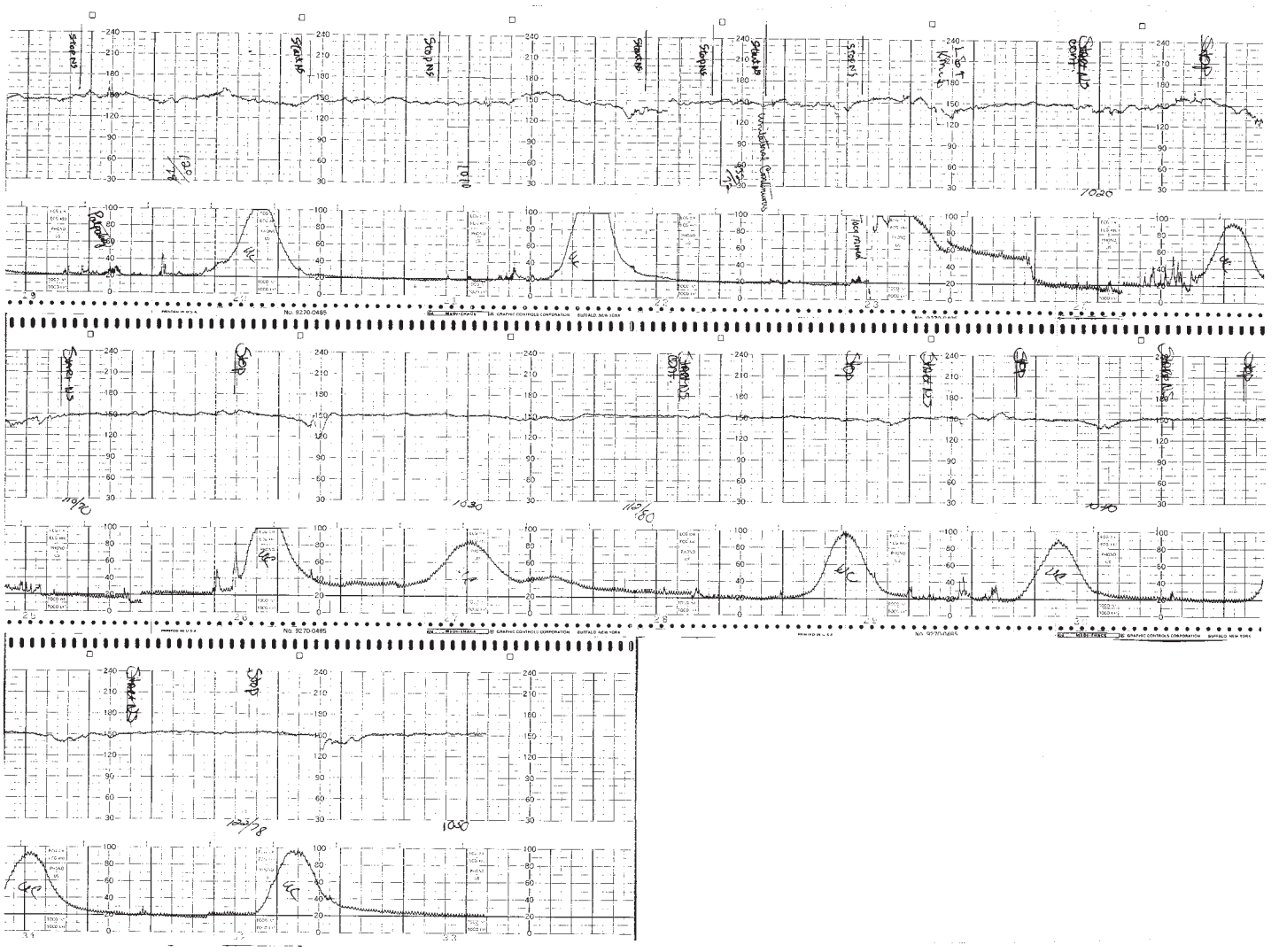

Figure 11.6. Minimally reactive positive contraction stress test using breast stimulation.

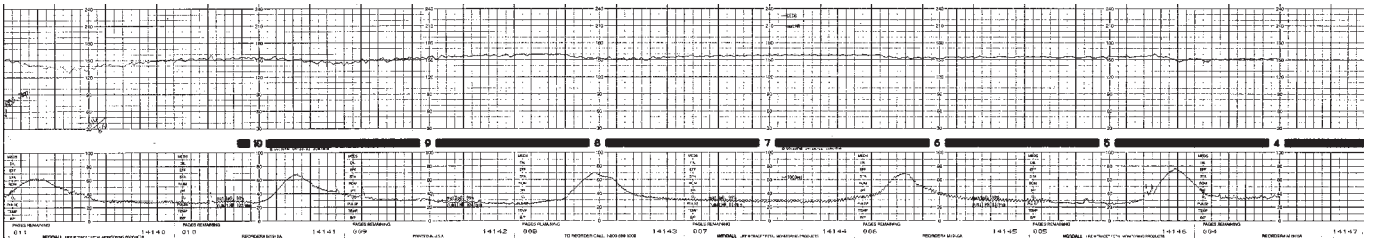

Figure 11.7. This is a nonreactive positive contraction stress test elicited with unilateral breast stimulation. The decelerations are subtle and frequently not appreciated. Note the complete absence of accelerations, which should always lead to a close evaluation of the heart rate.

Equivocal Test Results

Suspicious. A test result is considered suspicious if late decelerations are present with less than half of the contractions. It is sometimes necessary to keep a test going awhile longer to determine whether the late deceleration is persistent or only sporadic (Fig. 11.8).

Hyperstimulation. Decelerations after contractions lasting more than 90 seconds, or with contraction frequency greater than every 2 minutes, constitute hyperstimulation (Figs. 11.9 and 11.10). When such prolonged frequent contractions occur without late decelerations, it is not hyperstimulation (Fig. 11.11). Hyperstimulation

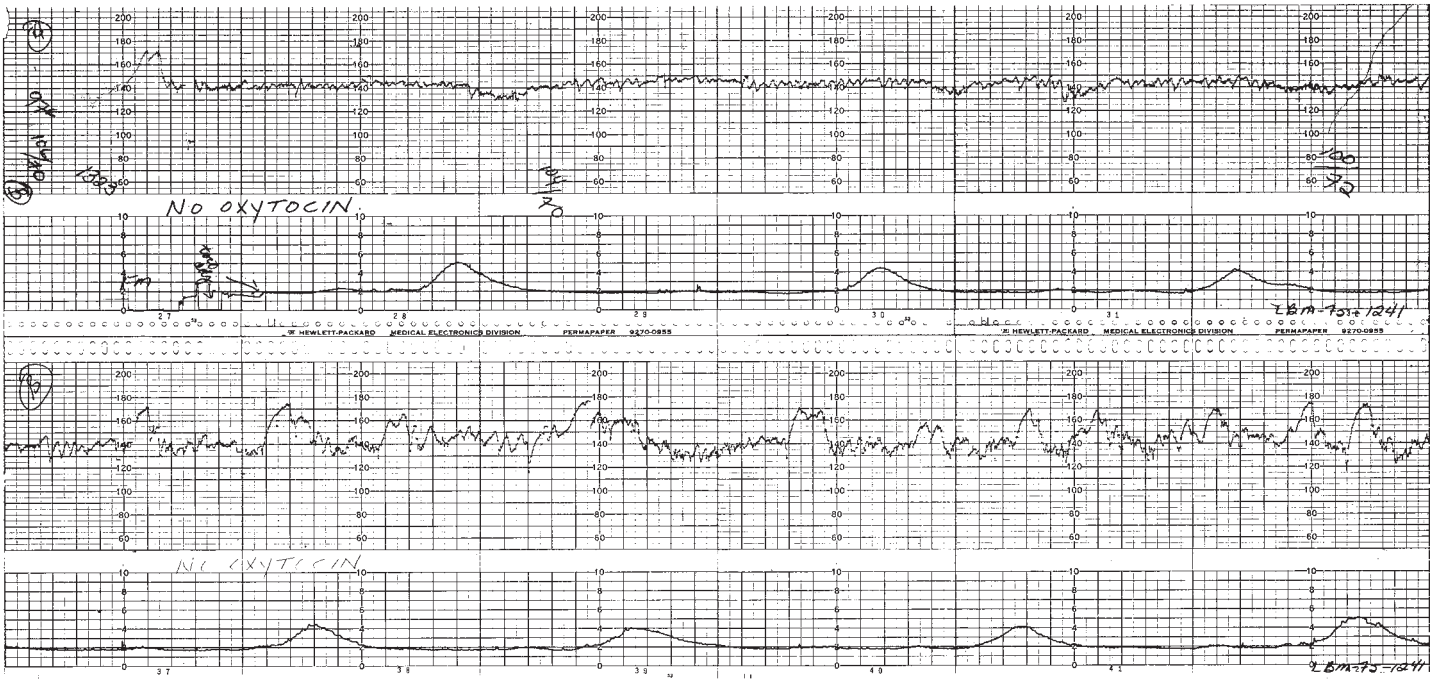

Figure 11.8. Suspicious oxytocin challenge test. Late decelerations are seen in **panel A** but disappear in **panel B**. As is often the case, the late decelerations are seen during a period of absent reactivity (no accelerations) but resolve when the fetus becomes reactive. The explanation for this is unclear.

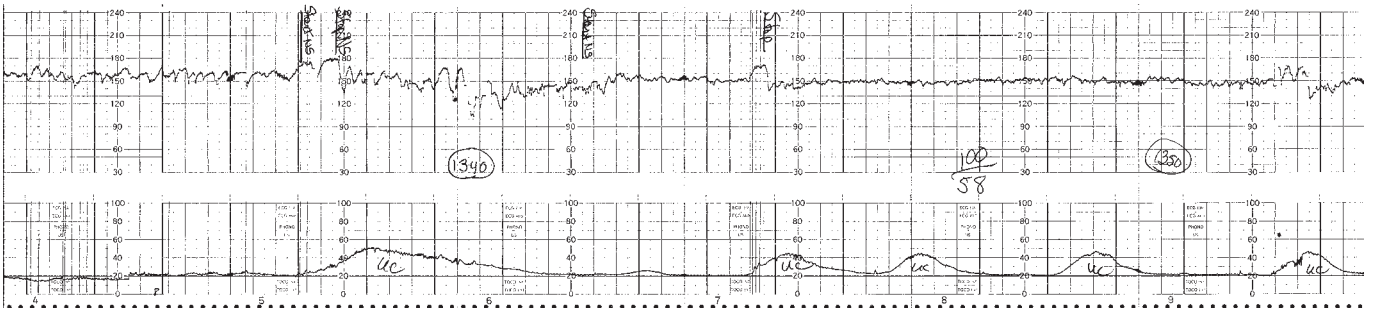

Figure 11.9. This tracing is read as equivocal due to hyperstimulation. Note the deceleration associated with a prolonged uterine contraction.

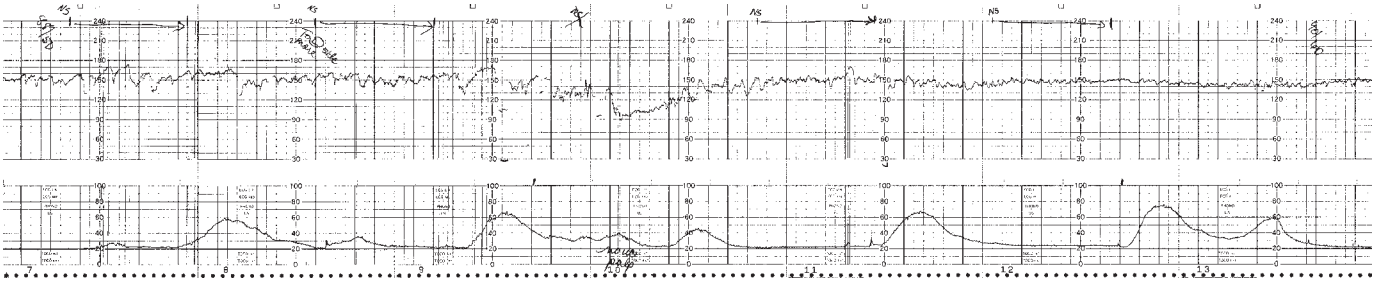

Figure 11.10. This tracing is read as equivocal due to hyperstimulation. Note the deceleration associated with frequent contractions.

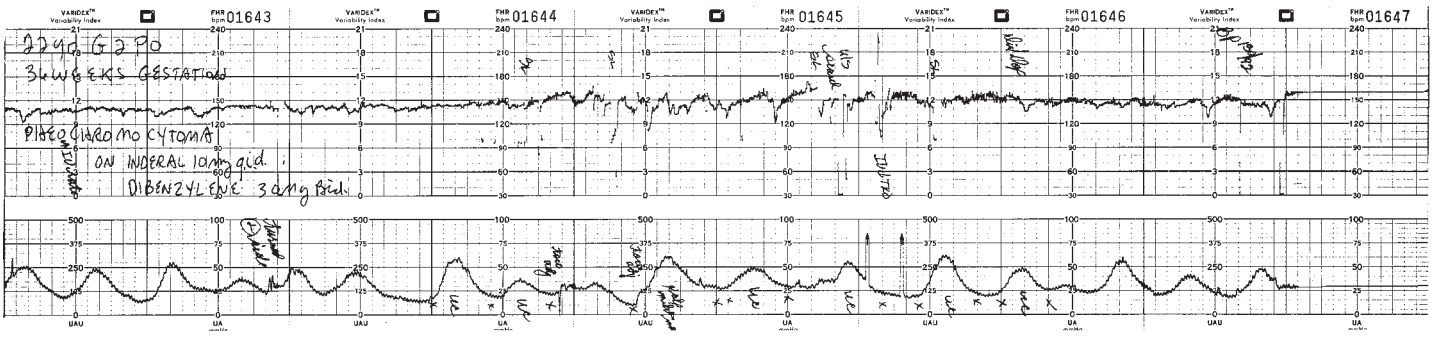

Figure 11.11. Very frequent uterine contractions are noted in this patient who was known to have a pheochromocytoma. The norepinephrine from the pheochromocytoma is thought to be responsible for this. Because no significant decelerations are noted, this tracing is read as a reactive negative spontaneous contraction stress test.

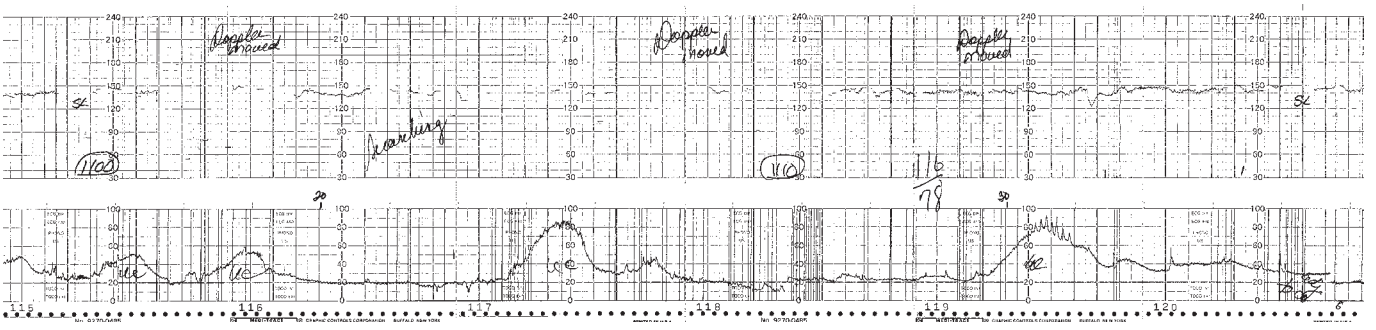

Figure 11.12. The quality of this recording is not sufficient for interpretation. It is therefore read as unsatisfactory.

may occur with either spontaneous or oxytocin-induced contractions.

Unsatisfactory. When adequate contraction frequency cannot be induced or when the FHR recording is not of sufficient quality to be sure about the presence of late decelerations, the test result is considered unsatisfactory (Fig. 11.12).

Other Patterns

When variable decelerations are seen, they are suggestive of oligohydramnios or cord entrapment (Fig. 11.13). Such test results are suspicious and usually suggest the need for a sonogram to look for oligohydramnios. Variables associated with loss of variability and blunting of decelerations have been observed by Freeman and James (43) and by Baskett and Sandy (44) and are found to be very ominous (Fig. 11.14).

The sinusoidal pattern consists of sine wave undulations of the FHR, with a cyclicity of approximately three to five per minute (Fig. 11.15). It is characterized by an absence of short-term variability with uniform long-term variability. The pattern is always nonreactive and should be present for more than a few minutes to be significant. Sinusoidal patterns fluctuate above and below the baseline and are uniform (45). Late decelerations are commonly seen in association with sinusoidal patterns. They have been reported to be associated with severe fetal anemia, as in Rh isoimmunization (46) or fetal-maternal hemorrhage (Fig. 11.16) (47). These patterns may also be seen with hypoxic fetal distress in the absence of fetal anemia. Generally, the sinusoidal pattern is ominous and has been associated with a high incidence of perinatal mortality. However, other tracings with increased long-term variability may be easily confused with sinusoidal patterns and might cause inappropriate intervention (Fig. 11.17). Although the literature is somewhat confusing on the significance of sinusoidal patterns, when the criteria described by Modanlou and Freeman (45) are used, we find the pattern to be uniformly ominous.

A very rare test result is a nonreactive negative CST. Theoretically, this should not occur if the appearance of late deceleration is an earlier finding than the loss of acceleration (48). A mistake commonly made in this situation is to miss very subtle late decelerations when the fetus may be severely hypoxic (Fig. 11.18). It is therefore important to be sure that no subtle late decelerations are present when reading a test as nonreactive negative. Grundy et al. (49) have shown that only 2 in 1,000 negative CST results are completely nonreactive. Fetuses with nonreactive negative CST results were often found in mothers taking central nervous system (CNS)–depressant drugs and in fetuses with CNS abnormalities. When excluding those with CNS abnormalities, the immediate perinatal

(Text continues on page 190)

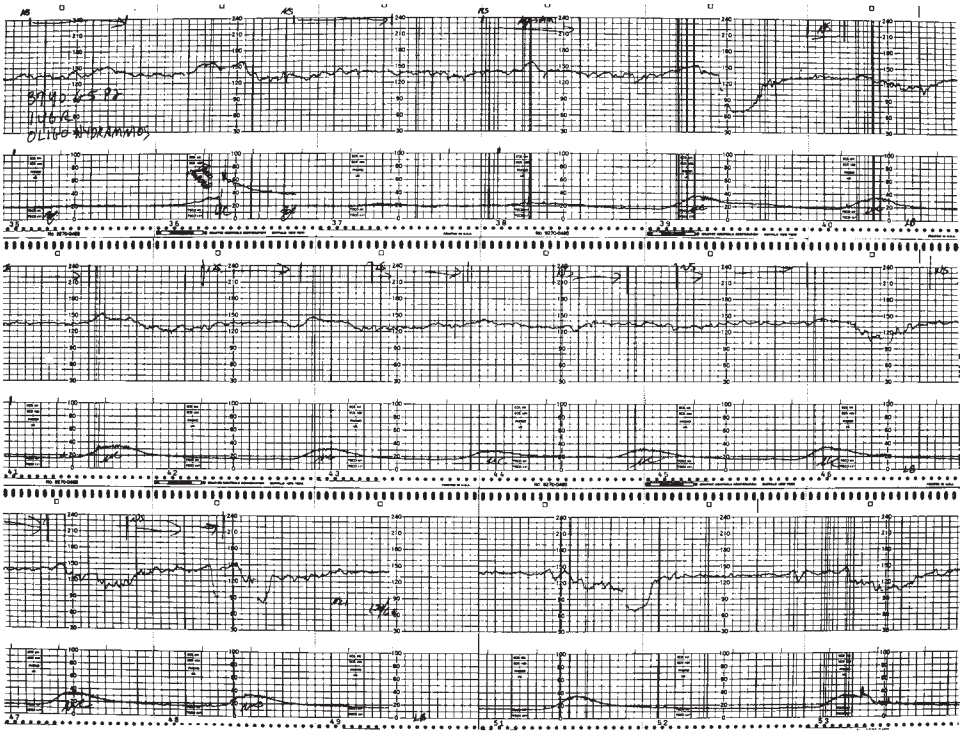

Figure 11.13. The variable decelerations noted on this contraction stress test are believed to be due to umbilical cord compression in this patient with known oligohydramnios.

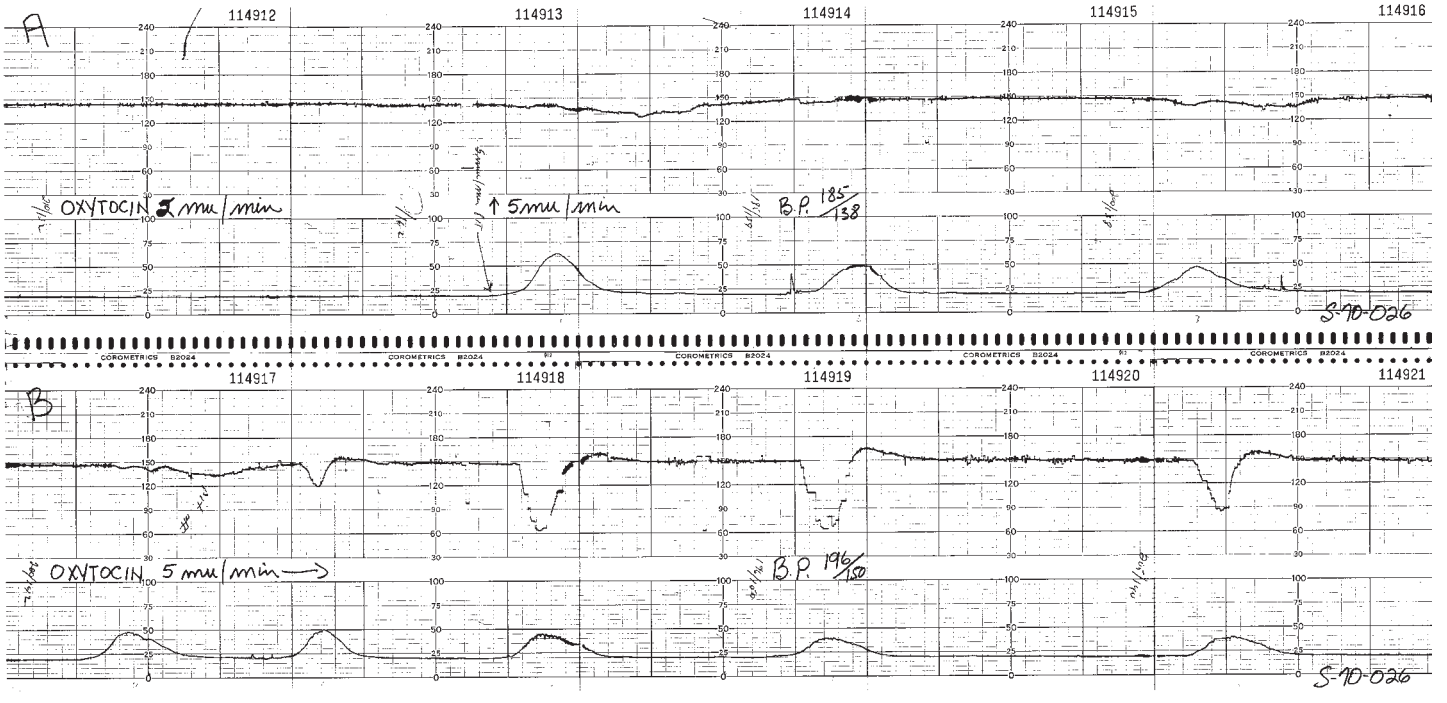

Figure 11.14. These variable decelerations are particularly ominous. They are deep, blunted, and associated with a prolonged terminal increase in fetal heart rate (overshoot) and with a smooth nonreactive baseline. Such patterns have been associated with a high rate of fetal death and neonatal depression.

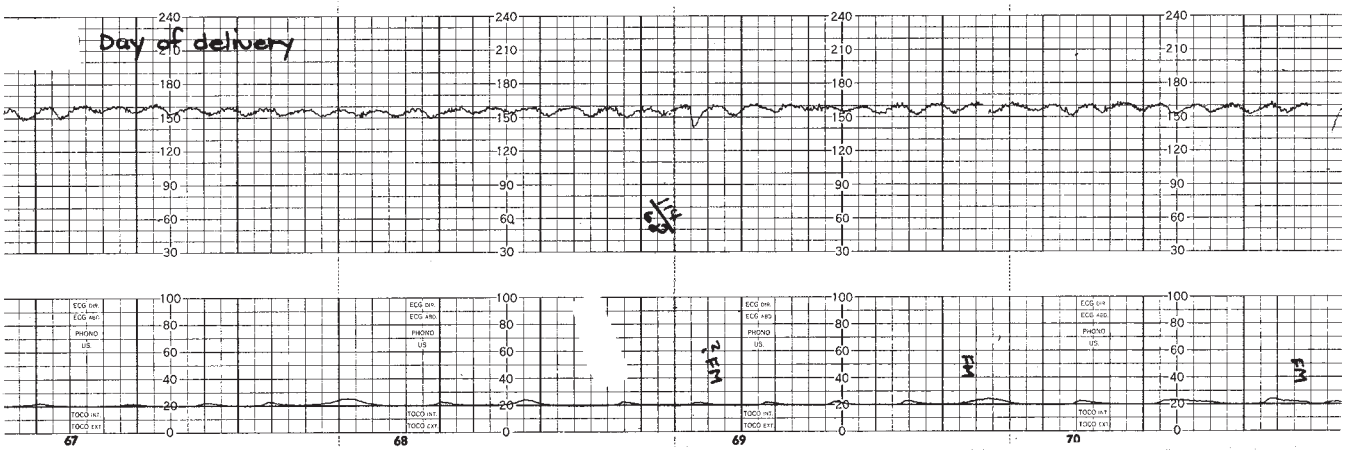

Figure 11.15. Sinusoidal pattern. This is an antepartum sinusoidal heart rate pattern. The sine wave, and long-term variability, may be seen to fluctuate above and below the baseline, is constant, and may be seen with late decelerations. This often represents severe fetal anemia or hypoxia.

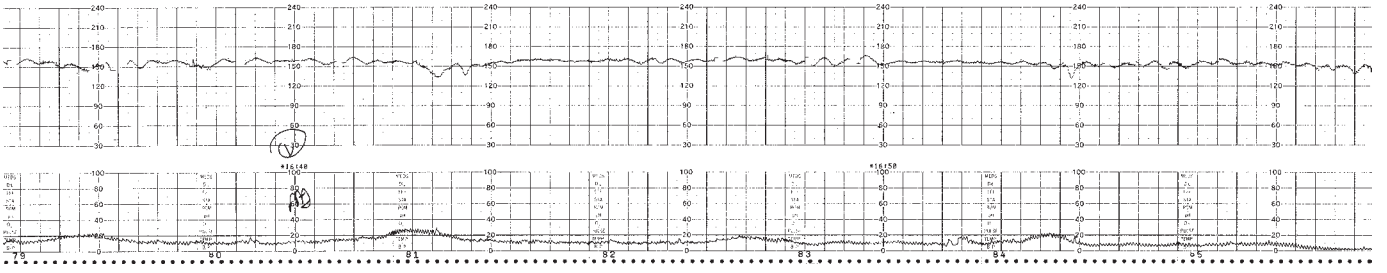

Figure 11.16. Sinusoidal pattern. This pattern is associated with a maternal history of Rh sensitization at 32 weeks' gestation.

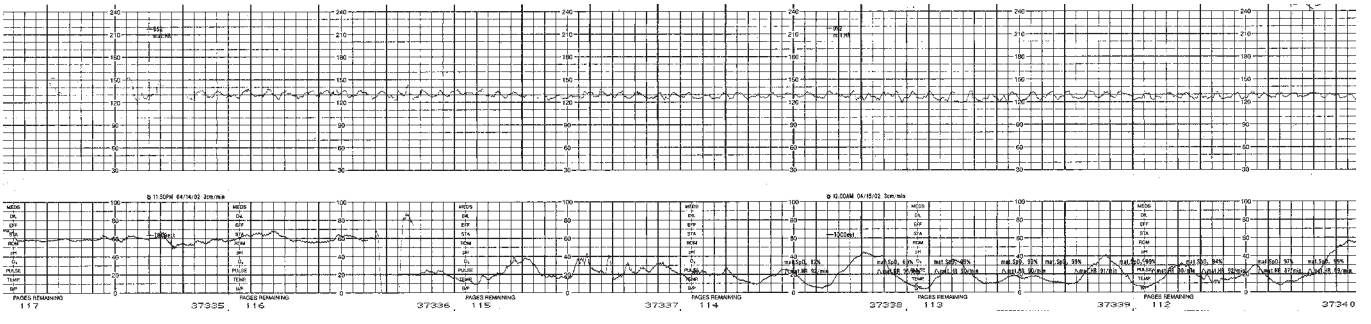

Figure 11.17. This is a pseudosinusoidal pattern. There is an increased cyclicity and presence of short-term variability. The fetal heart rate was reactive before and after this episode.

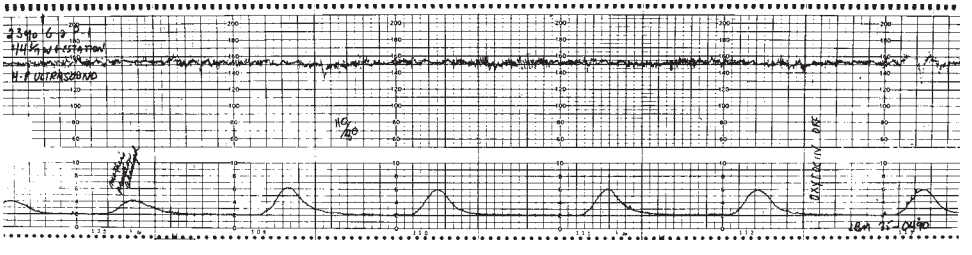

Figure 11.18. Nonreactive positive contraction stress test (CST) taken from a first-generation fetal monitor with significant baseline artifact. Note that there are very subtle late decelerations that are difficult to see. We have seen several similar recordings read as nonreactive negative CSTs followed by fetal death.

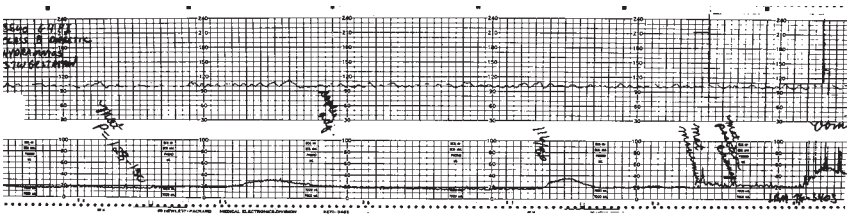

Figure 11.19. Tracing taken from a fetus with anencephaly. Note the absence of reactivity and the absence of late deceleration. This nonreactive negative reading is commonly found in fetuses with anomalies of the central nervous system.

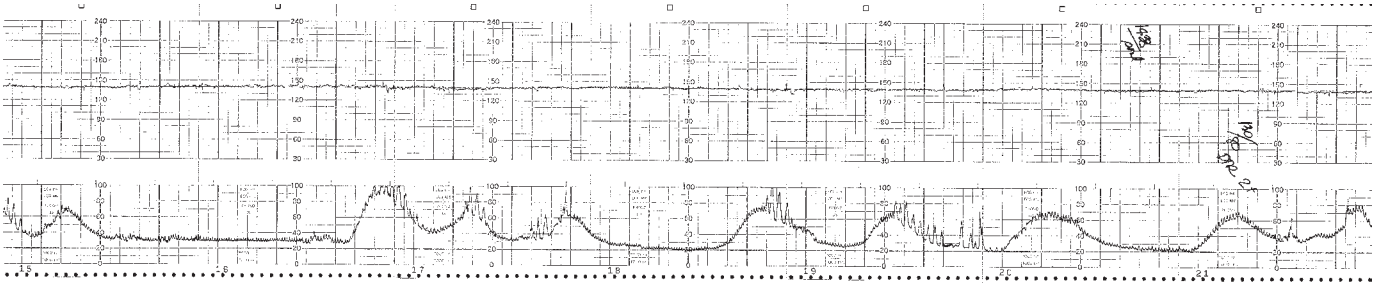

Figure 11.20. Tracing taken from an eclamptic patient who received a dose of phenobarbital for her seizure activity. Note the absence of reactivity and the absence of late decelerations. This nonreactive negative reading is commonly found in patients taking central nervous system–depressant drugs.

outcome in these rare situations did not differ from that of neonates with reactive negative tests. The long-term outcome would be guarded, however, since the lack of reactivity may indicate some preexisting CNS abnormality that was not immediately apparent in the neonate. When one encounters a fetus with a nonreactive negative CST result, it is reasonable to take a drug history and evaluate the fetus for CNS abnormalities with ultrasound and a BPP (Figs. 11.19 and 11.20).

Management

One significant advantage of the CST is that the test has historically been repeated weekly if there are negative test results unless there is some change in the clinical situation (Table 11.5). Equivocal test results require repeat testing the next day. Positive test results are acted on only in the context of the entire clinical condition. This includes gestational age, fetal maturity, maternal condition, and the results of other tests of fetal status. The limitations of abnormal tests must be considered and individualized to the specific clinical situation. Repeatedly equivocal CST results often require switching to another form of fetal assessment and rarely are an indication for delivery. Further, an abnormal CST result may be due to a reversible maternal condition. We have seen abnormal test results in patients with various conditions that have reverted to reassuring tracing after treatment of the maternal condition (Table 11.6).

These maternal conditions would also place the mother at increased risk if operative intervention were undertaken on behalf of the fetus before their correction.

Contraindications

The only part of contraction stress testing that carries any potential risk is the stimulation of uterine contractions. Patients with previous classical cesarean sections or other uterine surgery that has left a scar through the thickness of the fundal portion of the uterus would generally not be

TABLE 11.5 Common indications for shortening the interval between testing

Deterioration in diabetic control
Worsening hypertension
Need to introduce antihypertensives
Decreased fetal movement

TABLE 11.6 Reversible causes of abnormal testing

Diabetic ketoacidosis
Sickle cell crisis
Asthma attack
Dehydration
Maternal anemia

TABLE 11.7 Relative contraindications to the CST (with oxytocin or nipple stimulation)

Premature rupture of membranes
Previous classical cesarean section
Placenta previa
Incompetent cervix
History of premature labor in this pregnancy
Multiple gestation

CST, contraction stress test.

candidates for stress testing. We have used fetal stress testing with oxytocin or nipple stimulation in those patients with previous cesarean sections of the low transverse type and have not observed any problems related to the scars. There is no indication that stress testing causes premature labor (50), but we have generally avoided the CST in patients at high risk for prematurity. This would include preterm patients with multiple gestations, premature rupture of membranes, bleeding, cervical cerclage in place, or previous treatment for preterm labor in the current pregnancy (Table 11.7). Placenta previa is also a relative contraindication to testing with the CST. There are unusual situations in which one might employ the CST even in the presence of such relative contraindications. This would occur when all other indices suggest antepartum fetal distress and the only alternative to the CST is intervention.

NONSTRESS TESTING

The examination of characteristics of the baseline FHR unrelated to contractions and its application to the antepartum period came as a result of observations by Hammacher (28) in 1966 and by Kubli et al. (4) in 1969. They observed that healthy fetuses displayed normal oscillations and fluctuations of the baseline FHR, and when these were absent, there was an increased chance of depressed neonates and perinatal mortality. Trierweiler et al. (51) pointed out that accelerations of the FHR during antepartum stress testing seemed to correlate with fetal well-being. Subsequently, several investigators have looked at accelerations of the FHR and other parameters of the nonstressed antepartum FHR as a means of evaluating the fetus at risk (46,51–61). These other parameters include baseline heart rate, apparent heart rate variability, and the presence or absence of spontaneously occurring decelerations.

Accelerations of the FHR occur in association with FM (Fig. 11.21) or uterine contractions (Fig. 11.22), or in response to external stimuli (Fig. 11.23). Rabinowitz et al. (11) have shown that accelerations are virtually always associated with FM on simultaneous ultrasound imaging, but movement also frequently occurs without heart rate acceleration. Clinically, therefore, it is not necessary to document FM to satisfy the criteria for fetal reactivity. Accelerations of the FHR seem to be an objective reflection of FHR variability, which is not monitored well externally. Fetal sleep-like states and CNS-depressant drugs reduce both variability and reactivity (62). Accelerations may also be caused by partial compression of the umbilical cord resulting in venous occlusion and fetal hypotension

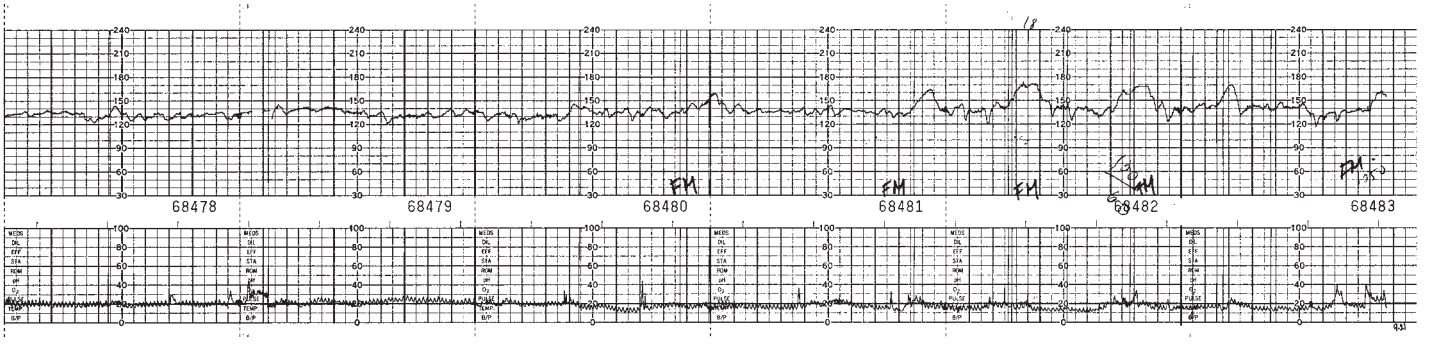

Figure 11.21. Reactive nonstress test showing accelerations associated with fetal movement, marked by the notation "FM."

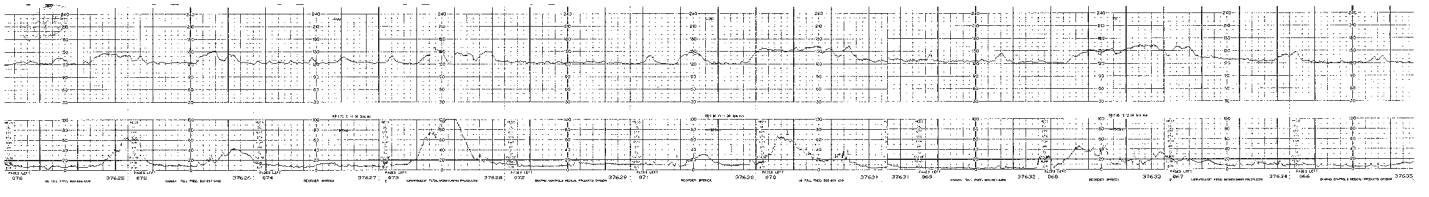

Figure 11.22. These accelerations appear to occur in association with uterine contractions.

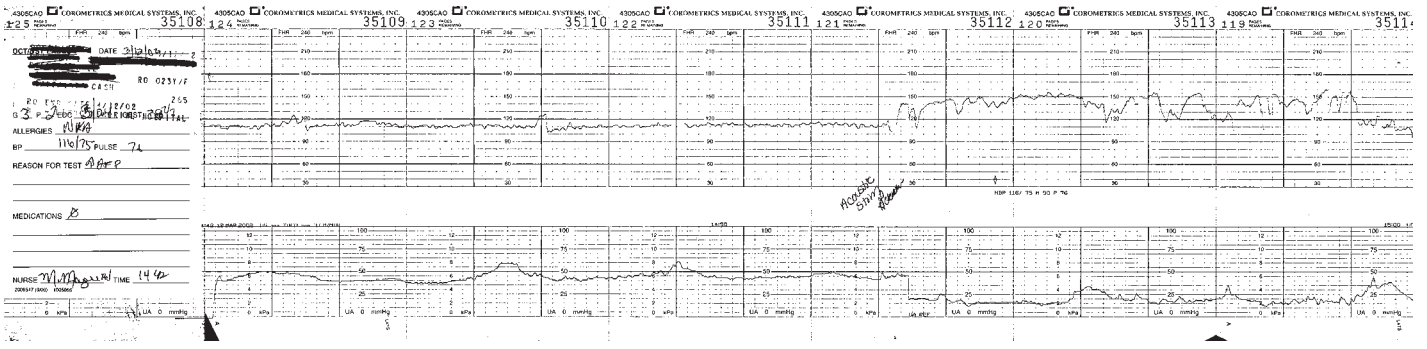

Figure 11.23. This is an example of an increased baseline fetal heart rate (FHR) with arousal from sleep. The fetus had been nonreactive for 30 minutes with an FHR baseline in the 110s when the sound stimulation (noted in **panel 121**) was administered. Note the acceleration induced by the sound stimulation.

without interference in umbilical arterial flow. Therefore, FHR accelerations appear to be a reflection of CNS alertness and activity, and their absence seems to depict CNS depression caused by hypoxia, drugs, fetal sleep, or congenital anomalies.

In 1977, Read and Miller first studied the fetal response to sound as a test for fetal well-being (63). In 1984, Serafini et al. (64) reported on the correlation between the lack of accelerations in response to sound and subsequent fetal distress in labor. They reported that the sound stimulation test also decreased the time necessary for an NST. Smith et al. (65) have evaluated the use of sound stimulation with an artificial larynx as a means of eliciting reactivity and shortening the time for an NST. The same group showed that there was also a good correlation between response to sound with acceleration and fetal pH during labor (66). The use of sound stimulation decreases the time for an NST, and the accelerations produced are as reassuring as spontaneously occurring accelerations.

The endpoint of the NST is the presence or absence of FHR accelerations within a specific time. A great variety of criteria have been set by different authors for what constitutes a reactive NST. Most clinicians use two accelerations of 15 beats per minute (BPM) for 15 seconds in a 20-minute period as satisfying the criteria for reactivity. Brown and Patrick (67) pointed out in 1981 that the length of time a fetus is nonreactive is highly correlated with fetal compromise, and when the fetus remains nonreactive for more than 80 minutes, it is virtually always found to have evidence of significant compromise and remains nonreactive. Leveno et al. (68) confirmed this finding. One of the problems with using FHR accelerations as an endpoint for fetal well-being is the extension of this to pregnancies of <32 weeks' gestation. A healthy fetus under 32 weeks may not have reactivity or accelerations that meet the magnitude requirement of 15 BPM peak and an acceleration duration of 15 seconds. Because nonreactivity may be due to prematurity and not fetal jeopardy, one must consider this fact in the interpretation of NST results before 32 weeks' gestation. The more remote from term that testing occurs, the more likely that nonreactivity will be due to fetal prematurity (69,70). We generally use 28 to 30 weeks as the gestational age at which we expect reactivity to be present or strongly suspect fetal CNS dysfunction. Devoe et al. (71,72) have used the percentage of acceleration time as well as sequential analysis of tests in a given patient to increase the accuracy of NST surveillance. In a recent ACOG Practice Bulletin, an acceleration of the FHR in a gestation of <32 weeks was defined as having a peak of 10 BPM or more above baseline, with a duration of 10 seconds or longer but <2 minutes from onset to return (73).

Performing the Nonstress Test

In the United States, the most common method of antepartum testing is the NST. The patient is placed in the semi-Fowler's position (74), blood pressure is recorded, and the external monitors are applied. The tocodynamometer is included for recording of spontaneous contractions and FM. FM is recorded on the lower channel in one of two ways.

Either the patient informs the nurse, who charts the FM on the tracing (Fig. 11.21), or an event marker (supplied with many monitors) is given to the patient to push each time the fetus moves (Fig. 11.24). This is recorded on the lower channel, usually with an arrow or vertical line (Fig. 11.25). A 20-minute period is recorded. According to the criteria of Evertson et al. (61), if there are two or more accelerations in 20 minutes, the test result is interpreted as reactive and is concluded (Fig. 11.26). Accelerations are defined as an increase of at least 15 BPM above the baseline lasting at least 15 seconds. If there are insufficient accelerations, fetal sound stimulation will often elicit an acceleration response. Manual manipulation of the uterus and fetus may elicit accelerations of the FHR, but Druzin et al. (75) showed no effect in a controlled study. Should the lack of accelerations (less than two) in a 20-minute period persist, the test is interpreted as nonreactive and a backup test or continued monitoring should be performed (Figs. 11.27 and 11.28).

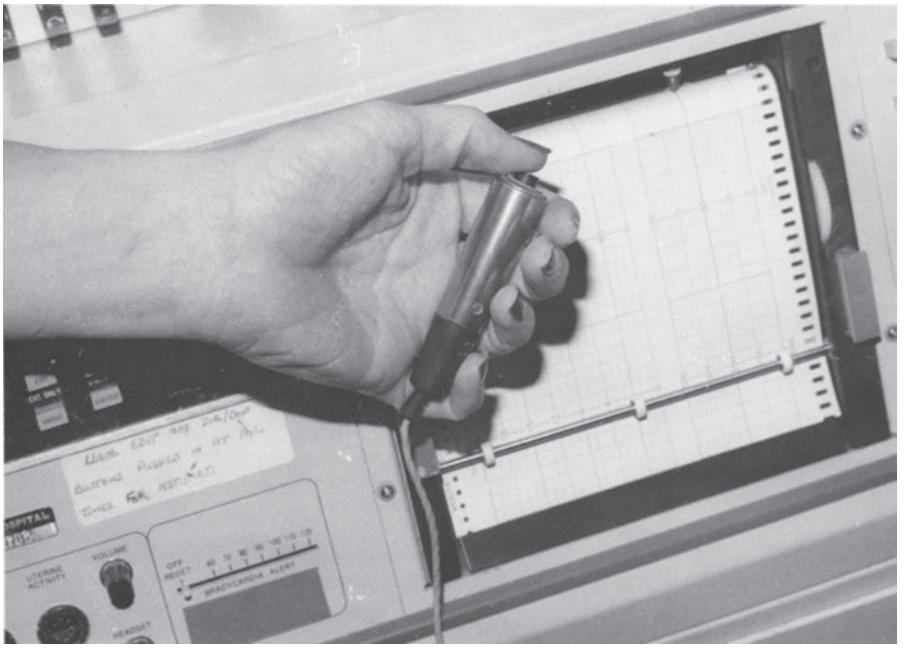

Figure 11.24. Event marker for recording fetal movement. When the patient presses the button, an arrow is printed on the lower channel of the fetal monitor record (see Fig. 12.25).

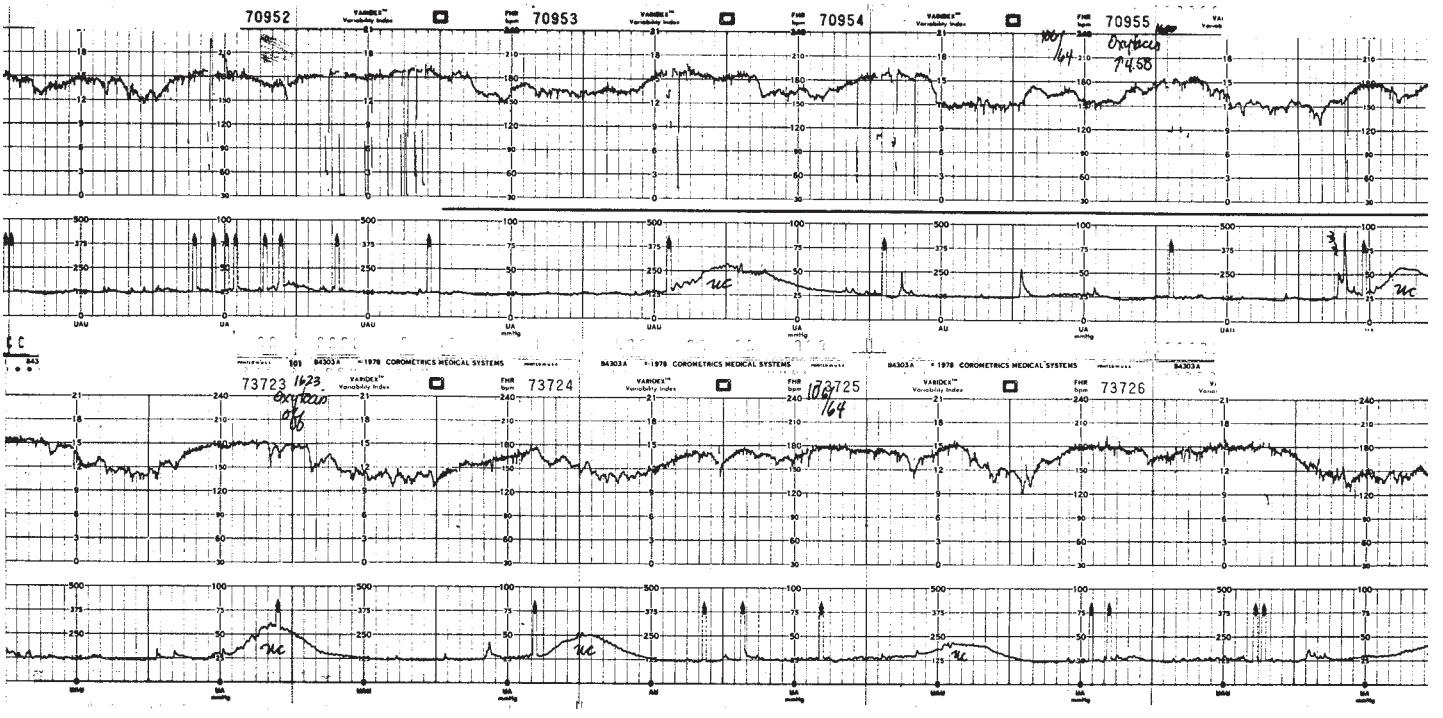

Figure 11.25. Note the vertical *arrows* produced by the fetal monitor when the patient pushed the event marker at the time of perceived fetal movement.

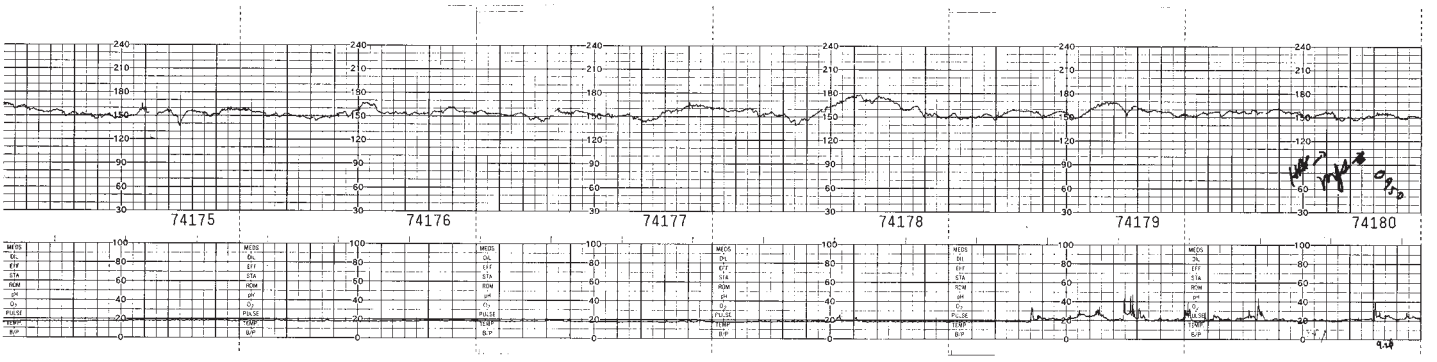

Figure 11.26. Reactive nonstress test characterized by fetal heart rate accelerations of 15 beats per minute or greater lasting 15 seconds or more from onset to offset.

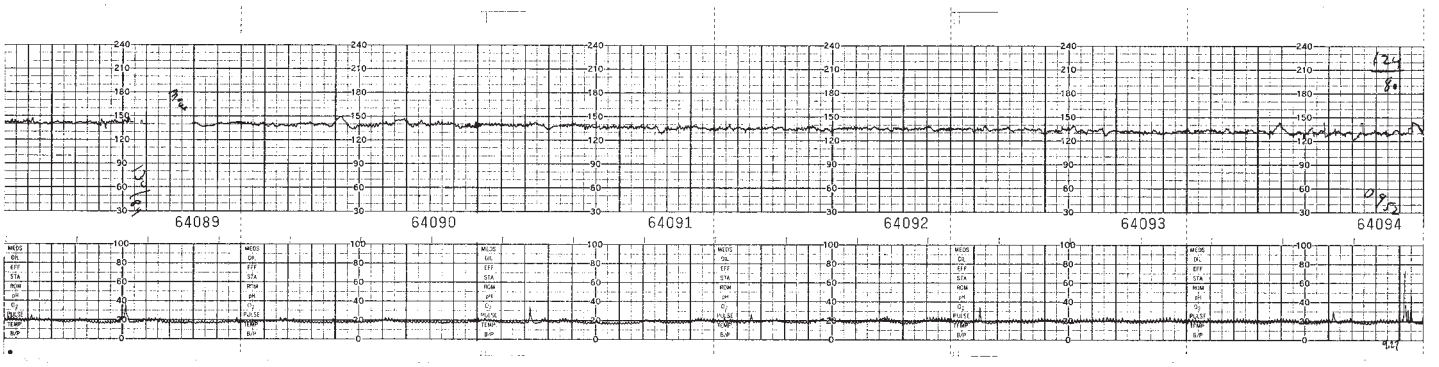

Figure 11.27. This nonreactive nonstress test does not show adequate fetal heart rate acceleration to meet the criteria for reactivity. It must be further evaluated.

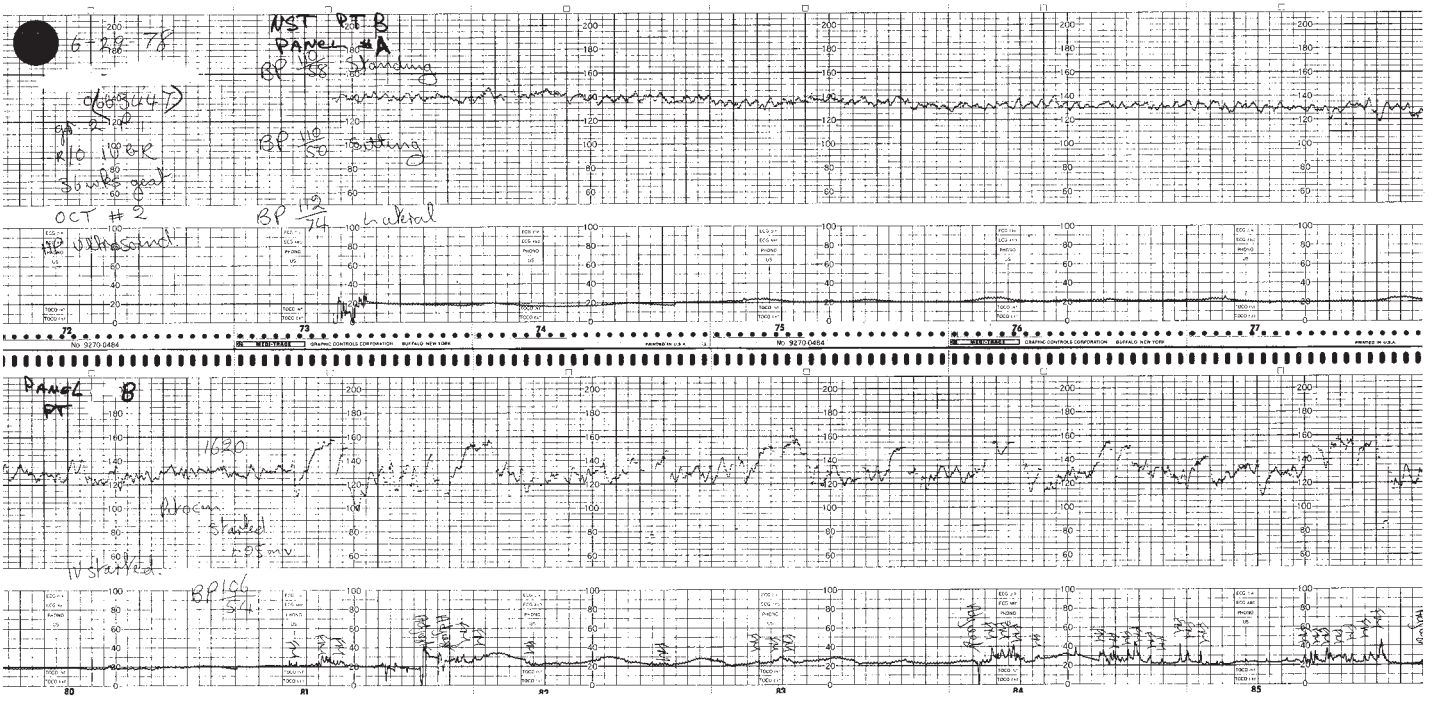

Figure 11.28. Tracing illustrating a reactive negative contraction stress test (CST) following a nonreactive CST. This is a reassuring test, and repeat testing is indicated in 1 week.

There has not been universal agreement on the number of accelerations required to consider a test result reactive. Evertson et al. (61) looked at this question specifically. They found that in NSTs followed by CSTs, there were no positive CST results when two or more accelerations were observed in 20 minutes. These are the only objective data available, and this seems the most appropriate way of choosing this endpoint. However, in our extensive experience with CSTs, we have seen many positive CST results with more than two accelerations in the prestress recording period. When interpreting a CST, we recommend including the presence or absence of FM and heart rate accelerations.

BIOPHYSICAL PROFILE TESTING

The BPP has become a common means of providing antepartum fetal assessment. The BPP score is based on a composite of four dynamic fetal variables (fetal breathing, movement, tone, and the NST) and one long-term variable (amniotic fluid volume) (Table 11.8). When first reported in 1980 by Manning et al. (76), the BPP score showed a very high correlation with fetal well-being when normal (i.e., scores of 8 or 10) as well as with fetal compromise when abnormal (i.e., scores of 4 or less). Further, there is a highly significant inverse relationship between the BPP score and pH in the fetal blood obtained either with antepartum cordocentesis (77) or at elective cesarean section in nonlaboring patients (78), with a normal BPP score always associated with the absence of fetal acidemia. Although commonly used as originally described, the BPP has undergone two modifications by Manning's group. The definition of oligohydramnios was changed from the largest pocket of amniotic fluid being no >1 cm to a pocket no >2 cm (79). A further modification was reported in 1987 with the NST being performed only when one or more of the dynamic ultrasound variables were abnormal (80). Other definitions of oligohydramnios using a composite amniotic fluid index (AFI) of 5 cm or less have also been reported with the BPP.

When used as the primary fetal surveillance test, the BPP has a very low false-negative rate reported between 0.7 and 2.3 per 1,000 tested patients (81). In specifically evaluating the various components of the BPP, Vintzileos et al. (82) reported that the strongest correlation with perinatal mortality was the absence of fetal tone. However, in cases of severe oligohydramnios (e.g., IUGR or preterm premature rupture of membranes [PPROM]), tone may be hard to evaluate. The use of the BPP in a setting of a nonreactive NST was found to be helpful. With a reactive NST, the addition of other BPP parameters did not improve predictability of fetal status. These findings supported the current common approach of using the BPP as a backup test for patients with nonreactive or equivocal nonstress testing.

MODIFIED BIOPHYSICAL PROFILE

As experience had been gained with antepartum fetal testing, it has become evident that the tests with the best predicative value have both an acute marker (acceleration of FHR, FM, fetal tone, fetal breathing) and a chronic marker reflecting uteroplacental reserve (amniotic fluid volume, FHR response to uterine contractions). Although, easy to perform, the NST has a relatively high false-negative rate of 3.2 per 1,000 (23). With accelerations of the FHR, fetal tone and movement are present. The addition of a semiquantitative amniotic fluid

TABLE 11.8 Biophysical profile scoring

Biophysical variable	Normal (score = 2)	Abnormal (score = 0)
1. Fetal breathing movements	≥1 episode of ≥30 s in 30 min	Absence or <30 s in 30 min
2. Gross body movements	≥3 discrete body/limb movements in 30 min	≤2 discrete body/limb movements in 30 min
3. Fetal tone	≥1 active extension/flexion of limb, trunk, or hand	Slow or absent fetal extension/flexion
4. Reactive fetal heart rate	≥2 accelerations of ≥15 BPM for ≥15 s in 20 min	>2 accelerations
5. Qualitative amniotic fluid volume	≥1 pocket of fluid >1 cm in two perpendicular planes	No pocket >1 cm in two perpendicular planes

Our protocol uses a 5.0-cm AFI cutoff for normal score.
BPM, beats per minute.
From Manning FA, Platt LW, Sipos L: Antepartum fetal evaluation: development of a biophysical profile. *Am J Obstet Gynecol* 136:787, 1980, with permission.

assessment to the NST provides a chronic marker to an acute marker. Using twice-weekly "modified BPP" testing, Clark et al. (83) suggested that such an approach was comparable in results to the weekly CST. In a large, prospective study, we report that the modified BPP had similarly low false-negative results to the CST, and using the BPP as backup for equivocal tests was associated with fewer interventions than with the CST as backup (8,84).

Performing the Modified Biophysical Profile Test

The modified BPP can be performed by antepartum testing nurses trained in the ultrasound measurement of amniotic fluid volume and the dynamic fetal assessments of tone, movement, and breathing. A standard NST is combined with an AFI (85). The test result is considered negative if the NST is reactive without decelerations and the AFI is >5.0 cm. If the NST is nonreactive, has decelerations, or if the AFI is 5.0 cm or less, a BPP is performed for backup test. If the BPP score is 8 or 10, the MBPP is repeated in 3 to 4 days. If the BPP score is 6 or less, clinical management may include delivery, continuous monitoring, or repeat testing in 1 day. As with all forms of fetal testing, decisions regarding clinical management must be individualized with consideration given to several clinical factors including maternal status, gestational age, and fetal maturity.

Negative test results are repeated every 3 to 4 days. If the AFI is >5.0 cm, a repeat assessment of amniotic fluid is not indicated for 1 week (86). We use the modified BPP in all indications for antepartum fetal surveillance with the exception of patients with insulin-dependent diabetes mellitus. In such patients, we perform weekly modified BPP with a midweek CST because AFI is not a reliable chronic marker in the insulin-dependent diabetic.

PRIMARY FETAL SURVEILLANCE

Historically, the CST, NST, BPP, and modified BPP have been reported as means of primary fetal surveillance testing. Although efforts have been made to establish the "best" test based upon various outcome parameters, there have been no adequate prospective randomized studies comparing the various testing modalities (87). Each method of antepartum testing has unique advantages and disadvantages. Although requiring only once a week testing and associated with a false-negative rate of 0.4 per 1,000, the CST is more difficult and costly to perform and has a relatively high rate of false-positive and equivocal results (23). The NST, although easy to perform and interpret, has a false-positive rate approaching 50% and a high false-negative rate of 3.2 per 1,000 (23). The complete BPP is more time-consuming than the NST and includes necessary technology and experience in ultrasound measurements of various parameters. It may also be significantly more expensive, depending on the specific costs applied to the BPP performance and interpretation. The most recent and largest report of cumulative false-negative rate was between 0.7 and 2.29 per 1,000 (81). The false-positive rate for the complete BPP is approximately 40% if the endpoint is fetal or neonatal compromise (88). The clinical significance of this is dependent on the gestational age and the specific perinatal endpoint examined. The modified BPP has a low false-negative rate of 0.8 per 1,000 and an intermediate false-positive rate of iatrogenic prematurity in 1.5% of women tested preterm (88). The final decision regarding choice of fetal surveillance test is most often determined by institutional preference and experience. What is clear is that all forms of fetal testing are valuable and need to be interpreted cautiously with full knowledge of the specific test limitations as well as the physiology of the indication for testing. Testing frequency of the CST and the BPP is generally weekly, but it is recommended that the BPP be performed twice weekly for certain high-risk conditions (postdate pregnancy, insulin-dependent diabetes mellitus) (7). Unpredictable morbid events such as massive fetomaternal hemorrhage may explain some cases of fetal demise following negative test results (81,89).

PRETERM PREMATURE RUPTURE OF MEMBRANES

Antepartum assessment in a pregnancy complicated by PPROM is both challenging and important because of the high degree of associated perinatal morbidity and mortality (90–93). Major risks include infection, cord prolapse, stillbirth, fetal deformity, and compromise. Although the CST is contraindicated with PPROM, various investigators have reported on the use of amniotic fluid assessment, nonstress testing, and BPP for this clinical condition (94–98). There appears to be a correlation between a low AFI and the development of variable decelerations and nonreactivity (94). The initial AFI is possibly associated with duration of latency period, response to tocolytic therapy, and possibly with infection, although there is not consensus on all these relationships (94,95,98,99). Following an initial period of clinical assessment and continuous FHR monitoring, a daily 1-hour NST is a reasonable protocol to follow in patients with PPROM.

Clinical Management with Nonstress Testing for Primary Surveillance

The NST is the most popular method for antepartum fetal surveillance because it is easy to perform, easy to interpret, has fewer equivocal results, and has excellent patient and physician acceptance. Traditionally, it has been offered on a weekly basis, but some authors have suggested increasing the frequency to twice weekly (74), especially in postdate and diabetic pregnancies. When the NST result is nonreactive or

has spontaneous decelerations, further testing is necessary. We recommend a full BPP as the backup test.

Some data indicate that the loss of reactivity occurs later than the appearance of late decelerations in a chronically deteriorating fetus. Murata et al. (48) showed that in the monkey fetus undergoing progressive intrauterine hypoxia ultimately leading to death, the first evidence of hypoxia on the FHR pattern was the appearance of late deceleration. Only after the development of significant fetal acidosis did the fetuses lose FHR acceleration. For this reason, it should be kept in mind that if a fetus is truly nonreactive, it may be in very serious condition.

Clinical Management with the Biophysical Profile and Modified Biophysical Profile for Primary Surveillance

As clinicians have gained experience with real-time ultrasound and the equipment has been improved, there has been an increasing interest in using this method for primary fetal surveillance. Manning et al. (80) have been the primary proponents of this approach and currently only use the NST as a backup about 3% of the time. Using this method twice weekly, Manning et al. report results that are comparable to those with the weekly CST and that identify more anomalous babies.

The most common finding triggering intervention is oligohydramnios, yet there is no universal agreement on what constitutes oligohydramnios. Manning et al. (76) originally said that a 1-cm vertical pocket was adequate, but Vintzileos et al. (82) advocated intervention with pockets measuring <2 cm. Perhaps a better approach is to use the four-quadrant measurement of 5 cm total or greater, as described by Rutherford et al. (85). Clearly, there is a very high perinatal morbidity among patients with significant oligohydramnios (76,77). Most studies have shown that primary NST surveillance has a higher antepartum fetal death rate than primary CST surveillance or primary BPP surveillance. Perhaps this is because, in the fetus with clinically significant oligohydramnios, the condition will not be detected unless there are contractions or the patient has an ultrasound study. Current recommendations for the frequency of testing with the BPP are not well established, varying from once weekly testing to twice weekly.

DOPPLER VELOCIMETRY

Doppler velocimetry measurements of various fetal blood vessels are an important additional clinical tool in the assessment of the fetus (100–103). In normal pregnancy, the physiologic conditions of the placenta present a vascular tree of low impedance, which allows continuous forward blood flow in the umbilical arteries throughout the fetal cardiac cycle. With increases in placental impedance, the mostly passive blood flow during diastole may be noted to progressively and unpredictably decrease in the umbilical arteries to, absent and potentially reversed flow. It has been established that these abnormal changes in the pattern of umbilical artery blood flow reflect the presence of placental vascular lesions and that such abnormal Doppler test results require more intensive fetal surveillance and management decisions as they are associated with an increase in adverse perinatal outcome (104,105). Umbilical artery Doppler is a placental test more than a fetal test. While in isolation, Doppler velocimetry is a poor indicator of fetal compromise or fetal adaptation to the placental abnormalities; Doppler greatly assists in the accurate identification of the fetus at increased risk for perinatal mortality.

Clinical studies employing Doppler velocimetry have revealed a strong correlation between high systolic to diastolic (S/D) ratios and IUGR (106–109). Similar associations between increased S/D ratio, absent or reversed diastolic flow, and adverse outcome have been reported in poorly controlled diabetes in pregnancy (110,111) and postdate pregnancies (112). Correlations between abnormal antepartum FHR tests and high S/D ratio have also been reported (113).

As with other forms of fetal surveillance, umbilical artery Doppler velocimetry appears to have clinical utility in the management of high-risk pregnancies, particularly in those pregnancies complicated with IUGR or preeclampsia. Meta-analyses of published, peer-reviewed, randomized controlled trials reveal a reduction in perinatal mortality in those patients whose fetal surveillance included Doppler evaluations without an apparent increase in the rate of inappropriate obstetric interventions (104,114). There is an accurate and critically important application of Doppler velocimetry of the middle cerebral artery in the noninvasive detection of fetal anemia in pregnancies with red blood cell alloimmunization or other causes of fetal anemia (e.g., fetal to maternal hemorrhage or parvovirus infection) (115,116). Umbilical artery Doppler studies play an important role in the staging and management of twin-twin transfusion syndrome with umbilical artery Doppler assessments of both the donor and recipient twins (117).

Doppler velocimetry of various fetal vascular structures (e.g., umbilical artery, middle cerebral artery, ductus venosus) continues to be reported for different indications. As discussed above, the most common current role for this modality in fetal assessment is in the patient with suspected intrauterine growth restriction. While correlations between absent and reversed diastolic flow and various measures of adverse perinatal outcome including stillbirth have been established, care should be given to ensuring the individualization of its application, particularly in pregnancies that are significantly preterm. It has been reported that protracted periods of time can pass without adverse outcomes in a setting of absent or even reversed end-diastolic umbilical artery blood flow documented by ultrasound (118). The clinician

is encouraged to utilize additional measures of fetal status, which include FHR monitoring and/or BPP, in such cases of abnormal Doppler findings with a markedly preterm fetus. Valuable time can often be gained to allow for administration of corticosteroids, attempt to improve the *in utero* environment, and optimize the timing of delivery while other means of monitoring are used for ongoing intensive fetal assessment.

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CHAPTER 12

Antepartum Management of the High-Risk Patient

The previous chapter was devoted to introducing antepartum fetal surveillance, including fetal heart rate (FHR) testing and biophysical profile (BPP); describing methodology; interpretation; and understanding the associated limitations and pitfalls. The appropriate application of these methods in various clinical situations is critically important.

First, we must decide which patients require antepartum testing. Almost half of the antepartum deaths occurring beyond 26 weeks' gestation are in patients at risk for some form of uteroplacental insufficiency (UPI) (1,2). Patients at increased risk for UPI comprise approximately 10% to 20% of the prenatal population, and various means have been used to attempt to correctly identify these women (3). The other half of antenatal deaths, however, occur in patients who would generally be classified as low risk. Low-risk women make up the majority of the obstetric population. One choice is to limit antepartum fetal surveillance to the 10% to 20% of patients at risk for UPI and thereby attempt to prevent this one-half of fetal deaths. Another choice is to test all patients in an effort to attempt to avoid all fetal deaths. This latter choice is an extreme alternative, and there is no study that shows a benefit to the routine fetal testing (beyond fetal movement assessment) of all low-risk patients. The more reasonable and well-accepted approach for identifying the low-risk patient destined to have a fetal death is to carefully observe all patients for normal fetal growth and fetal movement. Many patients with unexpected fetal death will have a growth-restricted fetus. Thus, the general approach is to routinely measure fundal height in all patients receiving prenatal care and evaluate the fetus with ultrasound when the fundal height is significantly less than expected. In addition, although not necessarily recommended, most patients do have a routine ultrasound in the second or early third trimester. When these examinations, for whatever reason, suspect intrauterine growth restriction (IUGR), antepartum testing becomes indicated. Routine fetal movement testing has been shown in large well-controlled studies to decrease the rate of antepartum fetal deaths in low-risk patients (4,5). Thus, all patients should be counseled at some time in the early third trimester to do some kind of daily assessment of fetal movement (Fig. 12.1). There is a great deal of variation in the exact method used for fetal movement counting, as outlined in the previous chapter. Regardless of the specific method chosen, when the patient calls with absent or substantially reduced fetal movement, and when this has been confirmed by having her concentrate for an hour or two in a quiet room, the patient must come in for an immediate nonstress test (NST) regardless of the time of day. Keep in mind, however, that whatever the indication, testing should be implemented only when a gestational age is reached where the clinician, if faced with definitely ominous data, is ready to intervene. Thus, in the 80% of otherwise low-risk patients, the main indications for testing are the development of suspected IUGR and decreased or absent fetal movement. In the remaining high-risk patients, the indications for antepartum FHR testing are summarized in Table 12.1.

TESTING PROTOCOL BY CLINICAL SITUATION

We shall attempt to describe an approach to antepartum testing in terms of categories of risk and specific diagnoses or risk factors that place the patient at increased risk for fetal death from UPI. We shall present our approach to antepartum testing and use this format to provide examples for illustration. It should be clear that there are many different approaches in terms of when to begin testing, when to intervene, different ancillary tests, etc. The approach presented here is based on extensive clinical experience with antepartum heart rate testing in high-risk patients and on data that have been published by the authors and many others (1,6,7).

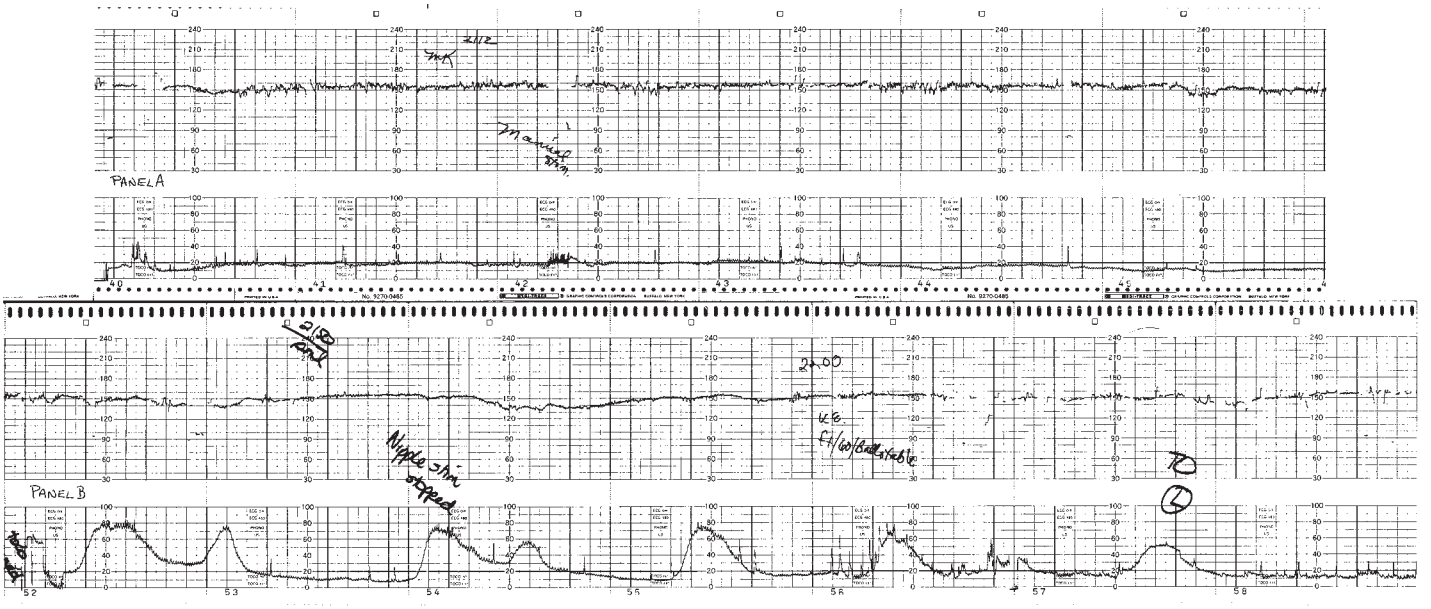

Figure 12.1. A primigravida woman at 39 weeks complained of no fetal movement for 1 day. Previously, there had been a decrease in movement frequency over 2 days. A nonstress test was performed (**panel A**) and was read as nonreactive with suspicious areas. This was immediately followed by a nipple stimulation contraction stress test (CST) (**panel B**). The CST result was interpreted as nonreactive and positive. Because the cervix was unfavorable, an immediate cesarean section was performed with delivery of a 3,180-g infant with Apgar scores of 4 at 1 minute and 7 at 5 minutes. Umbilical artery pH was 7.10 with a pCO_2 of 49 and a base deficit of 11. Fresh meconium was noted; there was no evidence of placental separation. The infant did well in the newborn period.

Which Test to Use

Controversy exists as to which form of antepartum surveillance provides optimal results. Issues such as patient population, available facilities, cost, convenience, testing interval, and the specific indication for testing must be considered when selecting the particular means of fetal assessment for your patient.

As described in the previous chapter, the most frequently used method for primary surveillance is the **modified biophysical profile**. The NST is almost always performed at least twice weekly. The frequency of the amniotic fluid (AF) volume assessment may be once or twice weekly. In a review of a large series of amniotic volume testing, Lagrew and coworkers found that in patients with an amniotic fluid index (AFI) of 8 cm or greater, a change to significant oligohydramnios (AFI <5) rarely occurred in less than a week, except in patients with IUGR and who go beyond 41 weeks (8). Thus, it is logical to test these latter two groups of patients and those with borderline AFI (5.0 to 7.9 cm) with twice-weekly NSTs and AFIs and the remainder with twice-weekly NSTs and once-weekly AFIs. One group of patients that we approach differently are those with diabetes for reasons described in the following section. In general, other tests of fetal well-being are then reserved for "backup" testing in those patients with an abnormal NST or AFI result (Fig. 12.2). The one exception perhaps is with Doppler testing of umbilical artery flow. This modality may have additional utility in clarifying the diagnosis of IUGR and prognosticating which fetuses are destined to require early delivery.

Moderate-Risk Groups

There is a group of conditions in which the risk for fetal death from UPI is only moderately increased above that of the low-risk population. Furthermore, in this group, fetal death tends to occur late in gestation. Thus, in general, for these patients, we recommend beginning testing at approximately 34 weeks. These clinical situations include advance maternal age, maternal hyperthyroidism, and previous stillbirth. Women over the age of 35 years have previously been thought to represent a risk group for whom fetal surveillance is indicated. However, at least up to the age of 40 years, unless the pregnancy is complicated by diabetes, hypertension, or other problems, women do not have any increased risk for stillbirth or abnormal FHR tests secondary to UPI (9,10).

Women with a diagnosis of hyperthyroidism or a history of hyperthyroidism may be at risk for fetal compromise. The etiologic agent is a thyroid-stimulating immunoglobulin of the IgG class. Thus, this antibody can cross the placenta and cause fetal hyperthyroidism and tachycardia. Women with a history of hyperthyroidism who are euthyroid following an ablative procedure (i.e., surgery or radioactive iodine) may still have the antibody present during a subsequent pregnancy, with potential fetal compromise. Even without the fetus

TABLE 12.1 Indications for antepartum fetal surveillance

Postdate pregnancy
Diabetes Class A2 through R Class A1 with History of stillbirth Pregnancy complicated by hypertension or suspected IUGR Class A1 at 40 weeks' gestation
Preeclampsia
Chronic hypertension
Decreased fetal movement
Previous stillbirth
Suspected IUGR
Discordant twins; concordant monochorionic twins at 37 weeks' gestation
Preterm premature rupture of membranes
Rh isoimmunization
Cyanotic cardiac disease
Hemoglobinopathy/severe anemia
Asthma
Hyperthyroidism
Chronic renal disease
Collagen vascular disease

Maternal age >40 y.
IUGR, intrauterine growth retardation.

being directly involved, poorly controlled hyperthyroidism may divert blood and oxygen from the fetus because of the increased metabolic demands of maternal tissues. For these reasons, we recommend testing in these women. Similarly, women with a history of an unexplained stillborn are begun on fetal surveillance at some point before the gestational age at which the loss occurred. Although this primarily provides maternal reassurance, we have found an increased incidence of abnormal test results in these patients, particularly in women with a history of previous stillbirth associated with a current diagnosis of hypertension or suspected IUGR (11,12).

Postdate Pregnancy

In most series, "postdate" pregnancy is the condition accounting for the majority of patients undergoing antepartum testing. Often this is because many patients have wrong dates. Postdate testing is relatively controversial, primarily from the standpoint of when to begin testing and when to deliver. Studies of contemporary practice suggest that the majority of practitioners will recommend delivery for patients at or beyond 41 weeks with a favorable cervix and for all patients after 42 weeks regardless of the condition of the cervix (13), which is our practice as well. Although the data suggest no substantial increase in asphyxia or stillbirth until after 42 weeks, a relationship exists between the incidence of abnormal antepartum testing results and advancing gestational age with positive test results increasing at and after 41 weeks (14) (Fig. 12.3). Thus, we begin testing at 41 weeks. Postdate patients also have a high frequency of oligohydramnios. Oligohydramnios (AFI <5.0 cm) in a patient with a well-dated pregnancy beyond 41 weeks is virtually always an indication for delivery. The FHR during an NST or contraction stress test (CST) in patients with oligohydramnios will frequently demonstrate recurrent prolonged or variable decelerations, as the protection the fluid offers the umbilical cord is diminished. Such patients pose an additional dilemma as cervical ripening agents often result in uterine hyperstimulation and prolonged cord compression. Often, it is wise to precede the placement of the ripening agent with a negative CST result, which can indicate how well the fetus will tolerate uterine contractions. If there are decelerations associated with contractions in such patients, it is preferable not ripening at all or alternatively to choose an agent that is not likely to result in uncorrectable prolonged contractions (e.g., Foley catheter).

Currently, it appears that most clinicians use the modified BPP for primary surveillance in the postdate pregnancy. As previously mentioned, we perform twice-weekly NSTs and AFIs in these patients. Because the only reason not to deliver these patients is an unfavorable cervix, which may increase the likelihood of failed induction and cesarean section, the threshold for delivery for abnormal tests is relatively low in this group of patients (15,16). Certainly, a positive CST result or low BPP (4 or less) is an indication for delivery (16–18). Most often, however, these patients will be delivered for a low AFI and/or significant variable or prolonged decelerations. In these patients, these less ominous results may also be an indication for delivery since the only reason not to deliver the postdate patient is the risk of failed induction and cesarean section, and thus, the risk benefit ratio requires less impetus to weigh in favor of delivery.

Preeclampsia

Preeclampsia is a disease for which it is particularly difficult to outline a routine for antepartum evaluation. This is because management is more often guided by maternal condition than by fetal condition. Conservative treatment of the maternal disease (i.e., bed rest) can improve fetal condition, and, conversely, deterioration of the maternal condition can result in worsening UPI. Gant et al. (19,20) showed that UPI precedes the clinical manifestations of preeclampsia by 1 to

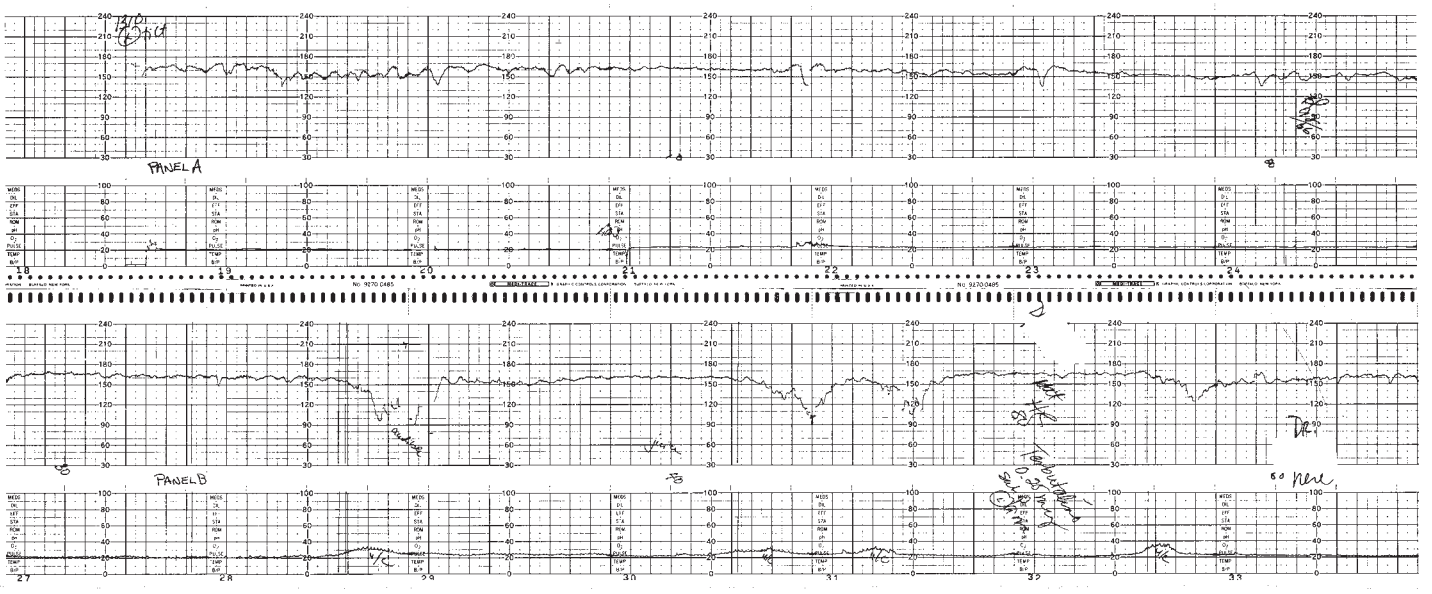

Figure 12.2. A 21-year-old gravida 1 para 0 at 34½ weeks was being observed for suspected intrauterine growth retardation with biweekly modified biophysical profiles (nonstress test [NST] and amniotic fluid index [AFI]). Her initial NST result **(panel A)** was reactive with occasional mild variable decelerations of unknown significance. Because her AFI was 2.8 cm, she received a backup test, which was a contraction stress test (CST) **(panel B)**. The CST result was read as positive and did not meet reactivity standards. The patient underwent immediate delivery by cesarean section, delivering a 1,730-g newborn with Apgar scores of 8 at 1 minute and 9 at 5 minutes. Umbilical cord gases were normal. Nuchal cord was noted with no amniotic fluid at delivery. The infant subsequently did well in the nursery.

3 months. The fact that IUGR can be seen in patients many weeks before developing signs and symptoms of preeclampsia corroborates these data.

Antepartum testing in patients with preeclampsia should be conducted with the following basic rules in mind:

1. Fetal jeopardy may exist even in the patient whose blood pressure normalizes at bed rest.
2. The clinical condition may change rapidly. Should such changes occur, repeat evaluation of the fetus must be performed even when the most recent assessment was reassuring.
3. Fetal condition is but one variable in the equation of evaluation and management of these patients.
4. Occasionally, abnormal antepartum heart rate patterns improve when the maternal condition significantly changes for the better (e.g., control of severe hypertension).
5. Patients with severe preeclampsia require continuous monitoring of FHR even if the decision is made to delay delivery for extreme prematurity or to take the time to administer corticosteroids.

Antepartum fetal monitoring is begun when the disease is first recognized and viability is considered likely. In our institution, this is around 24 weeks' gestation (Fig. 12.4). As with other antepartum conditions, testing should occur every 3 to 4 days using the modified BPP, although AF volume can be assessed every week if the previous value was normal (≥8 cm). If the preeclampsia is severe and the patient is not being delivered in an effort to gain additional maturity, consideration should be given to daily NSTs or continuous monitoring because the situation is very dynamic.

Chronic Hypertension

In terms of fetal jeopardy from UPI, chronic hypertension, especially when poorly controlled, is one of the most profound antepartum risk factors. Many cases of chronic hypertension are complicated with IUGR. In these cases, the risk is especially high, and fetal death may occur at any point. Consequently, we recommend initiation of antepartum surveillance beginning as early as 26 to 28 weeks in these patients, or sooner if there is suspicion of IUGR (21). As with other patients, testing can be conducted using the modified BPP with the NST done every 3 to 4 days and the AF volume assessed weekly unless there is IUGR or the previous value is <8 cm, where AF volume should be checked at least twice weekly. Often, when beginning testing in early gestation, the fetus may be nonreactive because of gestational age. It is reasonable to start with an NST regardless because 50% of fetuses will meet criteria for reactivity by 24 to 26 weeks (22). Some modification of the definition of reactivity may be made for gestations of <32 weeks. One should also use the patient as her own baseline because those fetuses that were

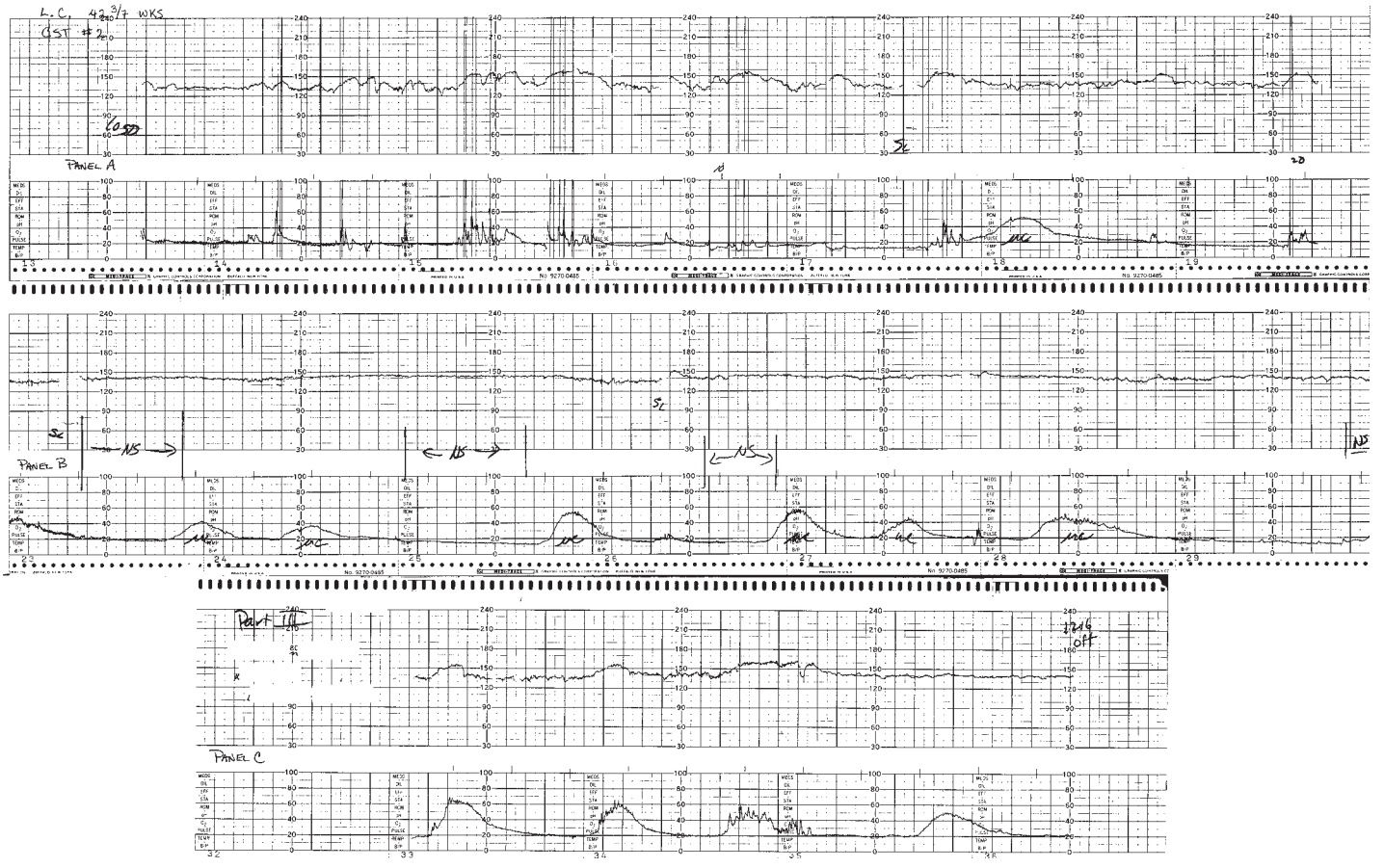

Figure 12.3. The patient was gravida 3 para 1 AB 1 at 42½ weeks by excellent dates and early ultrasound. A contraction stress test (CST) performed 7 days earlier had been reactive and negative. The initial portion of the CST (**panel A**) remains reactive. However, during the CST (**panel B**), there are late decelerations without reactivity. Because reactivity returned as the CST was continued (**panel C**), this test result was interpreted as reactive and positive. The patient was admitted and had artificial rupture of membranes and oxytocin augmentation. Other than passage of thick meconium requiring DeLee suctioning, labor and delivery were uneventful. Intermittent late decelerations were noted but were not persistent. The infant did well in the nursery. Some critics might call this a false-positive CST result; however, we believe that the discovery of late decelerations is potentially of critical importance in deciding whether or not to allow the pregnancy to continue.

previously reactive will not become nonreactive because of immaturity. Patients with chronic hypertension and reassuring fetal surveillance, stable blood pressure, and no evidence of IUGR can be delivered at 38 weeks or later.

Many patients with chronic hypertension develop superimposed preeclampsia. These patients should then be treated as preeclamptics. Patients with chronic hypertension are also at risk for placental abruption; this cannot be predicted, but vaginal bleeding or premature uterine activity requires immediate fetal evaluation with the potential of abruptio placentae in mind.

Patients with chronic hypertension may require changes or adjustments in their medications to control blood pressure during pregnancy. Commonly used antihypertensive medications are beta-blockers, which, among other things, may affect the amount of reactivity of the FHR, especially in high doses. This must be considered when attempting to interpret FHR information either antepartum or intrapartum in patients taking these medications. Another important point regarding hypertension control and FHR monitoring is that when patients with chronic hypertension develop sudden deterioration of their blood pressure control, or in patients with severe preeclampsia, acute lowering of blood pressure may adversely affect uterine perfusion. Care should be taken to avoid rapid reduction to normal or below normal blood pressure because the fetus may become acutely hypoxic and will exhibit late decelerations with too vigorous antihypertensive therapy (see Figure 8.33). Continuous monitoring of FHR while treating severe hypertension is critically important to ensure adequate placental perfusion and identify

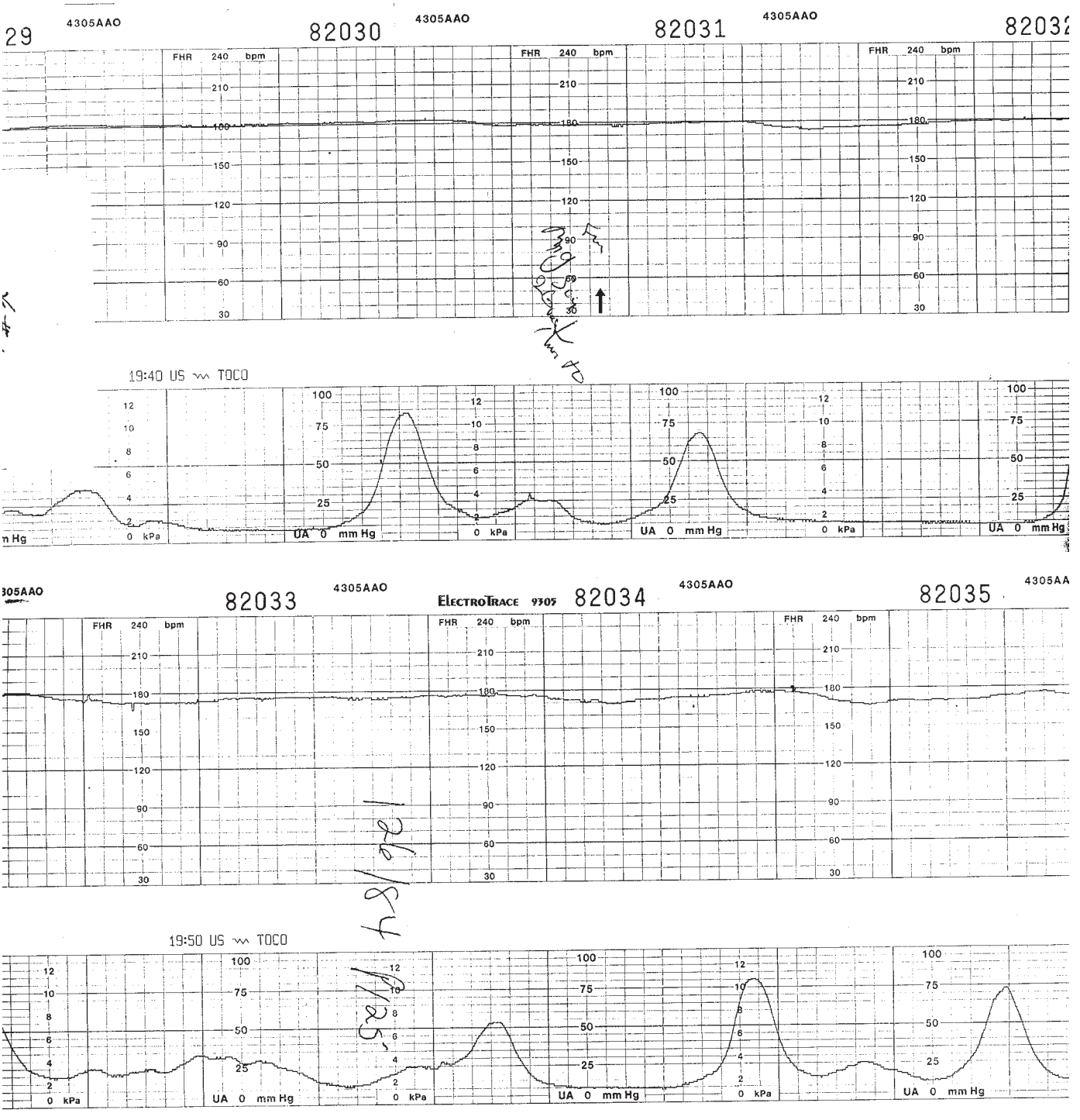

Figure 12.4. This is a 38-year-old gravida 4 para 3 at 29½ weeks admitted with mild blood pressure elevation, 1+ proteinuria, thrombocytopenia, and increased liver enzyme concentrations. She was seen 2 weeks previously without problems. The external fetal monitor revealed a flat baseline tachycardia and persistent late decelerations. After administration of magnesium sulfate, a primary cesarean section was done, and a baby was delivered with Apgar scores of 0 at 1 minute, 3 at 5 minutes, and 5 at 10 minutes, weighing 1,330 g. Cord pH arterial was 6.90 with BE −22 and venous 7.01 with BE −17. The baby ultimately survived.

possible fetal distress. Indeed, using FHR information is of great assistance in establishing an acceptable blood pressure endpoint for both fetus and mother.

Diabetes Mellitus

Pregnancy complicated by diabetes mellitus is an excellent example to demonstrate how the combination of antepartum testing and fetal pulmonary maturity studies has had a positive impact in improving perinatal outcome. In many centers, the perinatal mortality in pregnancies complicated by insulin-dependent diabetes has been reduced to that of nondiabetics when corrected for congenital abnormalities (23,24). Data suggest that class A1 diabetics have no increase in antenatal fetal mortality over the general population (25). This is true only for a class A1 diabetic who is not preeclamptic and has not had a previous stillbirth. Therefore, we do not use antepartum fetal testing in class A1 diabetics before 40 weeks' gestation. In the remainder of diabetics, there is some concern that the modified BPP may not be as effective a surveillance technique as in other pregnancy complications. This is based on both theoretical and experiential grounds. Presumably, because of the diuretic effect of elevated glucose levels in the fetus, these patients often have elevated or high normal AF volumes. This is especially true in the more poorly controlled diabetic who is at the highest risk of stillbirth. Thus, a high normal fluid volume may drop significantly but still remain in the normal range. Some centers with a high volume of diabetic patients have reported high unexpected fetal death rates with NSTs and AFV testing in diabetics (26). For this reason, in this group of patients only, we revert to using weekly CSTs and midweek NSTs for primary surveillance. For patients with diabetes complicating their pregnancies, both complicated class A (previous stillbirth or current preeclampsia) and all insulin-dependent diabetics are managed with weekly CSTs and midweek NSTs. For the majority of these women, testing is begun at 34 weeks' gestation. Such an approach has been verified using decision-analytic modeling (27). For diabetic patients whose pregnancies are further complicated by hypertension, suspected IUGR, or renal disease, fetal surveillance is begun earlier (Fig. 12.5). These are started at 28 to 30 weeks (28). With continued tight diabetic control, good fetal growth, and reassuring fetal surveillance tests, we allow diabetic pregnancies to continue to 38 weeks' gestation and beyond, depending on diabetes control, patient compliance, testing, and cervical condition.

Third-Trimester Bleeding

Patients with third-trimester bleeding are a difficult problem in antepartum management, and care must be individualized. With an acute episode of bleeding, the fetal condition is a critical factor in any decision about immediate delivery. Once it has been established that bleeding is

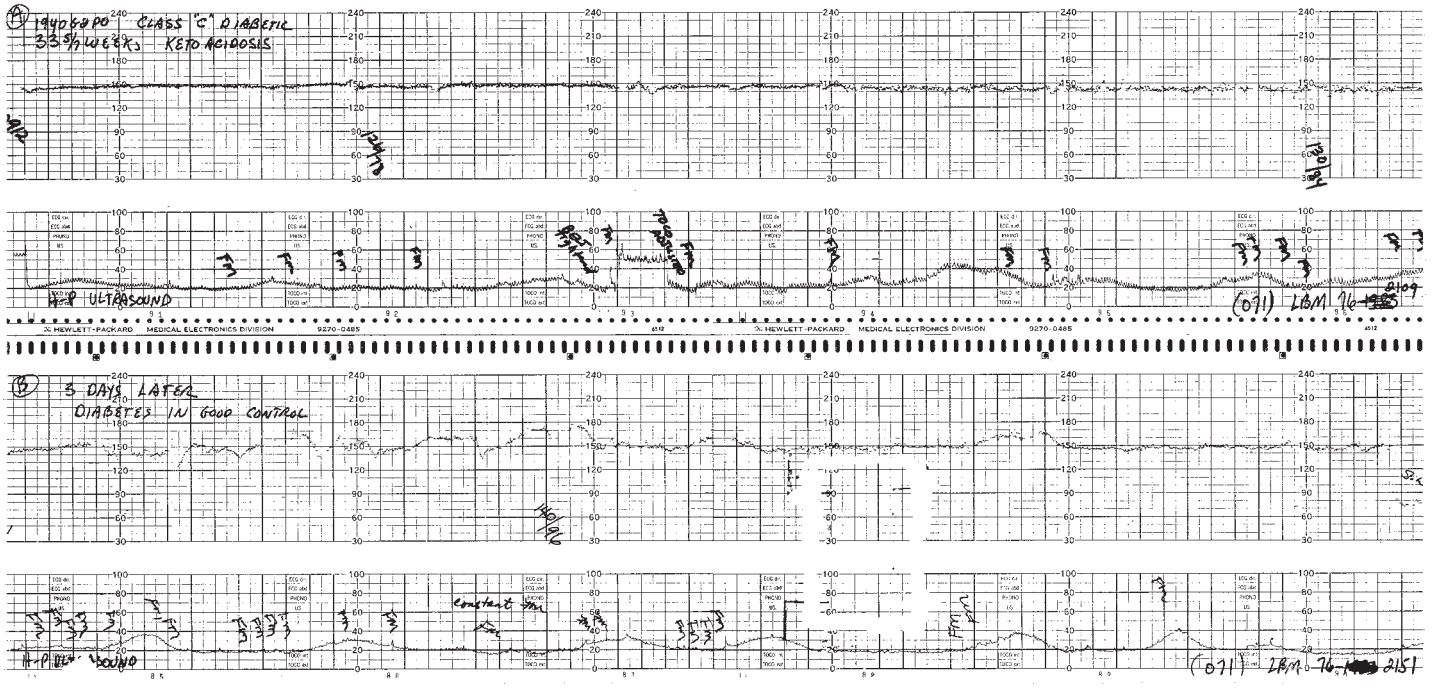

Figure 12.5. This case illustrates the effect of ketoacidosis on the fetus. **Panel A** is the fetal heart rate (FHR) tracing of a diabetic admitted in ketoacidosis at 33½ weeks. Note the nonreactive pattern. Three days later **(panel B)**, with the patient euglycemic and nonketotic, a normally reactive spontaneous contraction stress test result was seen. This illustrates the potential reversibility of FHR patterns when treatable conditions exist.

not excessive and there is no maternal coagulopathy, then fetal condition becomes the next concern. With, and subsequent to, any bleeding episode, continuous fetal monitoring is indicated. Occasionally, even with only minimal uterine activity, late decelerations can alert the physician to a significant abruption (Fig. 12.6). In premature gestations, especially those with contractions, even a small amount of bleeding is abnormal and should alert the clinician to the possibility of an underlying etiology. The characteristic picture of abruptio placentae on the fetal monitor are late decelerations and a tachysystolic uterine contraction pattern. One, both, or neither may be present and may or may not correlate with the apparent size of the abruption. With a major bleeding episode, a normal FHR tracing is an essential variable in the decision to attempt tocolysis or allow a trial of labor.

Once the acute episode is resolved and a reasonable time (usually at least 24 hours) of continuous fetal monitoring has provided reassurance, the patients can revert to intermittent antepartum testing. Our mainstay of testing for preterm gestations complicated by vaginal bleeding is the modified BPP. It does not appear that patients with uncomplicated placenta previa have an increased risk of UPI. Markers of UPI, such as IUGR and stillbirth rates, do not appear increased in those with placenta previa. Thus, it may not be necessary to test these patients unless they have an acute bleeding episode. Generally, uterine bleeding before term is a relative contraindication to performing a CST. Often, these patients have sufficient uterine activity for a spontaneous CST. With a nonreactive NST, the BPP should be the primary backup test in these patients. Patients with significant third- or late second-trimester bleeding are observed until fetal pulmonary maturity is documented, usually at or beyond 36 to 37 weeks' gestation, and then delivery is effected.

Hemoglobinopathy/Severe Anemia/Cyanotic Cardiac and Pulmonary Disease

Patients with substantially decreased effective oxygen-carrying red blood cells or with other reasons for decreased oxygen delivery to the placenta (e.g., cyanotic maternal cardiac disease or pulmonary disease) can have a form of preplacental UPI. Such UPI may lead to growth restriction and/or hypoxic compromise. Patients with sickle cell disease, severe anemia (hematocrit <25%), or chronic maternal hypoxemia should be observed during pregnancy, with fetal surveillance starting at 26 to 28 weeks. Sudden deterioration in maternal status, such as a sickle cell crisis or an acute asthmatic exacerbation, necessitates immediate FHR monitoring. Treatment of the maternal condition with oxygen therapy or transfusion may improve ominous FHR patterns (Fig. 12.7). If the clinical condition is stable and antepartum tests remain normal, these patients may be delivered after 36 to 38 weeks, with pulmonary maturity.

Suspected Intrauterine Growth Restriction

Even in patients without other risk factors, a lag in fundal growth or ultrasound findings may suggest IUGR. The ability to correctly diagnose IUGR depends on a high level of suspicion and accurate ultrasound measurements of different fetal parameters over time. Although the predictive value of any single parameter varies dramatically, the sensitivity of multiple ultrasound measurements in ruling out IUGR is very high. A normal ultrasound in a suspected case is very reassuring, as is the finding of normal interval growth. Umbilical artery Doppler may also be helpful in such cases (Fig. 12.8). Doppler may help both diagnostically, as IUGR from UPI may be associated with an index of increased resistance to flow in the umbilical artery. As a prognostic test, absent or reversed diastolic flow in the umbilical artery often portends the need for early delivery.

The clinical management of suspected IUGR is controversial. The primary issue centers around the timing of delivery. The issue is whether such patients should be delivered as soon as IUGR is severe and the fetus has a likelihood of surviving in the nursery versus as soon as pulmonary maturity can be documented versus using antepartum testing and only delivering these fetuses early with abnormal surveillance. The basis of this issue is the unanswered question over whether the nutritional UPI with resultant IUGR alone causes the neurologic damage often seen in these newborns or if intervening before hypoxic/asphyxial damage occurs is enough to avoid these injuries. If IUGR is suspected and delivery is not elected, some form of antepartum testing should be instituted. If testing is normal, expectant management with close follow-up of *in utero* growth should be continued until the decision for delivery is made.

Discordant Twins

Occurring in both monozygotic and dizygotic twin gestations, discordant growth is usually a form of growth restriction. The etiology may be typical UPI in one fetus either as a result of some maternal condition or from the other fetus being more successful in achieving adequate placental perfusion. Alternatively, in monozygotic twins, the discordance is often due to twin-to-twin transfusion, and there will be oligohydramnios in the smaller twin and polyhydramnios and even hydrops fetalis in the larger one. Ultrasound is the primary means of suspecting such a problem *in utero*, and chorionicity must be established in early gestation for all multiples. Serial ultrasounds are then employed to assess fetal growth and AF volume. When using the NST for fetal surveillance in dizygotic twins, the only yield has been in patients whose twin pregnancy is complicated by discordancy. If ultrasound does not suggest a 20% to 25% or greater difference in estimated fetal weight between the twins, or IUGR in one or both, antepartum testing is not indicated. Just as with singleton pregnancies, fetal testing in multiple gestations

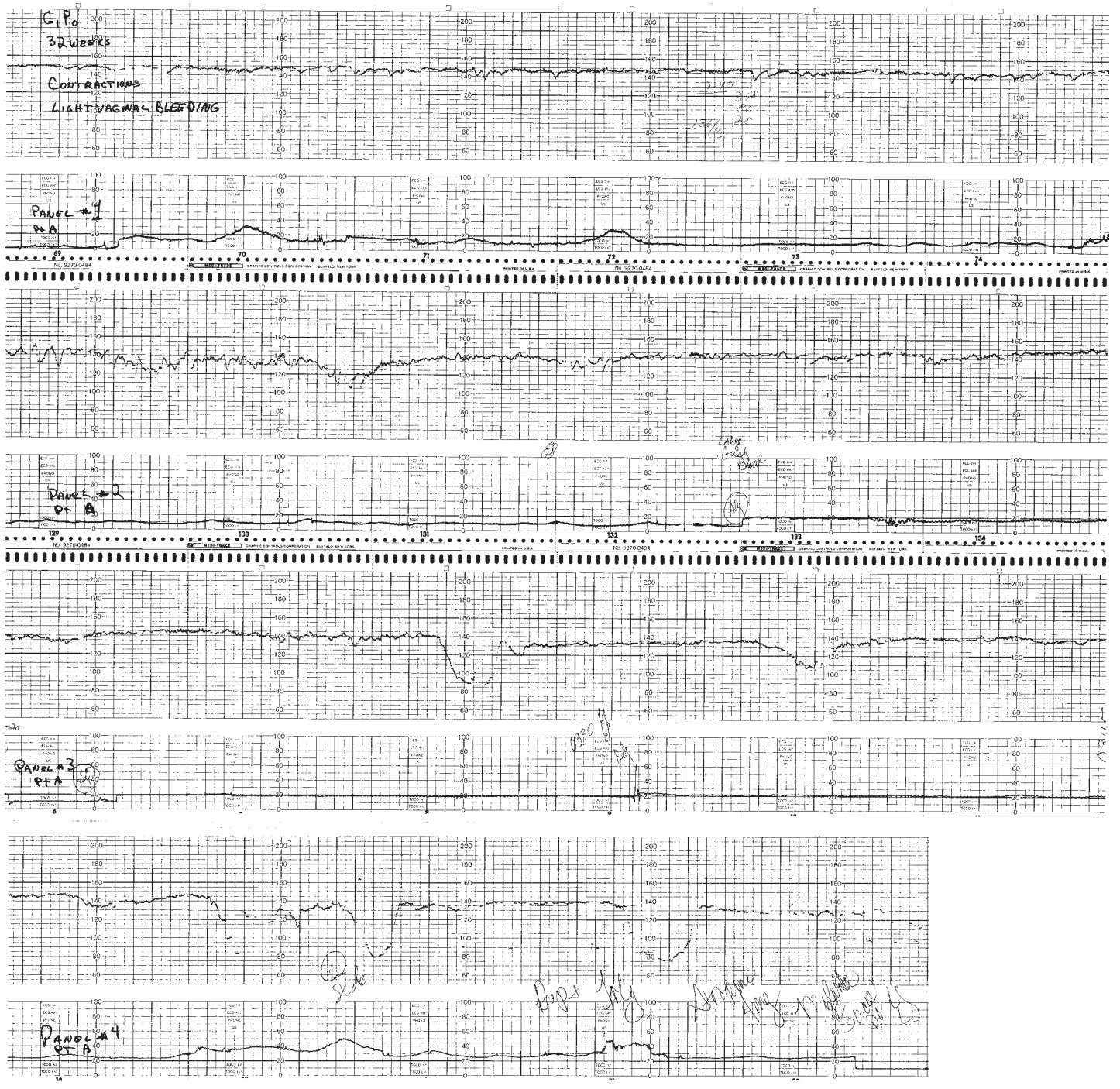

Figure 12.6. A gravida 1 was admitted at 32 weeks with mild uterine activity and light vaginal bleeding. The uterus was soft between contractions and nontender. Vital signs were normal. External monitoring revealed irregular uterine contractions that were difficult to record. At the **second panel**, the patient had a large gush of blood. Late decelerations are seen on the **second, third**, and **fourth panels.** Immediate cesarean section produced a 1,000-g female with Apgar scores of 1 at 1 minute and 6 at 5 minutes; the infant had moderate respiratory distress syndrome but subsequently did well. A 30% to 40% placental abruption was found. This case illustrates how fetal monitoring may be a sensitive indication of significant abruption.

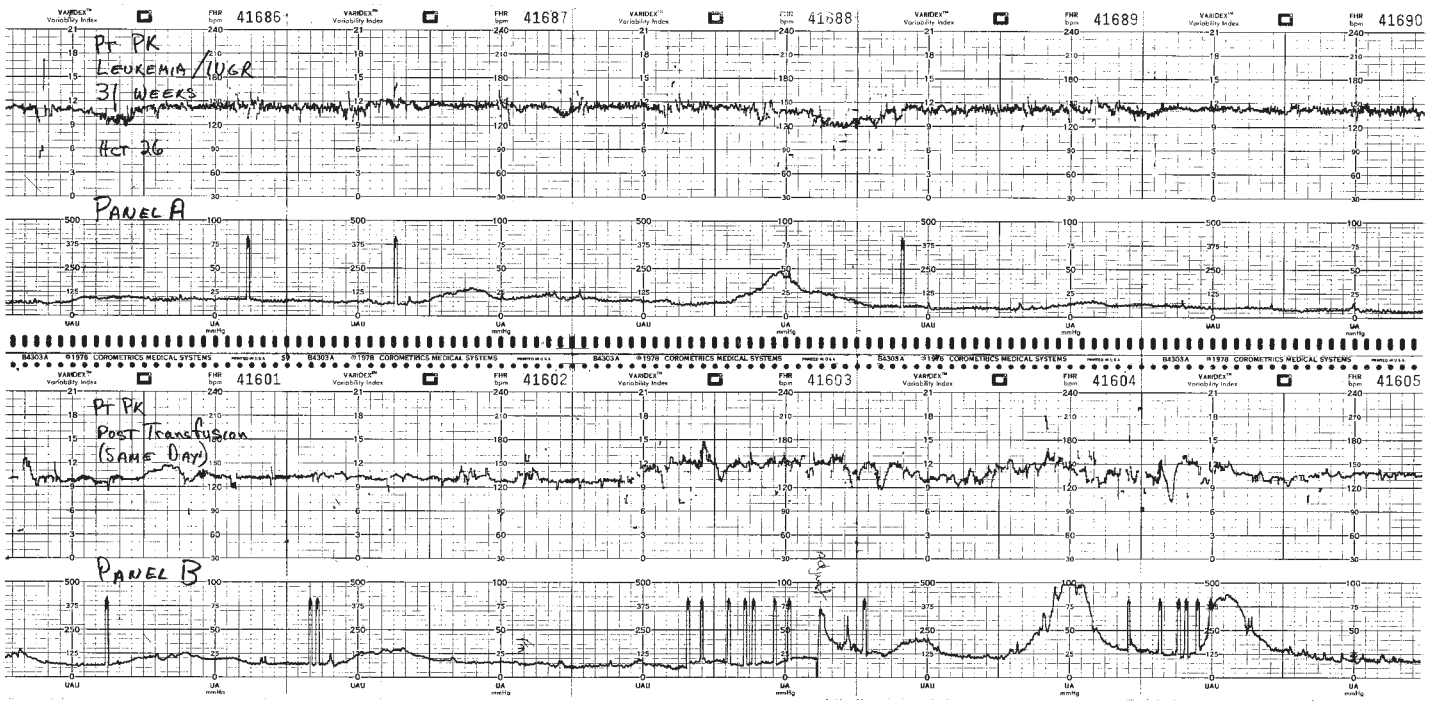

Figure 12.7. This 25-year-old gravida 1 para 0 presented with acute lymphocytic leukemia and suspected intrauterine growth retardation. At 16 weeks, the leukemia exacerbated, and chemotherapy was reinstituted. Uterine growth was lagging from 20 weeks. The patient was anemic secondary to bone marrow suppression. The initial nonstress test (NST) performed at 31 weeks **(panel A)** was nonreactive, and a contraction stress test (CST) was done with a positive result. After transfusion of 2 units of packed red blood cells, the test was repeated **(panel B)**. This is now a reactive NST result with a negative CST result. The patient did well until 37 weeks when the CST result was again positive, and she was delivered of a somewhat growth-restricted but otherwise normal female. Unfortunately, the mother died 18 months later from pneumonia secondary to a recurrence of her leukemia.

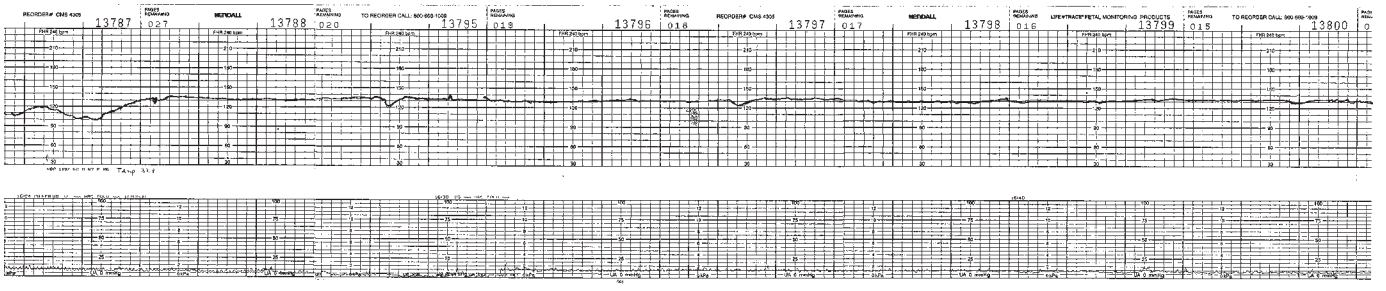

Figure 12.8. This patient was a 19-year-old gravida 1 para 0 at 26 weeks' gestation. She had a history of arthritis and laboratory abnormalities suggestive of possible systemic lupus erythematosus. An ultrasound done at the time of the initial consultation revealed a symmetrically growth-restricted fetus and an umbilical cord Doppler with reversal of end-diastolic flow. She was sent immediately to labor and delivery for fetal monitoring. The tracing was flat with spontaneous decelerations. The biophysical profile was 2 of 10 for fluid only. She underwent immediate cesarean section delivering a 526-g male with Apgar scores 6 at 1 minute and 6 at 5 minutes and cord gases of arterial 7.06 BE −12, venous 7.18, and BE −8. The baby ultimately survived and is developing normally at age 2.

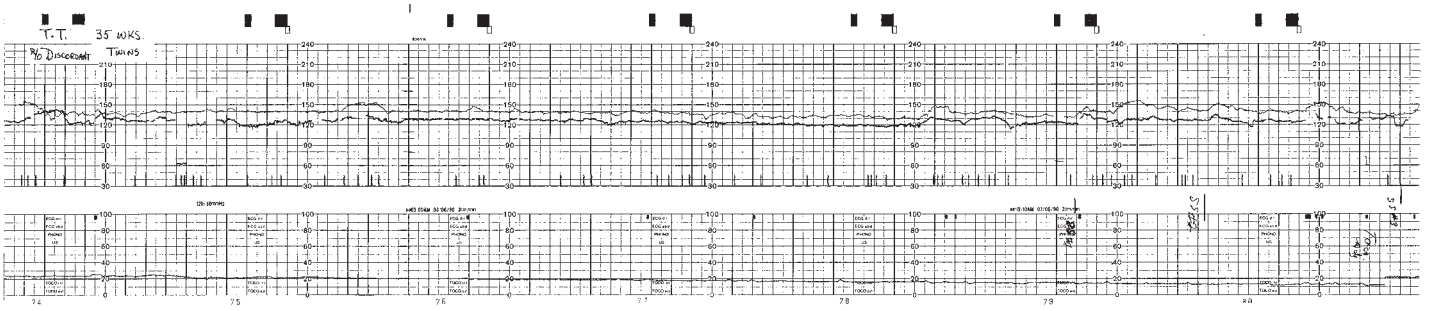

Figure 12.9. This patient had a twin gestation at 35 weeks with suspected discordancy. Biweekly nonstress tests were performed. Simultaneous monitoring of both fetuses is now possible using external Doppler. It is interesting to observe the degree of concordance of accelerations between the twins as well as similar changes in baseline.

should only be started at a gestational age when intervention might be entertained (Fig. 12.9). The finding of discordant growth in twins or triplets is not rare, but intervention in the very premature gestation for an abnormal FHR of the small fetus can only be supported after careful assessment of the other sibling(s) *in utero*. At times, decisions about intervention are perhaps medically indicated, but ethically, a major dilemma, in that intervention, will result in undue prematurity for the normally grown sibling fetus(es) without evidence of compromise.

One highly unusual condition is monoamniotic twins. The fetal mortality in such twins is approximately 50% and occurs primarily from cord entanglement but also can result from the other complications, such as twin-to-twin transfusion, seen in diamniotic monozygotic twins. Surveillance and timing of delivery in these patients is highly controversial as they are rare, <1% of all twins, and most reports of current management are based on very small series (29). Once viability is reached, these patients are usually hospitalized and monitored with daily NSTs, looking primarily for FHR findings suggestive of progressive umbilical cord entanglement with resultant intermittent compression/interruption of cord blood flow. If evidence of significant variable decelerations or occasional prolonged decelerations exists, continuous fetal monitoring may be warranted. Delivery may be for either nonreassuring/worsening evidence of cord compression or when the fetus reaches 34 weeks (30).

Decreased Fetal Movement

Not uncommonly, patients without other risk factors will complain of decreased or absent fetal movement. Clearly, all patients should be counseled to monitor fetal movement with some method beginning in the third trimester. Because 50% of stillbirths may come from the 85% of patients who are not at risk, fetal movement counting may be the only way to have an impact on this larger group. Although usually not a cause for true alarm, decreased fetal movement may be the only sign of impending fetal death in an otherwise normal pregnancy. In counseling patients with this concern, the first step should be to ask the patient to lie down on her side for 30 to 60 minutes and concentrate on counting movements. If she feels three or more movements in that time, she should be reassured. If the patient feels less than three movements in an hour and is beyond 26 weeks' gestation, the patient should be instructed to come in as soon as possible for an NST. An NST should not be postponed until the next day for a patient with this complaint. If the NST is reactive, no further testing is necessary if the patient has no other risk factors and the test does not need repeating. If decelerations are present and/or the tracing is nonreactive, further assessment is indicated immediately. A rare but very serious etiology for loss of fetal movement is fetal to maternal hemorrhage. Such a diagnosis should be considered in patients with this complaint of loss of fetal movement when there is a nonreactive or suspicious NST with spontaneous decelerations. A sinusoidal heart rate is not always present in such cases.

It is also important for high-risk patients to monitor fetal movement every day. Should a high-risk patient report decreased fetal movement, the same policy as described above is followed, regardless of when the last fetal surveillance was performed. It is of interest to note that the only type of prospective study employing some form of fetal surveillance that has been shown to be of significant value in decreasing antepartum stillbirth is one comparing patients instructed in monitoring fetal movements daily with a group not noting daily movements (31).

Premature Rupture of Membranes

Approximately 10% to 15% of pregnancies are complicated by preterm premature rupture of the membranes (PROM), which accounts for 30% of premature deliveries (31). Although controversy exists over the optimal management for this complication in the term gestation (i.e., expectant management versus oxytocin induction, with or without allowed latency period), there is continued disagreement over the management of this complication in the preterm gestation. Most

schemes of expectant management include the supplemental use of some means of fetal surveillance. Concern regarding infection, cord accidents, and abruption justifies the efforts to use frequent fetal monitoring in women with this complication. In pregnancies complicated by PROM that are being managed expectantly, we perform daily NST monitoring for 1 hour. The FHR baseline and the presence or absence of accelerations and decelerations are specifically noted. Variable decelerations, suggesting possible cord compression secondary to decreased AF volume, are not uncommon and may indicate early labor or fetal distress (32). Furthermore, from studies using ultrasound, there appears to be a relationship between the degree of oligohydramnios and the incidence of variable decelerations in patients with preterm PROM (33).

Vintzileos et al. (34), using a daily fetal BPP in patients with PROM followed serially, found a relationship between progressively lower BPP scores and findings suggestive of infection in the fetus. However, these patients will also have a nonreactive NST. A randomized trial comparing daily NST with the daily BPP by Lewis et al. concluded that the NST was equivalent to the BPP in this situation and significantly less time-consuming and more cost-effective except in babies <28 weeks where nonreactivity was common due to early gestational age (35). Thus, the NST is an appropriate screen for cord compression and fetal infection. Because of these significant maternal and fetal risks in patients with preterm PROM, careful and frequent determination of fetal well-being is critically important when expectant management is elected. Our patients with PROM and a nonreactive NST result have a BPP performed as their backup test.

MANAGEMENT OF PATIENTS WITH NONREASSURING TESTS

Nonreactive Nonstress Test

The NST should be viewed as a screening test when used as the primary means of antepartum testing. Nonreactive patterns may be caused by fetal hypoxia, previous central nervous system (CNS) injury, anomalies, sleep states, or CNS-depressant drugs. In performing the NST, if there are not two or more 15 beats per minute (BPM) accelerations above the baseline FHR within the initial 20 to 30 minutes, stimulation of the fetus with uterine manipulation or sound (Table 12.2) is indicated. Another 20-minute period is then monitored, and if the fetus is still nonreactive, an alternative form of assessment (backup test) should be done immediately. In this setting, we have historically used the CST if the NST is nonreactive. Alternatively, a BPP may be done. If the fetus becomes reactive during the CST, it may be discontinued or completed but will be interpreted as any other reactive tracing (Fig. 12.10).

TABLE 12.2 Acoustic stimulation test

Patient in left lateral recumbent position
External monitor of FHR and contractions
Establish baseline FHR for 5–10 min
Apply artificial larynx over fetal head and stimulate for 1 s
Restimulate if no acceleration within 10 s (repeat up to four times)
Continue to monitor for 15 min following accelerations

FHR, fetal heart rate.

An NST that remains nonreactive and is followed by a negative CST result or an otherwise normal BPP may be repeated in 1 week. In these cases, there is an increased incidence of fetal anomalies. In addition, consideration should be given to the possible use of sedative drugs (e.g., phenobarbital, illicit depressant drugs, or antihypertensive medications such as beta-blockers) (36). A very careful review of the test for subtle late decelerations is critical, because the combination of true nonreactivity with a negative CST is unusual and should heighten one's clinical suspicion (Fig. 12.11).

AMNIOTIC FLUID VOLUME

In doing the modified BPP, often, the NST result will be reactive but the AFI abnormal. AFIs in the borderline range (5 to 8 cm) are not an indication for delivery, but the AFI should

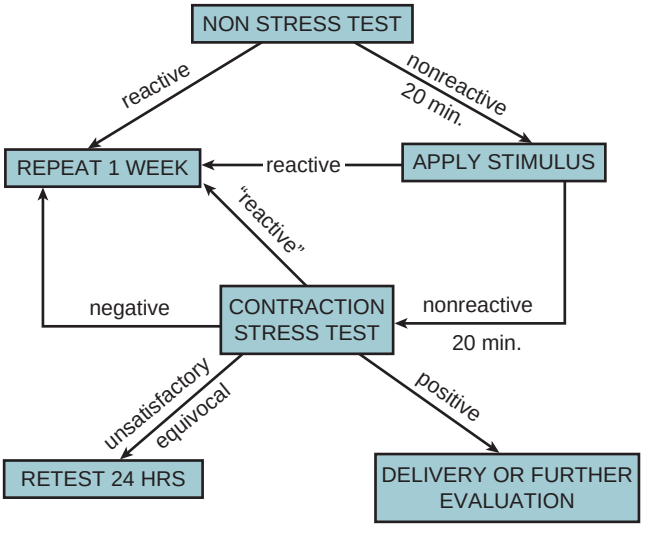

Figure 12.10. Outline for conducting the nonstress test. (From Evertson LR, Gauthier RJ, Schifrin BS, et al.: Antepartum fetal heart rate testing. I. Evolution of the nonstress test. *Am J Obstet Gynecol* 133:31, 1979, with permission.)

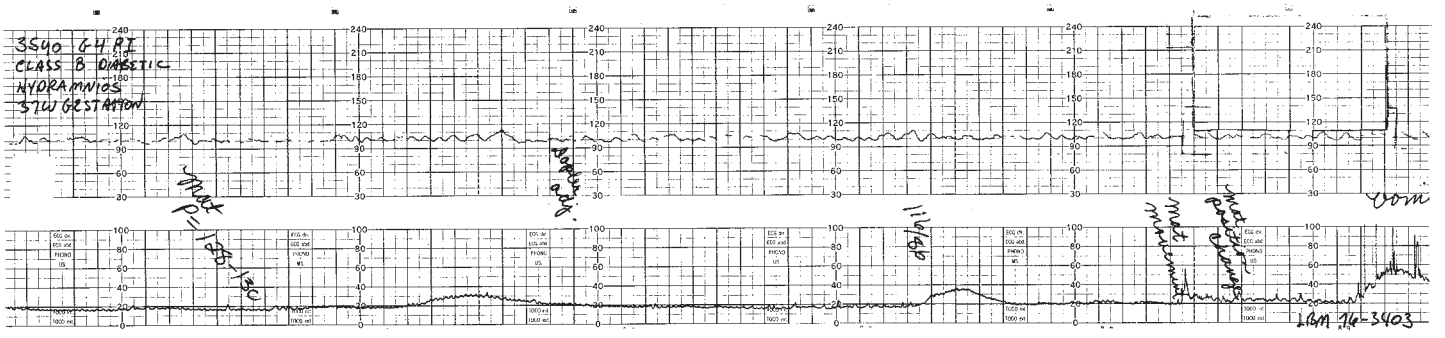

Figure 12.11. Nonreactive negative oxytocin challenge test done in a diabetic with hydramnios. There is a baseline bradycardia of 100 beats per minute. No late decelerations are seen, but no accelerations are seen. This unusual pattern occurred in a patient who later delivered a baby with multiple congenital anomalies including congenital heart disease, meningomyelocele, and hydrocephaly.

be repeated at least twice weekly in these patients. An AFI 5 cm or less is abnormal and requires careful consideration of fetal condition. The first important issue to realize is that the AFI may be considerably dynamic. As many as 30% to 40% of AFIs in the 3- to 5-cm range will be >5 cm in 24 hours (8). Thus, except in the postdate pregnancy where virtually any abnormality warrants delivery, consideration should be given to repeating the test in 24 hours before deciding on delivery. An AFI 3 cm or less in the term patient is usually an indication for delivery. In the 33- to 36-week gestational ages, consideration of amniocentesis and delivery if maturity is found is reasonable. Before 33 weeks, testing frequency may be increased or a CST considered if such a finding occurs and delivery reserved for patients who have additional nonreassuring FHR patterns, a nonreassuring BPP, or other additional indications (Fig. 12.12).

Equivocal Tests

In doing an NST, or with a spontaneous CST or an oxytocin challenge test (OCT), any late deceleration, significant variable deceleration (deceleration dropping more than 15 BPM lasting more than 15 seconds), and/or prolonged decelerations are often seen, even when the fetus is reactive,

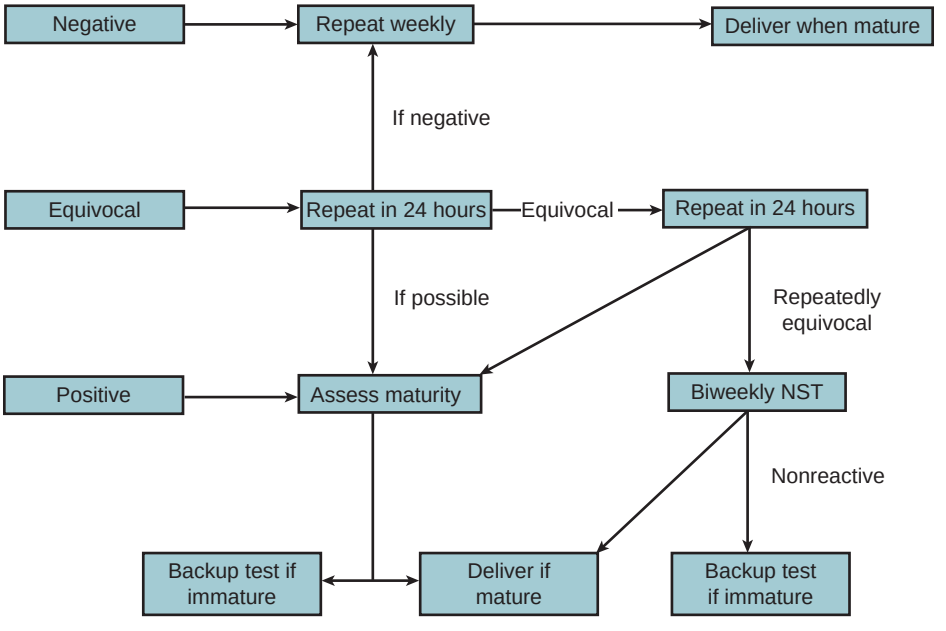

Figure 12.12. Contraction stress test management protocol.

and require further evaluation or repeat testing. When this occurs with an NST, a backup CST or BPP may be performed and, if reassuring, the test repeated the next day. With the CST, approximately 20% to 30% of test results are equivocal. An equivocal CST only suggests that the test cannot be used to be sure that the fetus can be safely left alone for 1 week. There are basically two types of equivocal CSTs: the equivocal hyperstimulation and the equivocal suspicious. The equivocal hyperstimulation is seen in a CST that has a late deceleration associated with a prolonged contraction or contractions more frequent than every 2 minutes. The equivocal suspicious test has late decelerations with less than half of the uterine contractions that are not prolonged or of high frequency. With an equivocal test result, we require that the test be repeated the next day or have an immediate backup test. Equivocal tests are rarely indications for delivery, although the repeatedly equivocal test in the postterm patient or significantly complicated pregnancy (e.g., diabetes) may warrant delivery.

Positive Contraction Stress Test Results

As previously stated, positive CST results have a high correlation with fetal and neonatal morbidity and even with fetal mortality if not acted on. However, there is a high rate of false-positive test results. In a premature gestation, this is an important consideration when making decisions about delivery. A term or preterm mature fetus with a positive CST result should be considered for delivery (Fig. 12.12). Although fetal pulmonary maturity does not ensure that the newborn will not have other complications of prematurity, lung maturity is generally the limiting factor in normal survival of the premature neonate. Furthermore, if hypoxic UPI is allowed to persist and a depressed newborn is delivered, many complications of prematurity are more likely, including respiratory distress syndrome, even with a mature lecithin-to-sphingomyelin ratio (37). Therefore, a reactive positive CST result is generally an indication for an amniocentesis in preterm gestation (32 to 36 weeks). In a well-dated pregnancy at or beyond 37 weeks, however, delivery is indicated following a positive CST result without need for documentation of fetal pulmonary maturity (Fig. 12.13). With an immature fetus, a reactive positive CST requires further evidence of fetal jeopardy before intervention is justified. Daily testing with NST and/or BPP may be appropriate in these immature fetuses with a reactive positive CST result (Fig. 12.14).

For the fetus with a persistently nonreactive positive CST result, the likelihood of a false-positive result is very low, unless the absence of reactivity is due to immaturity. Therefore, at or beyond 28 to 30 weeks, a nonreactive positive test result, where the nonreactivity continues for at least 90 minutes, justifies delivery. In the more premature fetus, we have successfully used BPP testing and usually repeat these daily. Daily BPP testing in these situations has allowed us to gain time to attempt acceleration of pulmonary maturity with corticosteroids or delay delivery for several days to weeks (38).

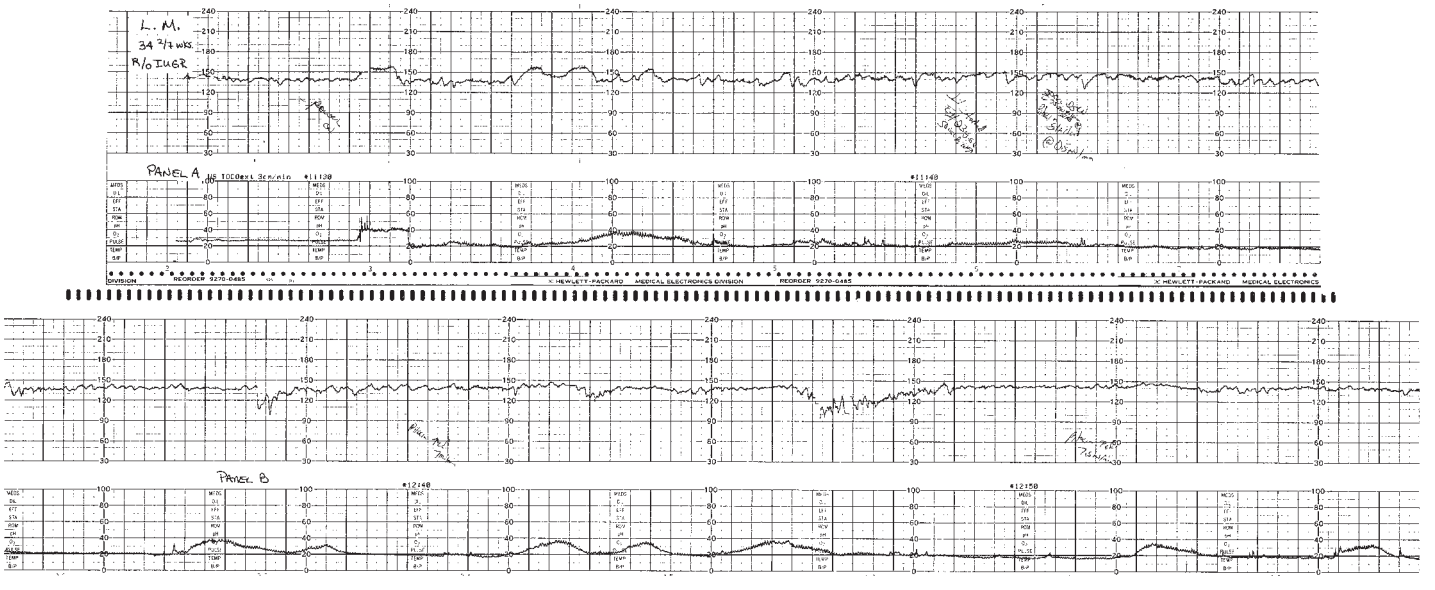

Figure 12.13. The history of this gravida 3 para 0 included a stillborn at 34 weeks. The etiology of the stillbirth was unknown. This pregnancy was further complicated by possible intrauterine growth retardation. During the initial portion of the contraction stress test (**panel A**), the fetus was reactive. However, persistent late decelerations appeared with contractions (**panel B**). Because of this positive test result, an amniocentesis was performed revealing meconium-stained amniotic fluid with a lecithin:sphingomyelin ratio of 2.6. Delivery was accomplished by cesarean section because of persistent late decelerations in labor. The infant did well following delivery.

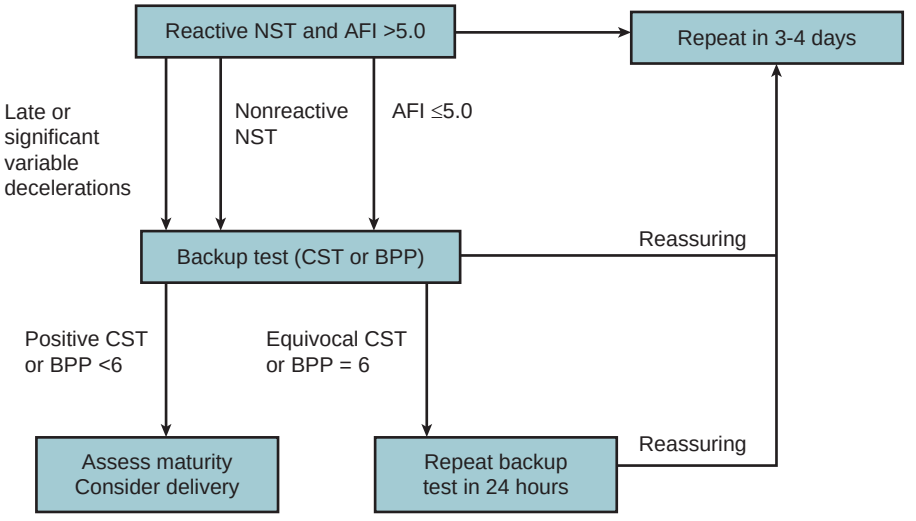

Figure 12.14. Modified biophysical profile: nonstress test with amniotic fluid index.

ABNORMAL BIOPHYSICAL PROFILE

When using the BPP, either as primary surveillance or as backup to the nonreactive or equivocal CST result, the response should be, as with the CST, based on the entire clinical situation. A BPP of 8 or 10 is normal and requires repeating in 3 to 4 days with an NST, or sooner if there were decelerations or oligohydramnios. A BPP of 6 is equivocal and usually requires repeating the next day. If part of the reduction in BPP score is due to decreased AF volume, the management will be as outlined in the previous section on AF volume. A BPP of 4 may be treated similarly to the reactive positive CST, with delivery warranted for term fetuses and those with mature lung profiles, and if delivery is not undertaken, either continuous monitoring or repeat BPP in 6 hours. Finally a BPP of 0 to 2 is almost always an indication for delivery in the viable fetus. The only exception may be the very premature gestation (e.g., ≤27 weeks), where the decision for delivery should never be undertaken unless all tests indicate an absolute need, and thus, the addition of the nonreactive positive CST result and the BPP of 0 to 2 would be enough to warrant delivery at this time in gestation (39) (Table 12.3).

Choosing the Route of Delivery

Once the decision has been made to intervene on behalf of the fetus, the route of delivery must be considered. The presence or absence of reactivity is definitely a factor in a patient's ability to tolerate labor following a positive CST result (40,41). Virtually all patients with persistently nonreactive positive CST results have persistent late decelerations in labor. On the other hand, when accelerations are present, 50% of patients will tolerate labor without persistent late decelerations.

In performing the induction, the patient should be placed on her left side to maximize uterine perfusion. Oxygen should be administered by mask. Membranes should be artificially ruptured from the start of the induction, FHR monitored by scalp electrode, and the contractions monitored with an internal pressure transducer. Oxytocin is administered with special care to avoid uterine tachysystole. Should late decelerations persist despite all these measures, the patient should be delivered by cesarean section. The patient with an unfavorable cervix and/or nonvertex presentation can be delivered by cesarean section without a trial of labor. Additionally, patients with positive CST results with no accelerations present should be delivered by cesarean section without a trial of labor, as it is very unlikely that these fetuses will tolerate labor.

Finally, a note that is partly philosophy and partly good practice. Once the decision has been made to intervene, proceed without undue delay. This is not a delivery reasonably put off until the next day. While preparing for cesarean section with a delay for blood cross-match, mobilization of a surgical team, or whatever reason, the fetus should be continually monitored until the time of the abdominal preparation in the operating room. This is not a situation where a 30-minute rule for delivery after the decision is made exists. The fetus with a nonreassuring antepartum test result does not have the ongoing stress of labor. However, it is a situation that requires integration of all the variables available in making a decision regarding the urgency of delivery. For example, a patient who comes in with vaginal bleeding and a nonreactive baseline with late decelerations even with occasional contractions probably has an abruption and needs delivery urgently. Alternatively the 41-week fetus with the nonreactive test result that becomes reactive during an OCT that is positive and has an unfavorable cervix should probably be delivered by cesarean section that same day, but the urgency is much less. No cookbook statements can be made regarding how soon these patients should be delivered and clinical judgment is key.

TABLE 12.3 Biophysical profile scoring, management protocol

Score	Interpretation management
10	Normal infant, low risk. Repeat testing at weekly intervals for chronic asphyxia. Repeat twice weekly in diabetics and patients ≥42 weeks' gestation.
8	Normal infant, low risk. Repeat testing at weekly intervals for chronic asphyxia. Repeat testing twice weekly in diabetics and patients ≥42 wk. Oligohydramnios is an indication for delivery.
6	Suspect chronic asphyxia. Repeat testing in 24 h. Deliver if oligohydramnios is present.
4	Suspect chronic asphyxia. If ≥36 wk and favorable, then deliver. If <36 wk and L/S = 2.0, repeat test in 6 h. If repeat score ≤4, deliver.
0–2	Strong suspicion of chronic asphyxia. Extended testing time to 120 min. If chronic asphyxia persistent score ≤4, deliver regardless of gestational age.

L/S, amniotic fluid lecithin:sphingomyelin.
From Manning FA, Baskett TF, Morrison I, et al.: Fetal biophysical profile scoring: a prospective study in 1,184 high-risk patients. *Am J Obstet Gynecol* 140:289, 1981.

SUMMARY

Managing the antepartum patient is a difficult and challenging problem. Decisions regarding intervention have potentially grave consequences and should never be made on one parameter alone without knowledge of gestational age, fetal maturity, and maternal condition. The goal should be to deliver a healthy neonate as near term as possible by the safest route for mother and fetus. Antepartum heart rate monitoring can contribute significantly toward this goal if understood well and used appropriately. Equivocal tests are rarely an indication for delivery, and unnecessary inductions may result in unnecessary cesarean sections, or worse yet, unnecessary premature delivery that may have dire neonatal and long-term consequences. One other point to make is that no matter what means of surveillance is chosen, remember that negative tests are associated with a very low likelihood of adverse outcome. Positive test results should be thoroughly evaluated to avoid unnecessary premature intervention. Therefore, we recommend the use of backup tests as assistance in making the decision whether to deliver, rather than relying on one test, particularly in very premature gestations (Figs. 12.11–12.13).

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CHAPTER 13

Fetal Heart Rate Patterns Associated with Fetal Central Nervous System Dysfunction

For some time it has been known that fetal heart rate (FHR) patterns preceding hypoxic fetal death *in utero* have characteristics that are not usually found in well-oxygenated fetuses or in fetuses with hypoxia that is not severe and/or prolonged (1–6). Martin et al. (7) have shown that with progressive fetal hypoxemia, late deceleration is first produced by a central nervous system (CNS) reflex that can be inhibited by autonomic blockade. When hypoxia advances to a point where significant fetal acidemia occurs, however, FHR variability disappears and one can no longer inhibit late deceleration with autonomic blockade. We therefore know that severe hypoxemia and acidosis are capable of altering CNS responsiveness and that this is reflected in the FHR pattern. Similarly, it is well established that fetuses with severe nonhypoxic CNS abnormalities may have FHR patterns that differ from those in babies with intact CNSs (8–12). Because the ultimate goal is the prevention of fetal CNS damage from intrauterine hypoxia, we must examine what we have learned about FHR patterns associated with CNS dysfunction. Furthermore, recent research shows that maternal infection with chorioamnionitis and funisitis may result in cytokine-mediated CNS dysfunction and damage (see Chapter 3), and it is our recent experience that FHR patterns found in this condition may also reflect CNS dysfunction without preceding FHR patterns known to be associated with fetal hypoxia. FHR monitoring cannot truly determine whether preexisting brain damage was present at the time fetal monitoring was initiated. The patterns described in this chapter, fortunately, are not common and have not been studied in a systematic fashion, and we therefore depend on somewhat anecdotal accounts.

When the modulatory function of the CNS is impaired, a lack of variability is the most common effect seen in the FHR pattern (10). However, other changes are observed when CNS control is impaired (13). Many of these changes are also seen in very premature fetuses in which, presumably, the brain is less well developed. They can also occur when the mother has taken drugs that affect the fetal brain (14–19). Interestingly, FHR evidence of CNS dysfunction in anencephalic fetuses varies with the level of the defect, from normal to severely abnormal (8,20). It would appear that when a sufficient amount of cerebral cortex and midbrain is present in anencephalic fetuses, FHR variability may be normal and accelerations may be present. However, with no midbrain or cortex present, variability is absent. When the fetus has a complete heart block or when there is a supraventricular fetal tachycardia caused by an ectopic pacemaker not under CNS control, there will also be a complete lack of variability (10).

As more experience is gained with FHR pattern observation in fetuses that subsequently show CNS damage as neonates, it is increasingly clear that the patterns preceding birth are often more characteristic of a lack of CNS control than of ongoing hypoxia. Certainly, these same characteristics are seen in fetuses with CNS dysfunction resulting from acute ongoing hypoxia, and fetuses with chronic oxygen deprivation may also show evidence of CNS dysfunction in the FHR prior to labor. Therefore, while we recognize that preexisting CNS insults or abnormalities may produce changes in the FHR indicating CNS dysfunction, these same changes may accompany FHR patterns indicating ongoing hypoxia (late deceleration or severe variable deceleration); it is not possible to determine the degree of CNS dysfunction that is preexisting and the degree of dysfunction that is due to an ongoing hypoxic process. It should also be pointed out that not all fetuses with CNS abnormalities will have signs of CNS dysfunction on the FHR monitor strip.

The following FHR patterns are evidence of fetal CNS dysfunction:

1. Flat FHR (4,6)
2. Blunted patterns (4,6,21,22)
3. Unstable baseline (1)
4. Overshoot (23)
5. Sinusoidal patterns (1,24)
6. "Check mark" pattern (25)

FLAT FETAL HEART RATE

When CNS dysfunction is present, there may be virtually no fluctuation in the FHR pattern (Figs. 13.1–13.6). This change does not occur in cycles lasting 20 to 40 minutes, as are observed in cases of fetal state change. We are often restricted to external FHR recordings and, as a result, cannot be absolutely sure that short-term variability is absent; however, with the newer autocorrelation method (see Chapter 4),we can get a better idea of short-term variability. Long-term variability (three to five cycles per minute) is reduced or absent, and if present, takes on a very smooth shape (Fig. 13.4).

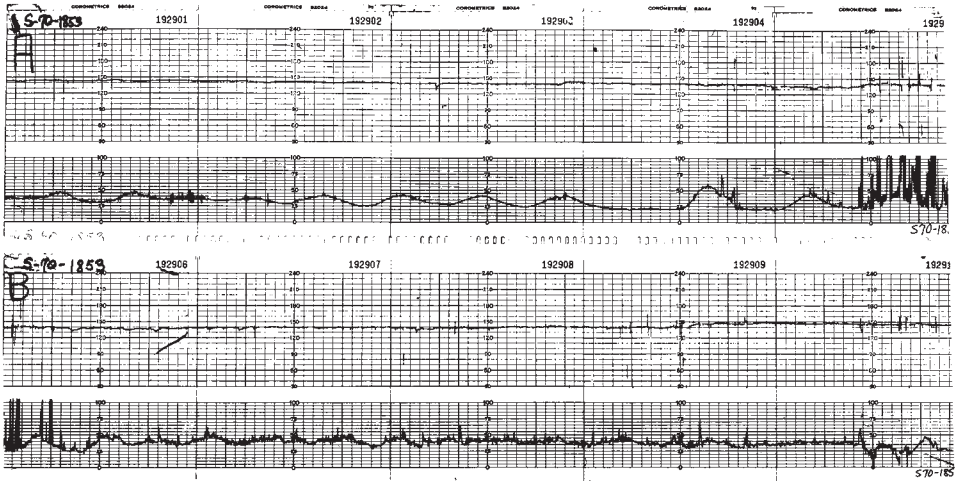

Figure 13.1. A flat fetal heart rate pattern with no periodic changes. It could be due to a preexisting central nervous system (CNS) abnormality, drugs, or, occasionally, a fetus with CNS dysfunction and ongoing hypoxia.

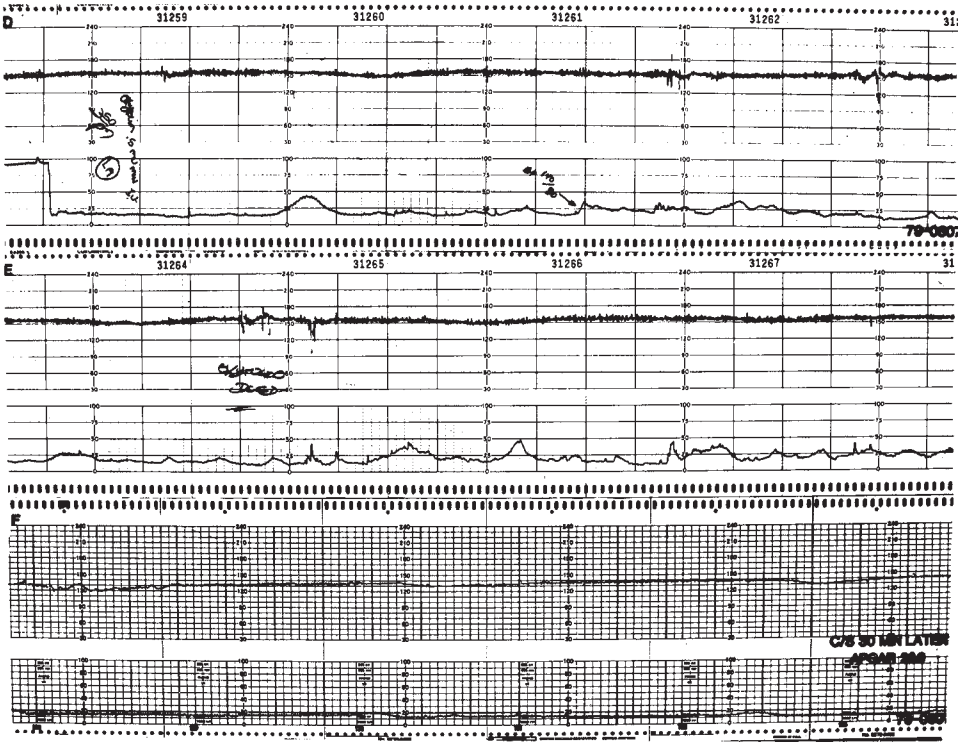

Figure 13.2. A flat fetal heart rate tracing with associated subtle late decelerations, indicating the probability of ongoing hypoxia. This fetus was found to have metabolic acidosis, a low Apgar score, neonatal seizures, and subsequent evidence of cerebral palsy.

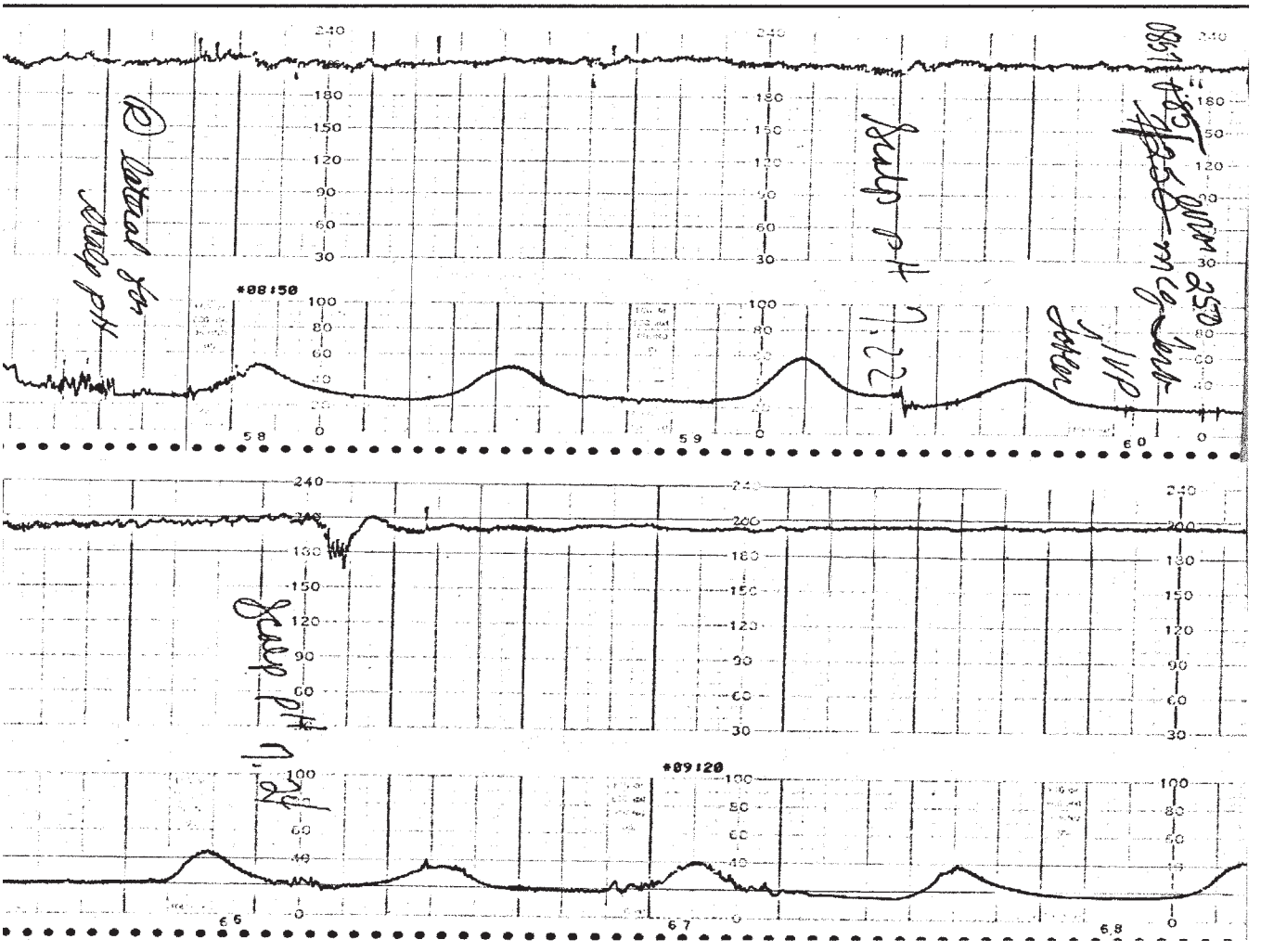

Figure 13.3. This tracing shows fetal tachycardia associated with maternal chorioamnionitis. Note the pH of 7.22, and the cord pH was in the same range. The neonate developed encephalopathy and now has cerebral palsy. This may be an example of the fetal inflammatory response to maternal chorioamnionitis mediated by cytokines, with neonatal encephalopathy not distinguishable from hypoxic ischemic encephalopathy.

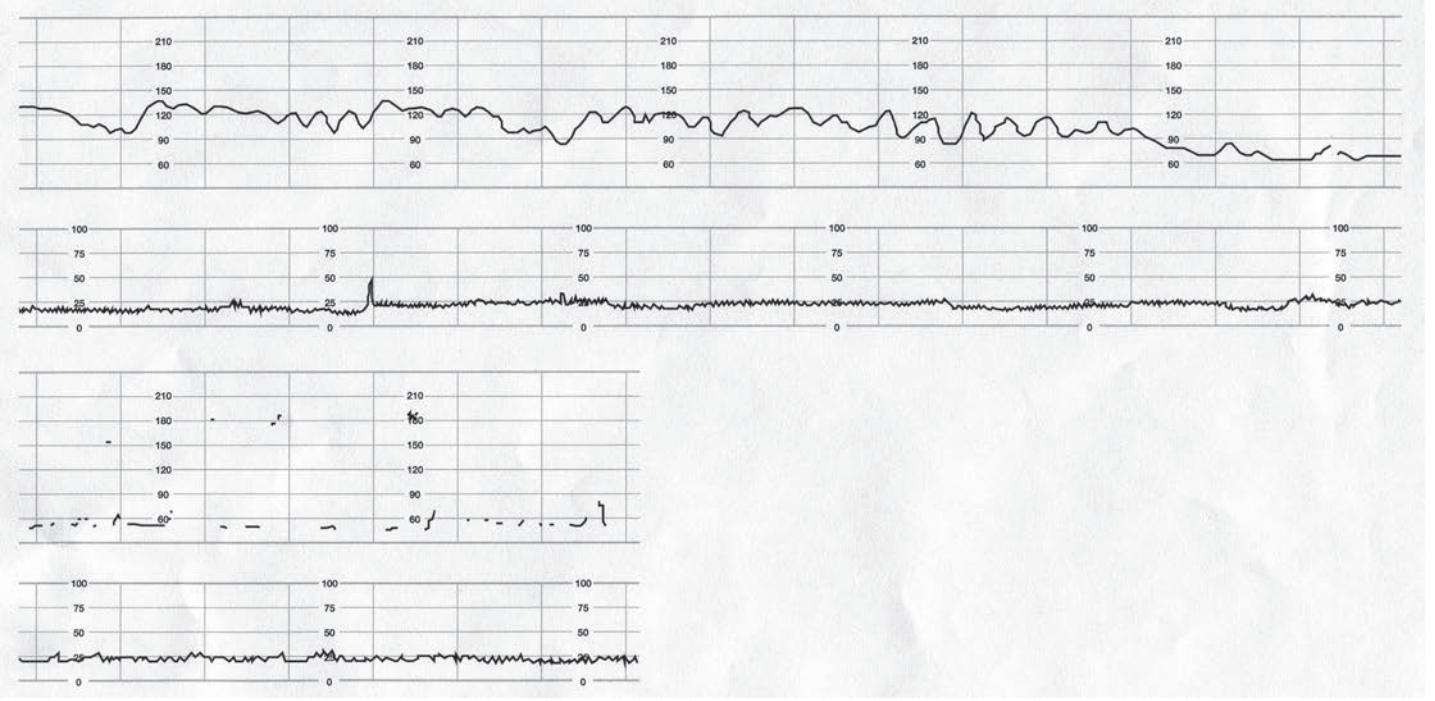

Figure 13.4. Example of a tracing from a fetus who died on the monitor with a blunted pattern leading to a terminal bradycardia. The placenta showed acute chorioamnionitis and funisitis, indicating a fetal inflammatory response as the cause of death.

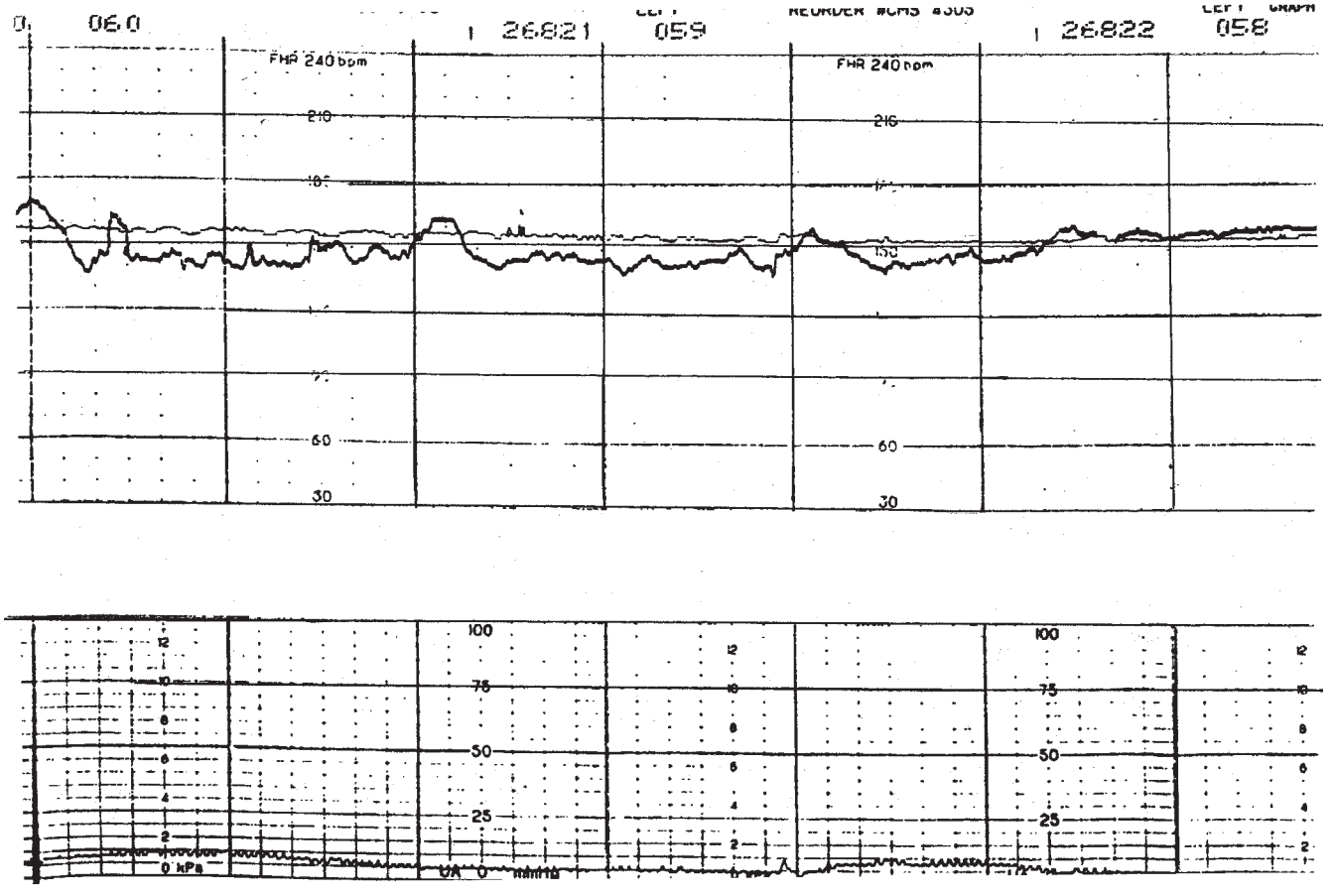

Figure 13.5. Twins at 32 weeks. Twin B is the light tracing showing a flat fetal heart rate. Twin A is the dark tracing showing average variability and reactivity. Twin B had a biophysical profile of 2 for fluid and delivered with low Apgar scores and decreased pH. Twin B now has cerebral palsy.

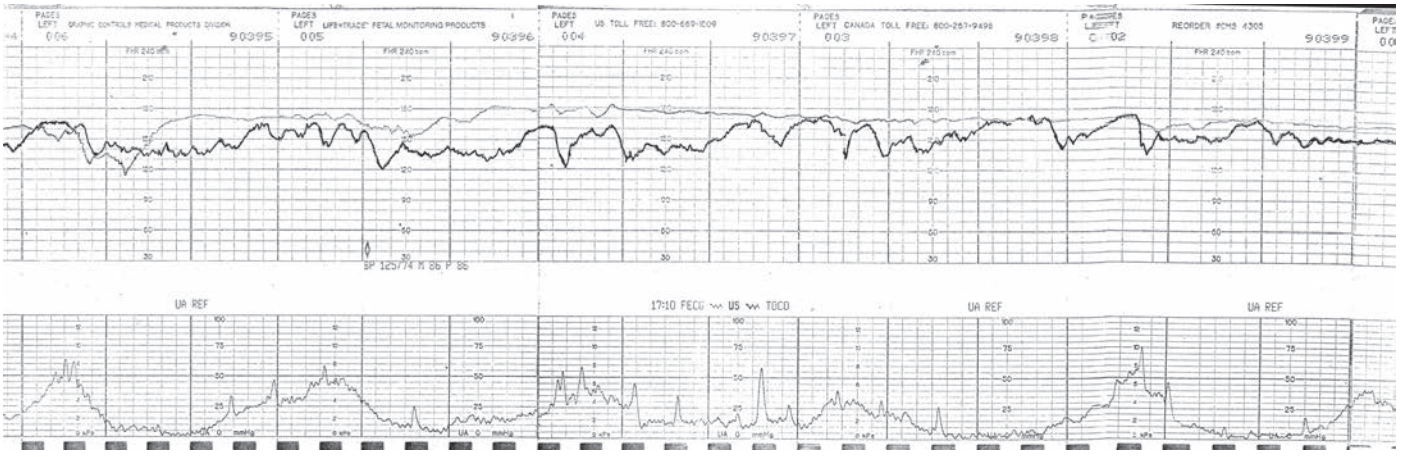

Figure 13.6. Twin pregnancy at 35 weeks. Twin A is the light tracing showing a flat blunted fetal heart rate pattern. Twin B is the dark tracing showing normal variability and reactivity. Twin A had a pH of 6.96 and Apgar scores of 0 at 1 minute and 2 at 5 minutes and 4 at 10 minutes. Twin B had a normal pH and Apgar scores. Twin B now has spastic diplegia and magnetic resonance imaging findings of periventricular leukomalacia.

BLUNTED PATTERNS

When periodic changes do occur in patients with fetal CNS dysfunction, the changes are usually smooth and take on a "blunted" characteristic (Figs. 13.7–13.9). This is especially evident with variable decelerations. There is a loss of the angular components of FHR change that are seen with intact fetuses. Blunted variable decelerations may be of low magnitude and, as a result, may not be noticed easily.

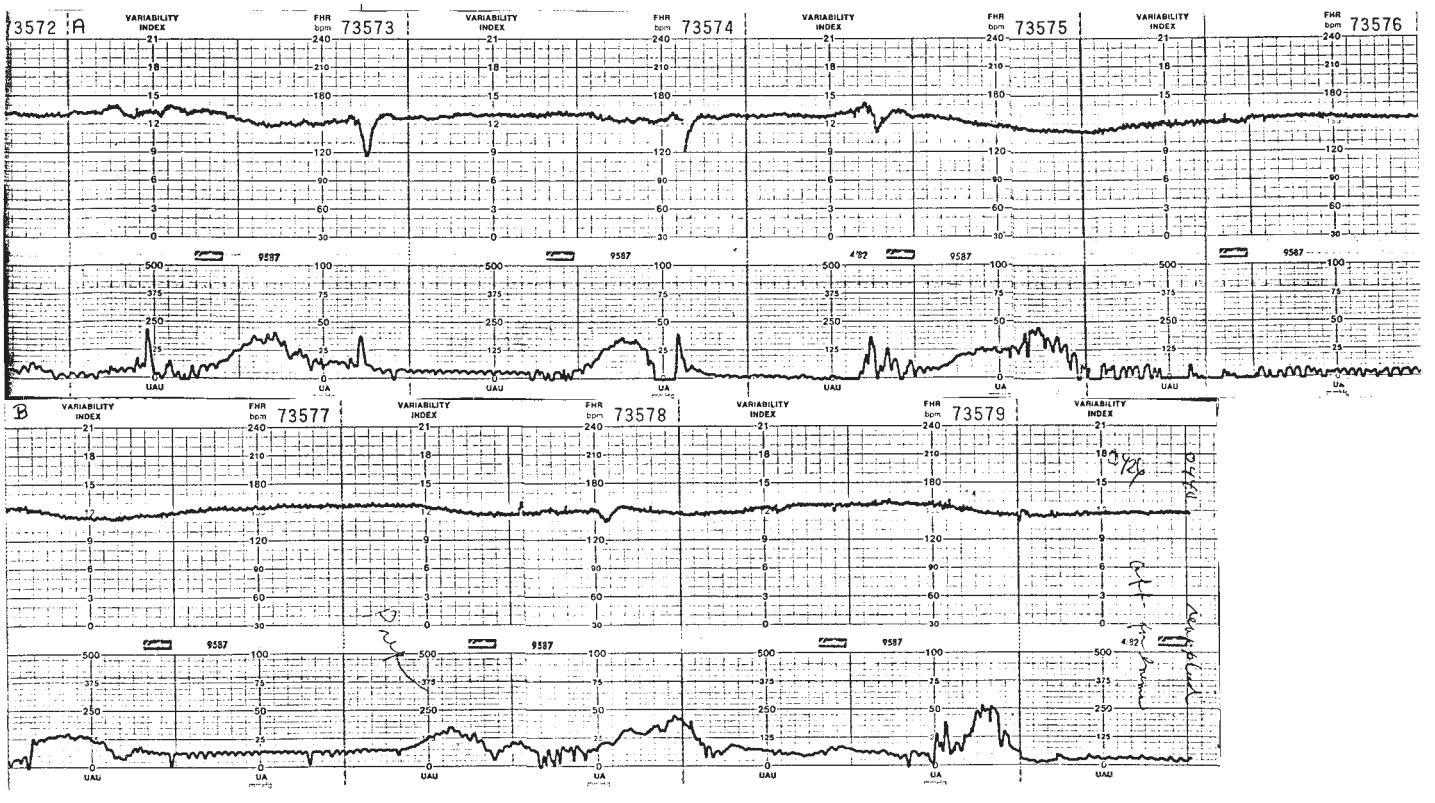

Figure 13.7. An example of blunted, rounded variable decelerations associated with a flat fetal heart rate. There is also an unstable baseline. This tracing was observed for several hours before delivery of a neonate with a normal neonatal pH but low Apgar scores. The neonate began convulsing soon after birth and now has cerebral palsy. This represents an example of preexisting central nervous system damage.

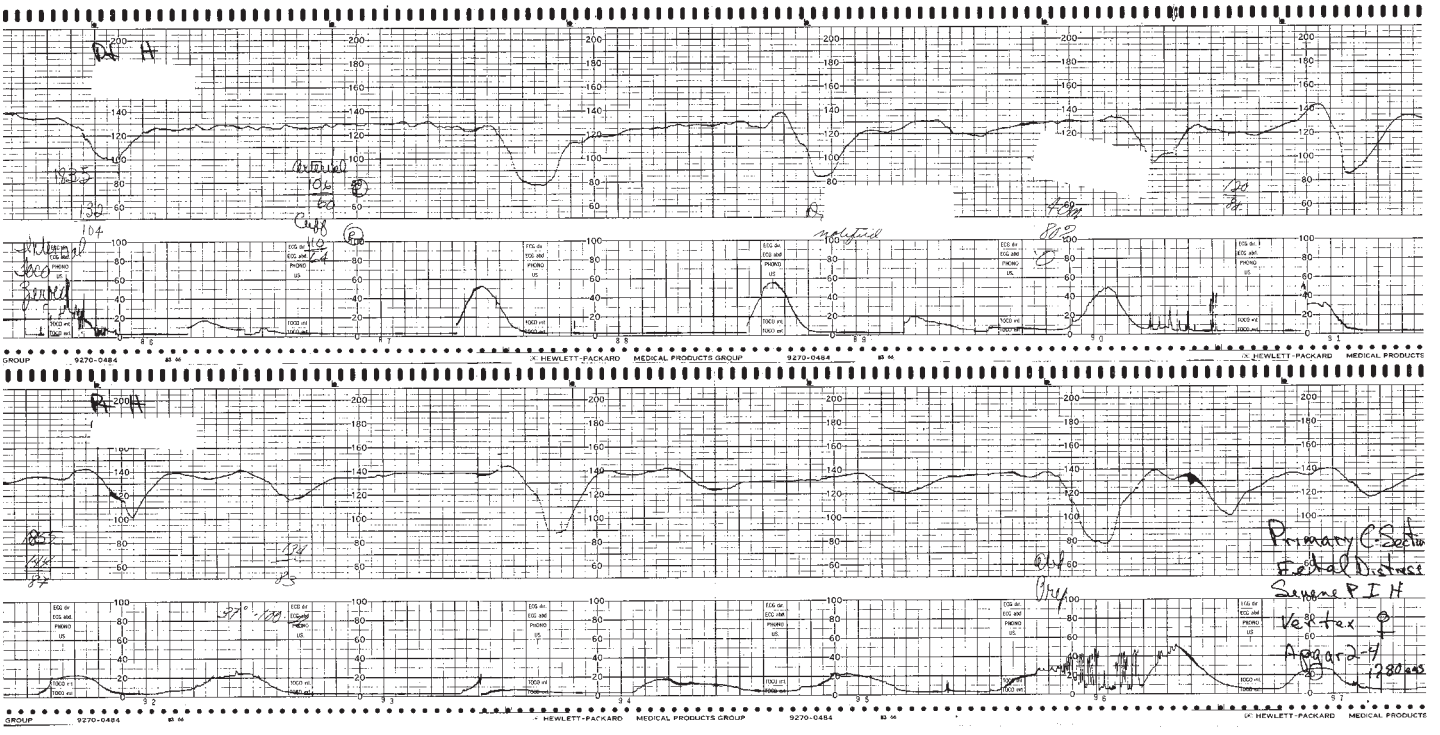

Figure 13.8. A flat fetal heart rate pattern and blunted variable decelerations representing central nervous system dysfunction. Note, however, the associated late decelerations indicating ongoing hypoxia. This fetus was born with fetal acidosis and began convulsing soon after birth. The child now has cerebral palsy.

UNSTABLE BASELINE

The baseline FHR characteristically remains constant for intact fetuses, although it can change over a long period or in response to maternal fever, acute hypoxia, or certain drugs. However, with the CNS dysfunctional fetus, the baseline may appear to wander, and it may be difficult to establish exactly where the baseline is (Figs. 13.10 and 13.11).

OVERSHOOT

Rarely, in fetuses with CNS dysfunction, a prolonged smooth acceleration occurs following a variable deceleration that may take several minutes to return to baseline. The accelerations that may precede and follow reassuring variable decelerations are often referred to as shoulders and should not be confused with overshoot. The variable deceleration may not be of large magnitude. It is always associated with a smooth baseline FHR. This "overshoot" has the shape of a rapid but rounded rise followed by a very gradual return to baseline (Figs. 13.12 and 13.13).

SINUSOIDAL PATTERNS

Sinusoidal patterns are described elsewhere in this book. They are very rare and are characterized by a complete lack of reactivity and by a basically smooth FHR, with the exception of the sinusoidal aspect (Fig. 13.14). There is no short-term variability, and the long-term variability is uniform, going above and below the baseline, with a smooth character and a frequency of three to five cycles per minute with an amplitude that may vary from 10 to 40 beats per minute. There are often areas of completely flat FHR, and late decelerations may also occur. This pattern has been classically associated with fetal anemia, but it can also occur with fetal hypoxia without fetal anemia (Fig. 13.15A,B). It does not indicate irreversible damage in all cases.

"CHECK MARK" PATTERN

This is an extremely rare pattern that was first described by Cruikshank (25) in a case where the mother had had a previous cardiorespiratory arrest. The neonate began convulsing

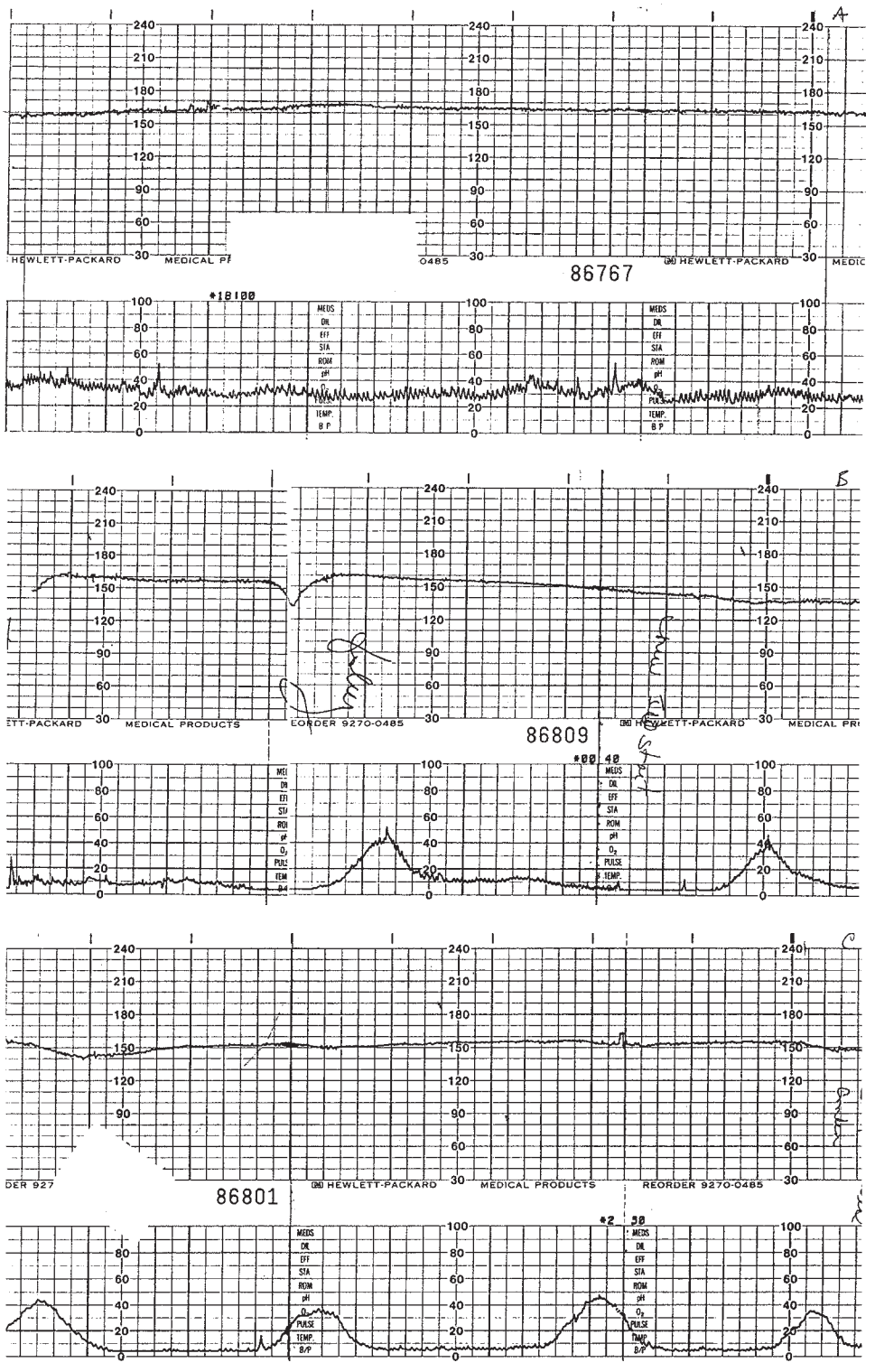

Figure 13.9. Decreased fetal movements at 34 weeks lead to this antepartum tracing. There is an unstable baseline associated with blunted variable decelerations. The neonate had a pH of 6.9 and a base deficit of 18. The neonate had seizures and an intraventricular hemorrhage.

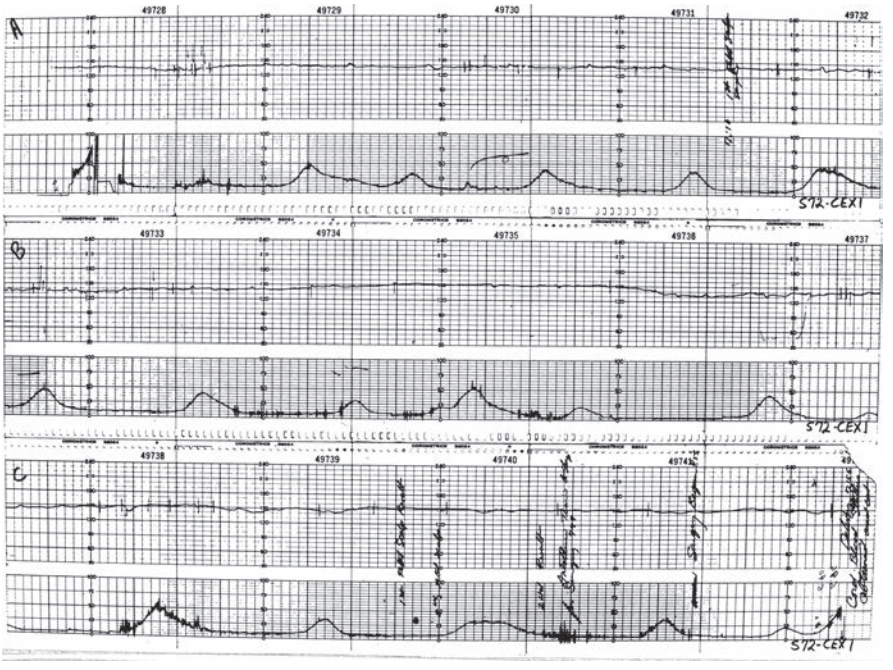

Figure 13.10. A flat fetal heart rate pattern with a wandering or unstable baseline. Note there are no late decelerations or other periodic changes suggesting fetal distress. The fetal scalp pH, however, was 7.01, and the neonate was born with low Apgar scores and subsequently died with evidence of central nervous system dysfunction.

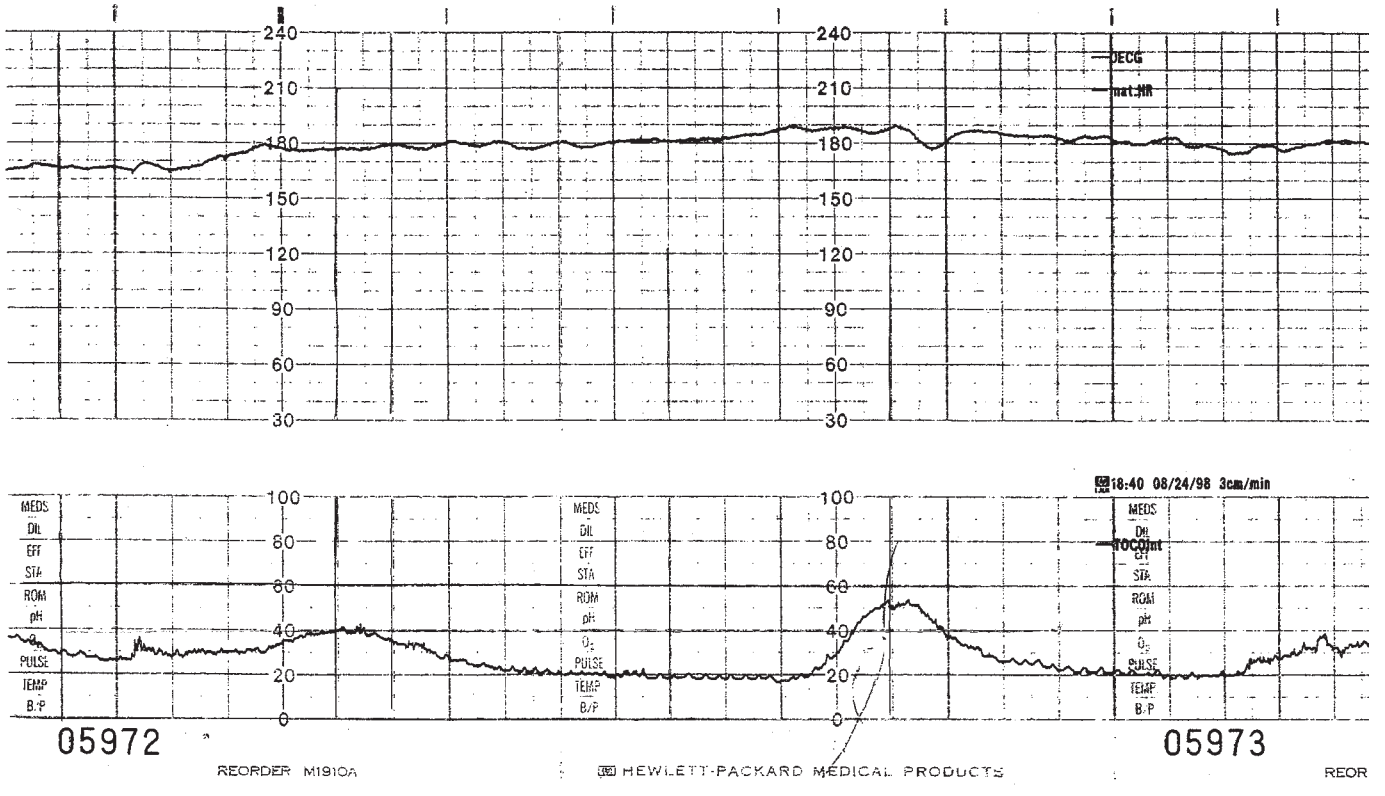

Figure 13.11. Unstable baseline with fetal central nervous system dysfunction.

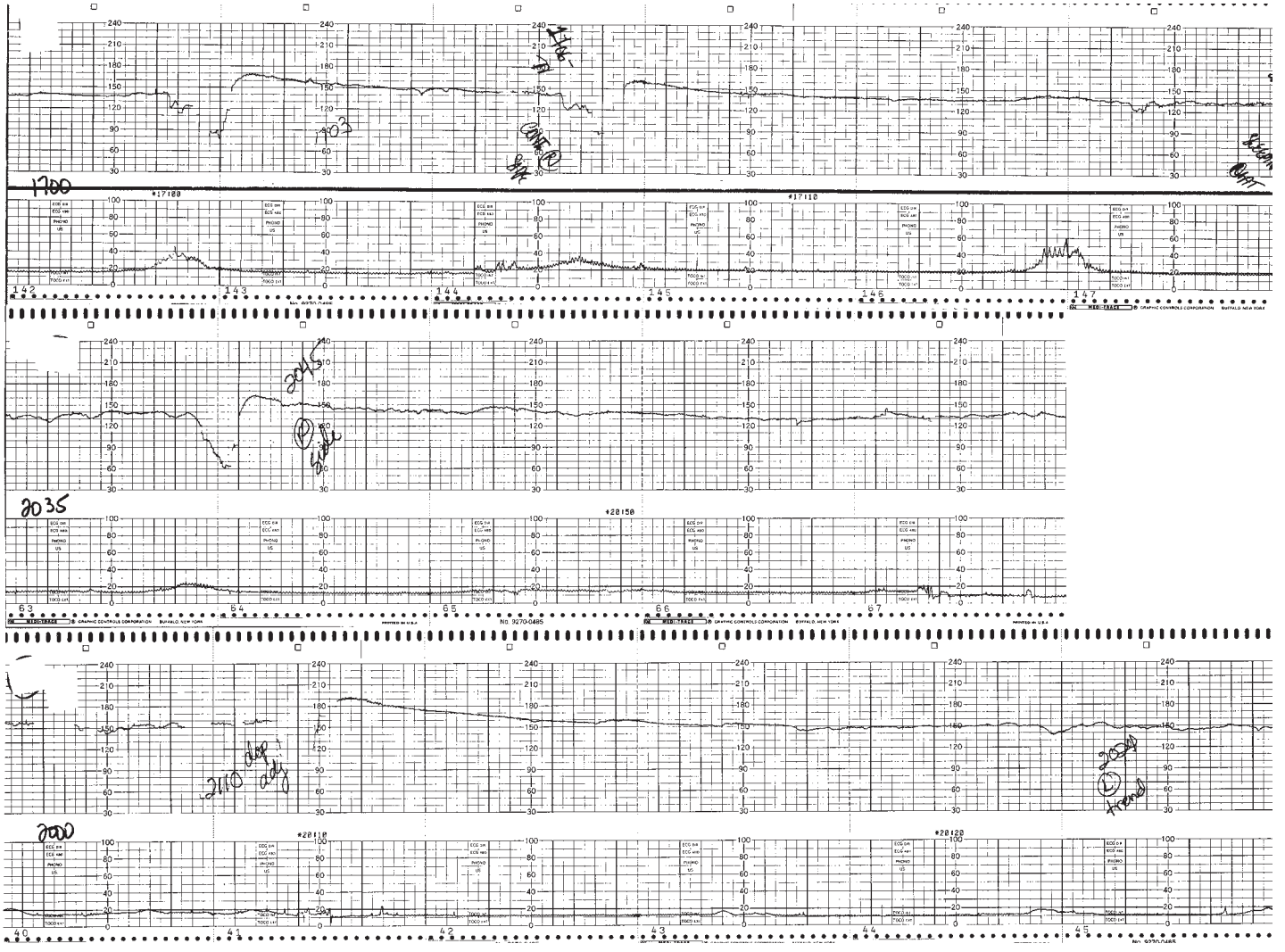

Figure 13.12. The **upper tracing** is an example of overshoot following variable decelerations in a fetus who delivered with a subsequent umbilical arterial pH of 7.10. Note the very slow return to baseline and the associated flat fetal heart rate (FHR). The **lower tracing** is from an anencephalic fetus. Note the flat FHR, blunted variable decelerations, and overshoot. This fetus died during labor, and it is not possible to determine whether this FHR change was all due to the central nervous system (CNS) anomaly or was partially related to hypoxic CNS dysfunction.

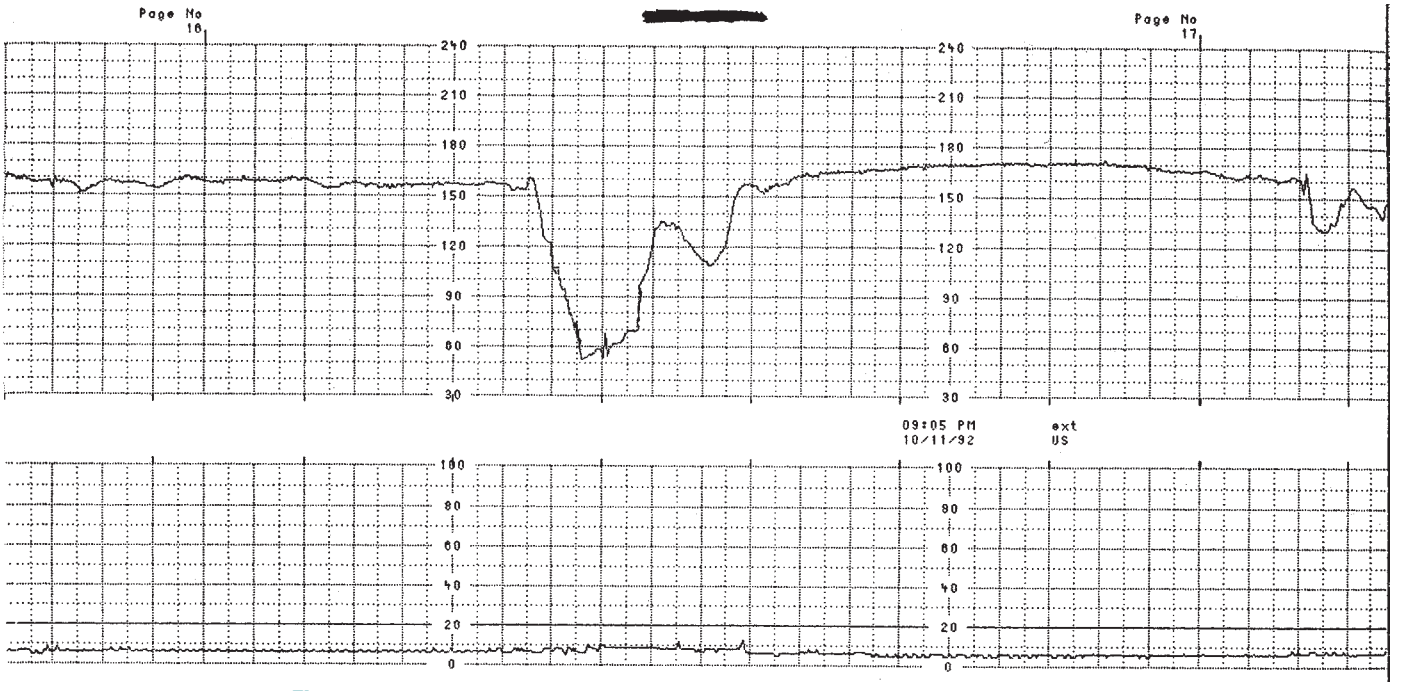

Figure 13.13. This tracing shows blunted variable decelerations with overshoot.

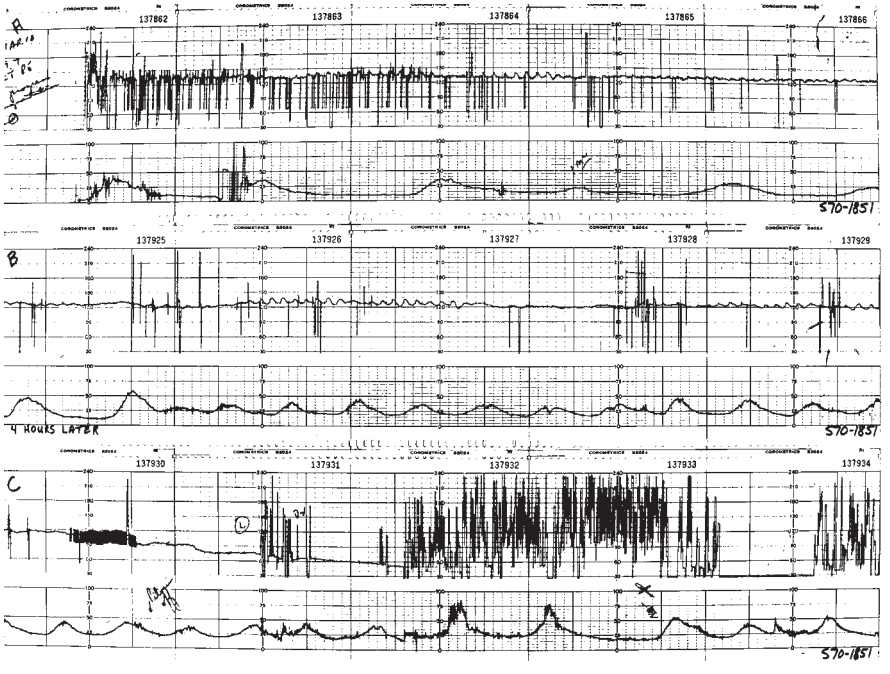

Figure 13.14. A flat fetal heart rate (FHR) pattern with intermittent bursts of sinusoidal heart rate. This fetus was severely hypoxic and died during labor. Note that there are no late decelerations or other periodic FHR changes suggesting fetal distress.

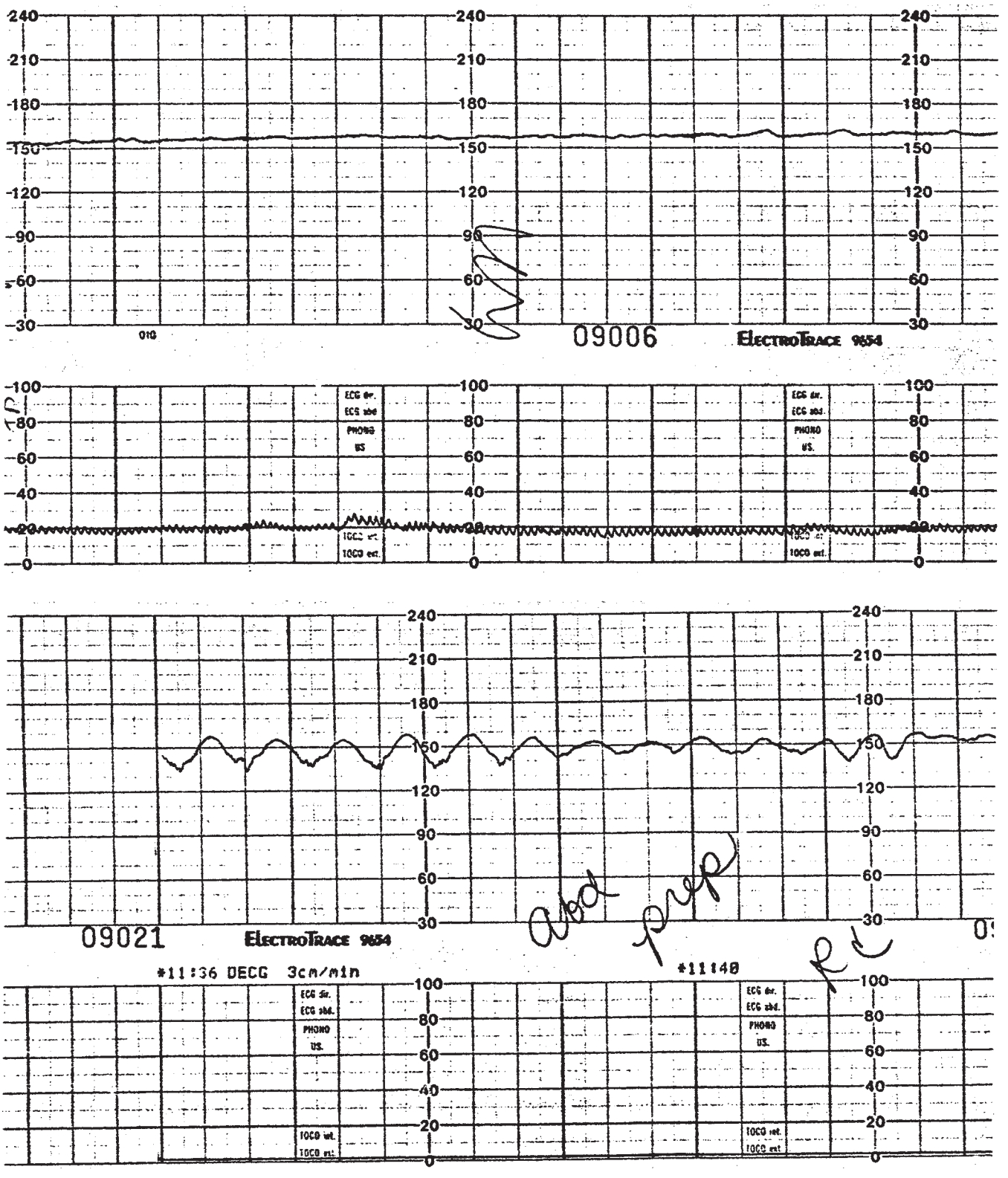

Figure 13.15. This tracing was from a patient with gestational diabetes mellitus at term found at the time of a routine nonstress test done at 0830 in **panel A**. The patient was sent to the hospital for a contraction stress test and when first observed at 1200 had the sinusoidal pattern seen in **panel B**. The neonate was delivered by cesarean section and had Apgar scores of 2 at 1 minute and 8 at 5 minutes. The cord arterial pH was 7.06, and the cord hemoglobin was 19.8 gm%. The neonate developed neonatal seizures with cerebral edema seen on a computed tomography scan in the first 24 hours. Now the child has spastic quadriplegia. The sinusoidal pattern appears to be associated with chronic hypoxia without fetal anemia.

immediately after birth. We have seen this pattern with both normal pH and low fetal pH. Interestingly, there is normal short-term variability, and the check mark pattern occurs approximately every 20 seconds (Figs. 13.16–13.18). The check mark pattern may represent intrauterine fetal convulsions, because the three cases that we are familiar with all were associated with immediate neonatal convulsions.

It is possible to see some elements of one or all of these patterns in any given fetus with CNS dysfunction. With the exception of the check mark pattern, FHR variability is characteristically absent. When one sees such a pattern, a fetal scalp blood pH may help to determine whether there is ongoing hypoxia or probable CNS dysfunction without ongoing hypoxia. It is never possible to tell from the FHR pattern whether the CNS dysfunction is reversible or not.

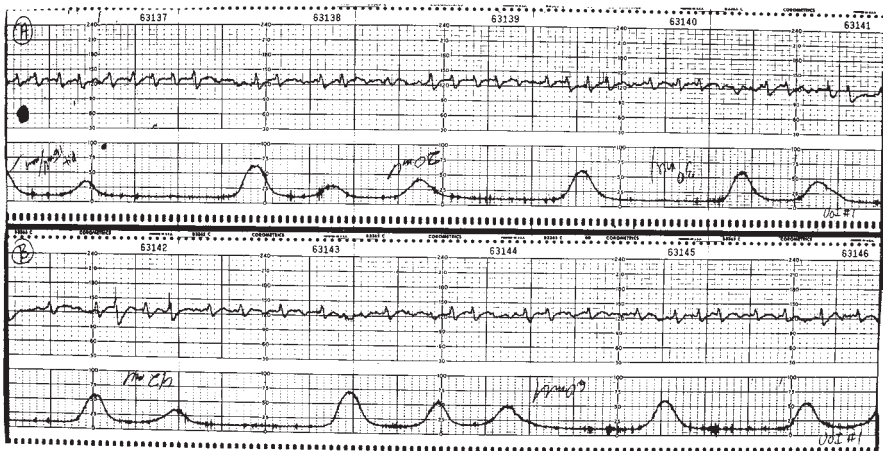

Figure 13.16. A pattern with characteristic "check mark" fetal heart rate changes. Note the normal variability between the check mark changes. This fetus was born following maternal recovery from a severe hypoxic episode and had an umbilical arterial pH of 7.25 but Apgar scores of 1 and 5. The child never breathed spontaneously and had a flat electroencephalograph until its death on the fifth day of life.

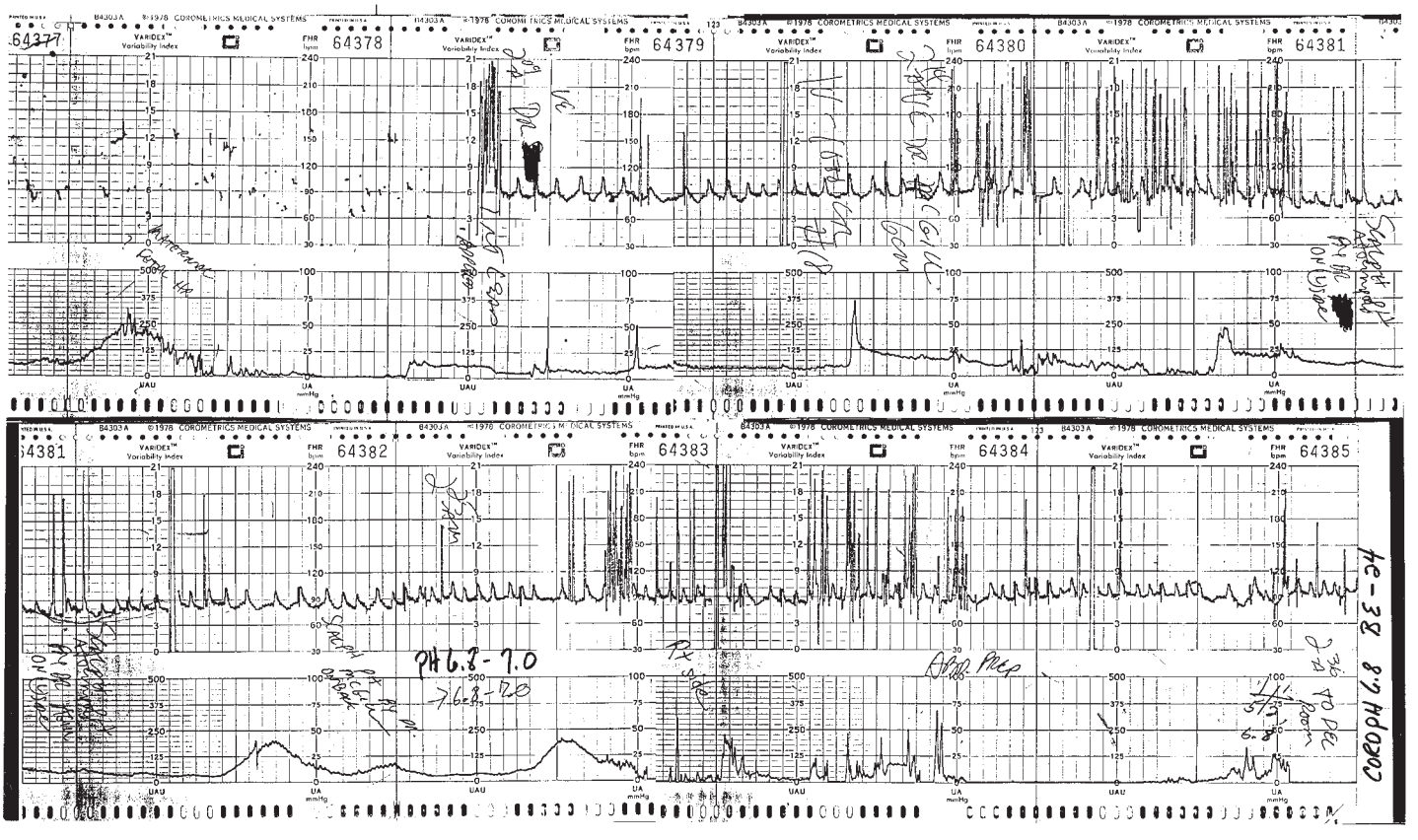

Figure 13.17. Check mark pattern in a fetus with ongoing hypoxia.

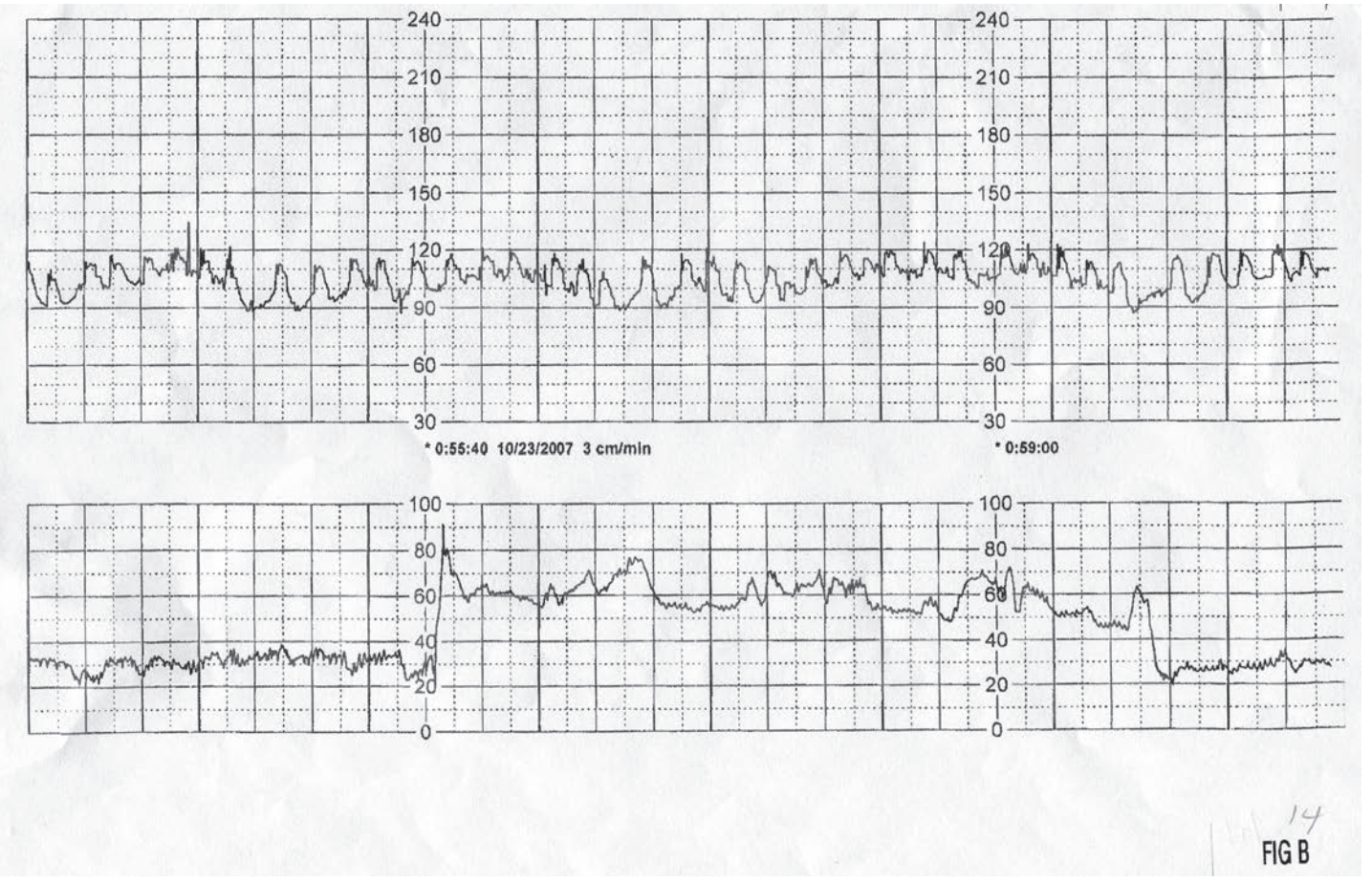

Figure 13.18. This pattern was seen in a patient who had had a very abnormal pattern on admission and was then not monitored for over an hour. Delivery resulted in a neonate with low Apgar scores and severe umbilical arterial metabolic acidosis. The neonate developed neonatal encephalopathy and now has cerebral palsy. The pattern is probably a variant of the check mark pattern.

ETIOLOGIES OF FETAL CENTRAL NERVOUS SYSTEM DYSFUNCTION

The following are examples of clinical situations in which CNS abnormalities may be evident on the FHR tracing as a result of a preexisting condition with a hypoxic or nonhypoxic cause:

1. CNS malformation, for example, anencephaly, hydrocephaly (Fig. 13.19)
2. CNS destruction, for example, tumor (Fig. 13.20)
3. CNS infection, for example, rubella, cytomegalovirus, syphilis, and toxoplasmosis (TORCH)
4. Fetal inflammatory response from maternal chorioamnionitis
5. CNS anoxic insult, for example, maternal cardiac arrest (Fig. 13.21)
6. CNS physical trauma, for example, cerebral contusion
7. Toxic fetal encephalopathy, for example, mercury poisoning
8. Fetal cerebral irradiation with resulting microcephaly
9. Intrauterine fetal CNS hemorrhage
10. Developmental abnormalities, for example, chromosomal (Fig. 13.22)
11. Drugs (Fig. 13.23).

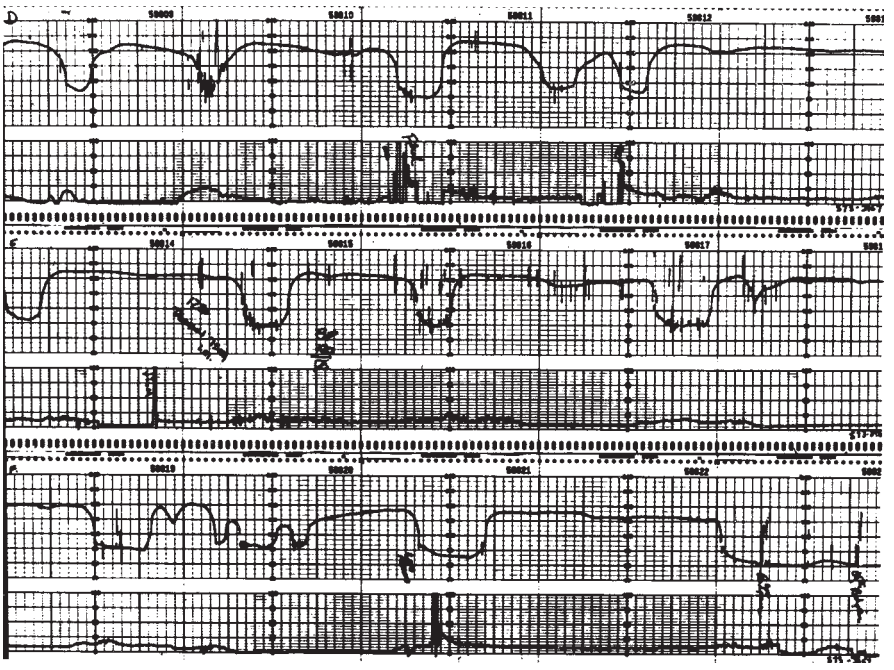

Figure 13.19. A tracing from an internal fetal scalp electrode on an anencephalic fetus. Note the blunted character of the variable decelerations and the flat fetal heart rate baseline.

Fetuses with chronic hypoxia preceding labor (nonreactive positive contraction stress test) will usually have reasonably normal umbilical cord blood gas and pH values if they are delivered without the acute stress of labor. When the chronically hypoxic fetus does show metabolic acidosis at birth without having gone through labor, it is probably very near death. When a chronically hypoxic fetus is subjected to labor, it will usually develop metabolic acidosis very rapidly, indicating that the reserve is limited. We believe these observations are explained by the placenta's ability to maintain fetal acid-base balance even when the fetus is utilizing anaerobic metabolism; when labor is superimposed, uterine contractions further interfere with uteroplacental exchange. When a fetus demonstrates evidence of CNS dysfunction on the FHR tracing, possible explanations include the following:

1. A preexisting abnormality of CNS function with no hypoxia at any time
2. A previous hypoxic injury with resolution and no current hypoxia
3. Ongoing chronic hypoxia but normal acid-base balance because of placental exchange
4. Chronic hypoxia with superimposed acute hypoxia from labor and, therefore, fetal metabolic acidosis
5. No chronic hypoxia and only acute intrapartum fetal hypoxia with metabolic acidosis

How we attempt to differentiate these situations is important both for patient management and from a medicolegal causation standpoint (Table 13.1).

If, in a laboring patient, there are no patterns of persistent late deceleration, severe variable deceleration, or prolonged decelerations accompanying signs of CNS dysfunction, it is unlikely that there is ongoing acute hypoxia. When possible, a normal fetal scalp blood pH value or normal fetal oximetry value can confirm this, and intervention would not be indicated. If, however, there are persistent late decelerations, severe variable decelerations, or prolonged decelerations accompanying signs of CNS dysfunction, ongoing fetal hypoxia can be presumed, and intervention is indicated even without fetal scalp blood pH determination.

Medicolegally, in cases with evidence of CNS dysfunction on the FHR pattern, it would be desirable to be able to assess when CNS dysfunction became irreversible. Unfortunately, the current state of knowledge does not allow us to make this determination. We can only say that the FHR pattern is consistent or not consistent with CNS dysfunction. The observations described in this chapter have usually been made from retrospective analysis of FHR patterns from babies with CNS damage. For this reason, we are unable to determine how often fetuses with evidence of CNS dysfunction on their FHR patterns develop normally, but it certainly does happen. Therefore, we know that these patterns do not always mean that damage

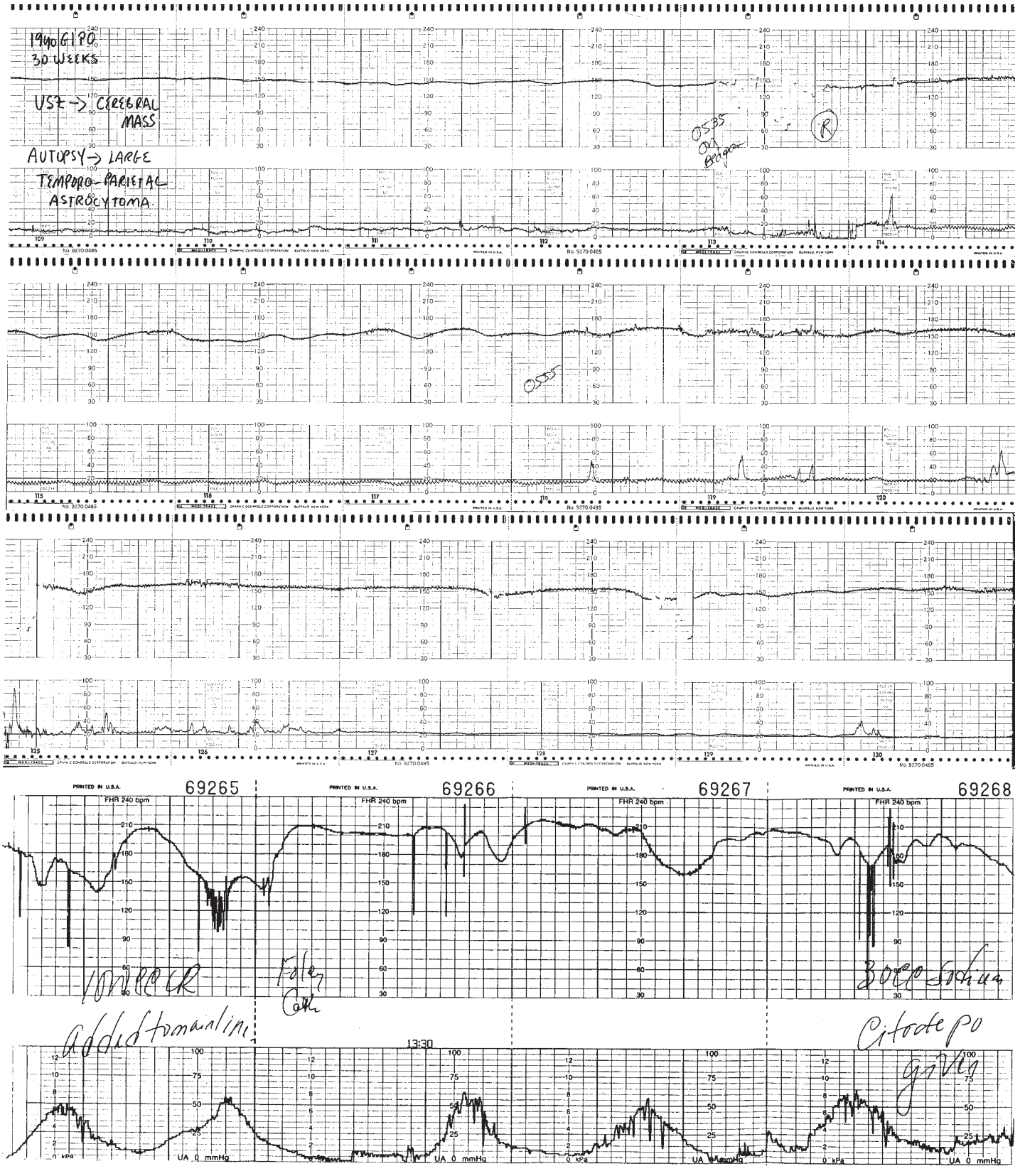

Figure 13.20. **A:** An external monitor recording of a fetus with an intracranial lesion detected on ultrasound that was diagnosed as a parietal astrocytoma in the neonate. Note the flat, blunted character of the pattern. **B:** An internal tracing taken during labor. The outcome was a cesarean section for fetal distress. The fetus had a brain tumor.

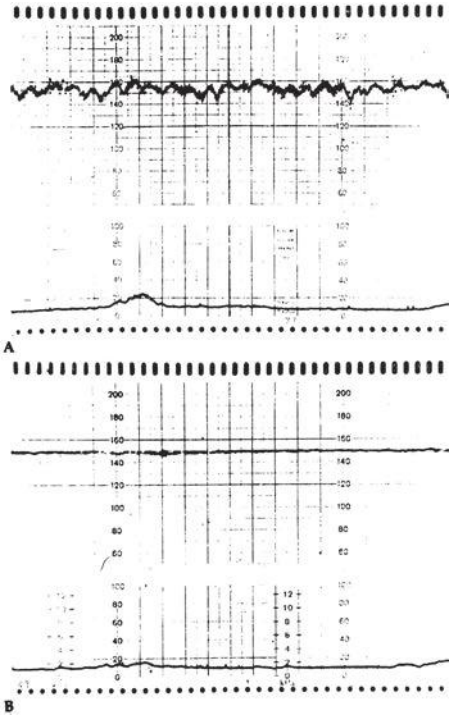

Figure 13.21. The **upper tracing** is an example of a 2-cm-per-minute (20 beats/minute/cm vertical scale) external Doppler tracing taken prior to a maternal cardiorespiratory arrest. Note the normal variability. The **lower panel** represents a tracing from the same fetus following resuscitation of the mother with no ongoing hypoxia. Note the flat fetal heart rate that represents central nervous system (CNS) damage from the prior CNS hypoxic insult. (From van der Moer PE, Gerretsen G, Visser GH: Fixed fetal heart rate pattern after intrauterine accidental decerebration. *Obstet Gynecol* 65:125, 1985, with permission.)

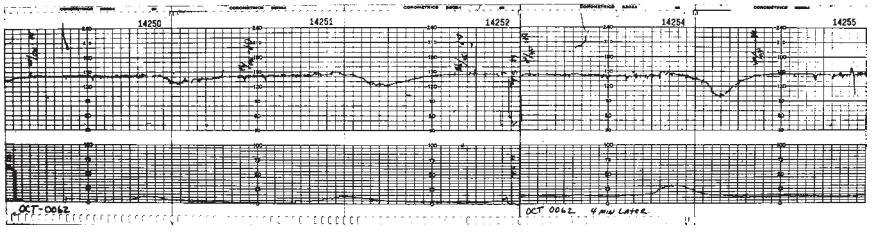

Figure 13.22. A tracing from a fetus with trisomy 18.

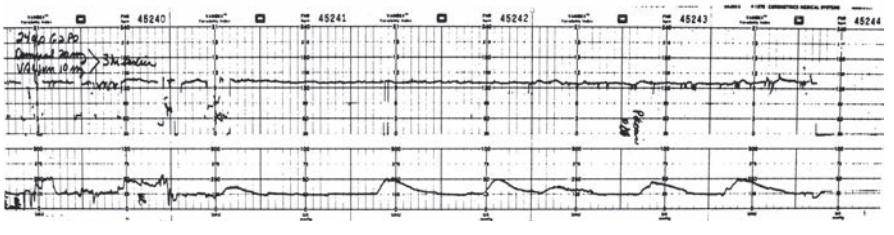

Figure 13.23. An external Doppler tracing from a fetus whose mother received meperidine (Demerol) and diazepam (Valium). Note the flat fetal heart rate pattern without any decelerations, representing central nervous depression due to drugs.

TABLE 13.1 Relationship between fetal heart rate changes, hypoxia, and central nervous system dysfunction[a]

	Group 1	Group 2	Group 3
Late deceleration	Yes	No	Yes
Variable deceleration	Yes	Yes	Yes
Decreased variability	No	Yes	Yes
Blunted patterns	No	Yes	Yes
Acidosis	No	No	Yes
Scalp acceleration	Yes	No	No
Unstable baseline	No	Yes	Yes
Sinusoidal patterns	No	Yes	Yes

[a]Group 1, hypoxia without CNS dysfunction; Group 2, CNS dysfunction without current hypoxia; Group 3, CNS dysfunction with ongoing hypoxia.
CNS, central nervous system.

has occurred, and an element of reversibility is clearly possible at least in some cases. In addition, we now know that neonates and children demonstrate improvement with time in many instances of initial developmental handicap.

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CHAPTER 14

Liability and Risk Management in Fetal Monitoring

Malpractice litigation is prevalent in modern clinical practice, and the associated stress affects all health care providers (1,2). Although not all obstetric malpractice cases involve fetal monitoring, electronic fetal monitoring (EFM) is one of the most common procedures in obstetrics (3), making allegations related to EFM a familiar occurrence in obstetric litigation. Some of the most common allegations leveled against obstetric teams related to fetal monitoring include failure to correctly interpret and/or manage fetal heart rate (FHR) tracings, failures related to team communication, and failure of appropriate provider response to nursing notification of FHR findings (4). And while many clinicians continue to believe that most malpractice suits in obstetrics are frivolous, evidence suggests that substandard care can be linked to adverse outcomes. Specifically, in claims related to EFM and fetal hypoxia, substandard care has been reported to be an issue in as many as 60% of cases (5). Several centers have successfully instituted safety approaches to reduce adverse obstetric outcomes and improve teamwork (6–8), with some interesting similarities among these initiatives. These include communication and teamwork training, standardized approaches to common clinical practices, improvements in situational awareness, and rapid response teams for emergency situations.

This chapter will address common areas of risk for nurses, midwives, and physicians, with suggested practice strategies using a case-based approach. We will review key aspects of risk management related specifically to fetal monitoring, including suggestions for evaluation and management, documentation, and nurse-provider communication. The case example approach will also illuminate two types of defenses to malpractice allegations. Finally, public policy efforts aimed at the establishment of alternatives to the current tort system will be briefly presented in an effort to enhance awareness of these initiatives.

THE MEDICAL LIABILITY PROCESS

Following a poor obstetric outcome, patients will often initially consult an attorney because they are seeking an answer to the question "Why did this happen?" But the legal process in medical malpractice cases is not necessarily designed to provide answers. Claims of medical and nursing malpractice are adjudicated in civil, versus criminal, courts under the area of law known as tort law. This system is therefore adversarial, with attorneys representing each side of the action, or lawsuit. The goal of the plaintiff is to win the case by proving negligence, or malpractice, while the goal of the defense is to prevent the plaintiff from succeeding. For a plaintiff to win a malpractice suit, four elements must be proven:

1. Duty—that the patient was owed a specific standard of care by the defendant
2. Breach—that the defendant failed to meet the specific standard of care
3. Causation (proximate cause)—that there is a direct relationship between the failure to meet the standard of care ("breach of duty") and the injury to the patient
4. Injury—that there was an actual harm or injury

Although "injury" is listed as the fourth element, in reality it is the primary element, for without a recognized injury there can be no lawsuit. The tort system provides only one remedy to an injured party and that remedy is a monetary award known as "damages." The system is therefore driven by "bad outcomes," with the severity of the outcome directly proportional to the amount of the potential award. Assuming there is a recognized injury, this leaves the plaintiff with three elements to prove in order to win monetary damages. The first element, duty, is easily proven by establishing a relationship between the health care clinician (nurse, midwife, resident, physician, etc.) and the patient. The next element is breach of duty, which must be established by proving a deviation from the standard of care. The third element, causation

or proximate cause, is proven by the plaintiff establishing a causal link between the alleged deviation(s) of the standard of care and the actual injury sustained. Thus, there are fundamentally two basic types of defenses to allegations of malpractice, and one or both may be used depending upon the facts of the case. The first is a defense premised upon the showing that there was no "breach"; that is, the defendant clinician provided care that was within the "standard of care," commonly defined in most jurisdictions as care that is reasonable and prudent. The second defense relates to the element of "causation." The basic assertion of a causation defense is that any breach of the standard of care could not be related to the harm or injury sustained by the plaintiff. In cases involving EFM, an understanding of both defenses is crucial. The following case examples will illustrate a variety of issues in obstetric malpractice related to both standard of

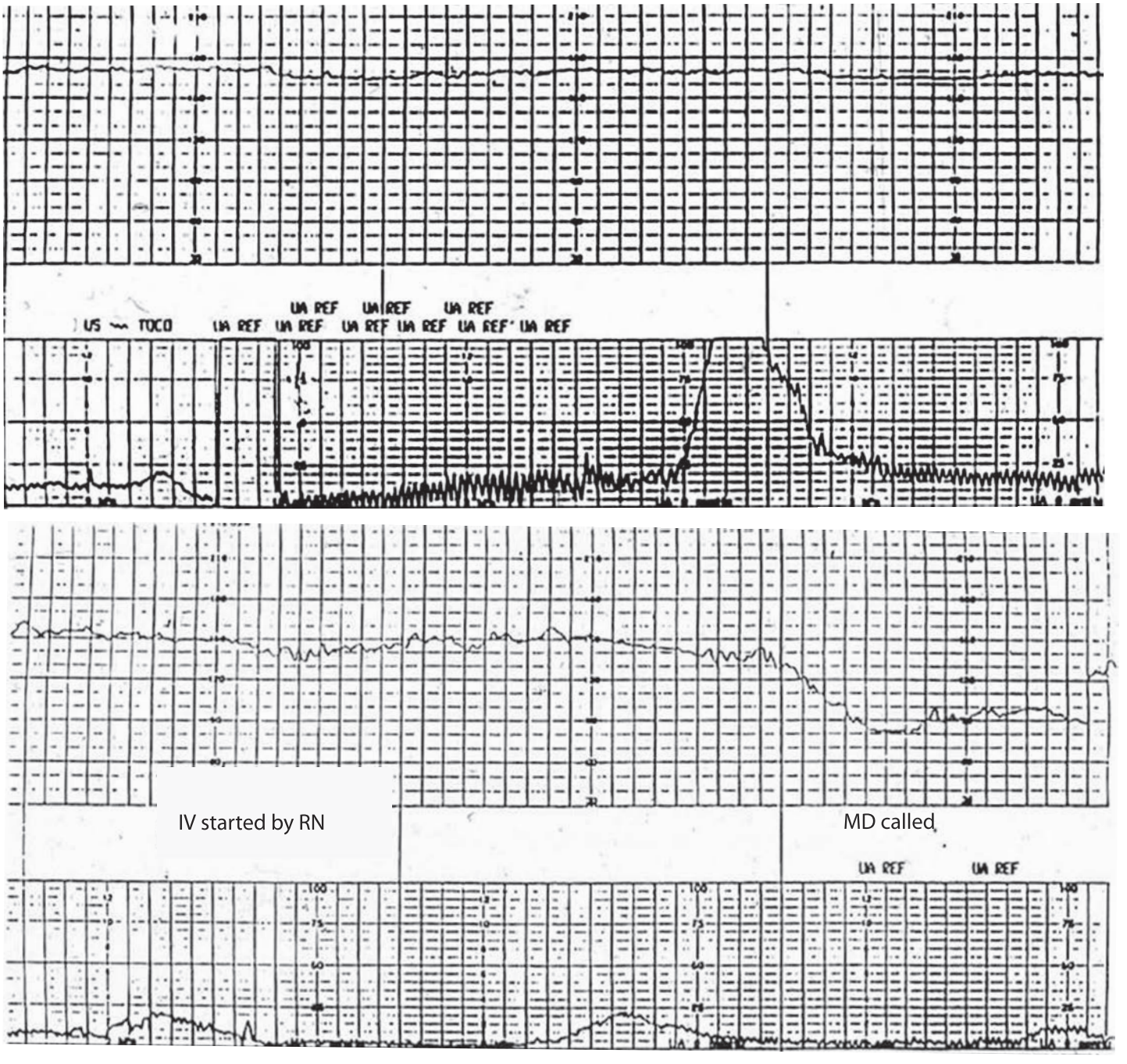

Figure 14.1. Note initial tracing with tachycardia and minimal variability (**A**), followed by an apparent late deceleration and a prolonged deceleration (**B**).

care and causation, and will provide suggestions for practices that both reduce the occurrence of preventable error as well as promote the vigorous defense of appropriate care.

Case Example No. 1

A 39-year-old G3P2002 presented to the hospital for a scheduled nonstress test (NST) at 40 weeks' gestation due to a complaint of decreased fetal movement in the physician's office earlier in the day. EFM was applied, and a prolonged deceleration was noted by the nurses (Fig. 14.1A,B), who turned the patient to her side, began an intravenous line, and immediately notified the physician. The physician arrived, viewed the tracing, and documented "NST nonreactive due to patient not eating. Discharge home and repeat NST in AM" (Fig. 14.2A,B). Although the nurses believed

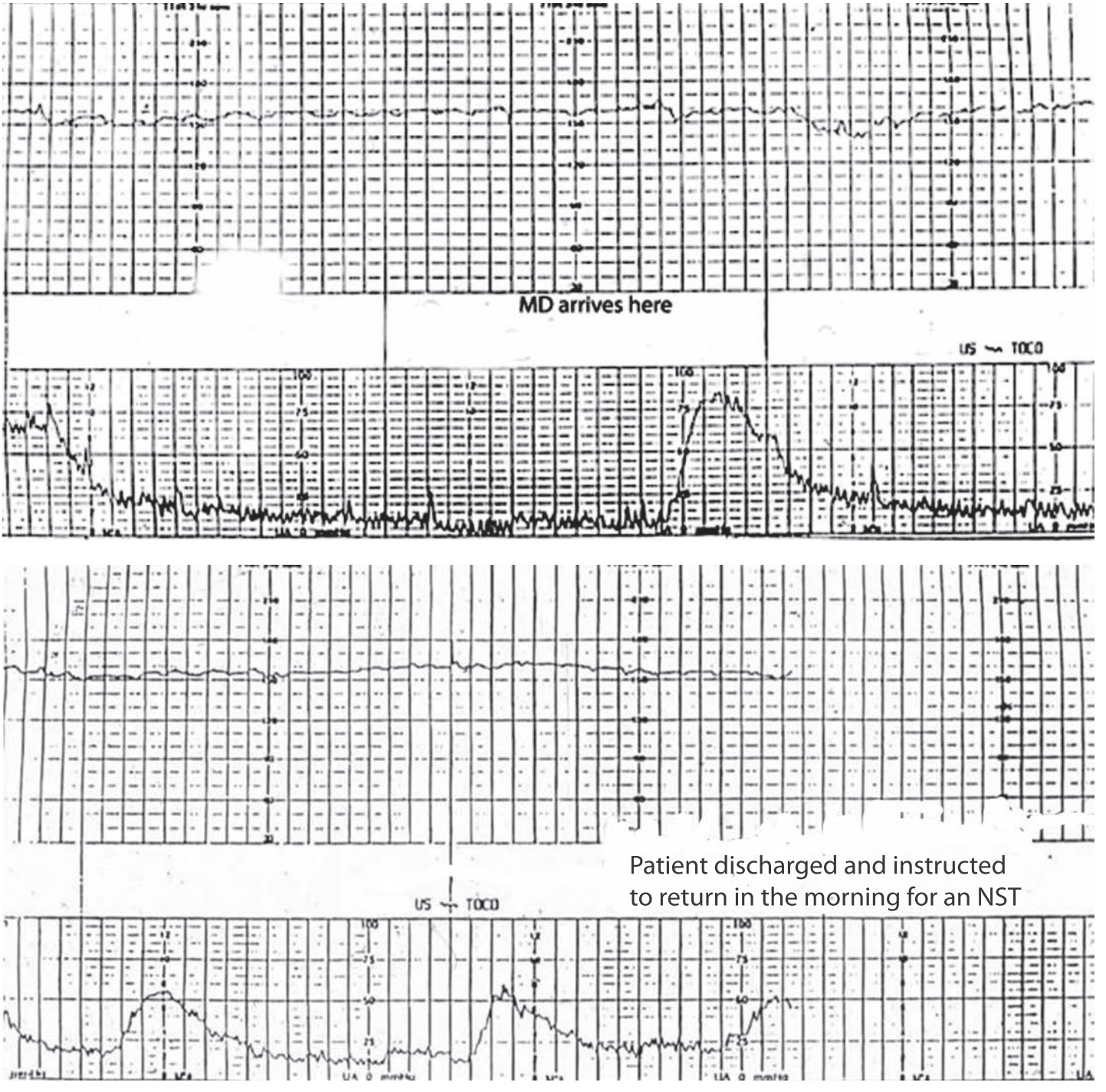

Figure 14.2. Physician arrives and reviews tracing (**A**); decision to discharge patient with instructions to return for a repeat NST the following morning (**B**).

the patient should not be discharged, they did not question the physician's decision, and the patient was sent home with instructions to rest, increase her fluid intake, and return in the morning. The patient returned and was found to have an intrauterine fetal demise after the nurses were unable to find fetal heart tones (FHTs). Litigation ensued and both the hospital and physician settled for an undisclosed amount.

In this case, both the physician and nurses involved deviated from the standard of care by discharging a patient with not only a nonreactive NST but also an FHR tracing demonstrating late and prolonged decelerations. The physician's apparent belief that the NST was nonreactive because the patient had not eaten was erroneous, and all nonreactive or abnormal antenatal tests require immediate follow-up tests or clinical intervention. Although the nurses disagreed with the physician's decision, they did not communicate their concern to the physician, nor did they institute the chain of command, or chain of communication, to resolve the issue. When interviewed after the fact, the nurses stated that they were afraid of the physician and "didn't want to get yelled at." In 2008, the Joint Commission on Accreditation of Healthcare Organizations (Joint Commission) issued a sentinel event alert addressing the issue of disruptive behavior and its negative impact on patient safety (9). Clinicians must have a mechanism for handling conflict or questioning the plan of care. The modern approach to patient safety recognizes that all team members, regardless of educational background, play pivotal and important roles in patient care. Both physicians and nurses must be able to recognize abnormal FHR changes and provide appropriate follow-up for antepartum testing. Hospital policies that provide a mechanism for appropriate discussions and input from other team members when questions or concerns arise regarding management plans or FHR tracing interpretation are integral to patient safety. Nurses, midwives, and physicians should receive ongoing communications training and be encouraged to speak up if questions arise, and questions should not be viewed as challenges to authority but rather as fail-safes to prevent unintentional errors. A sample chain of communication model is illustrated in Figure 14.3.

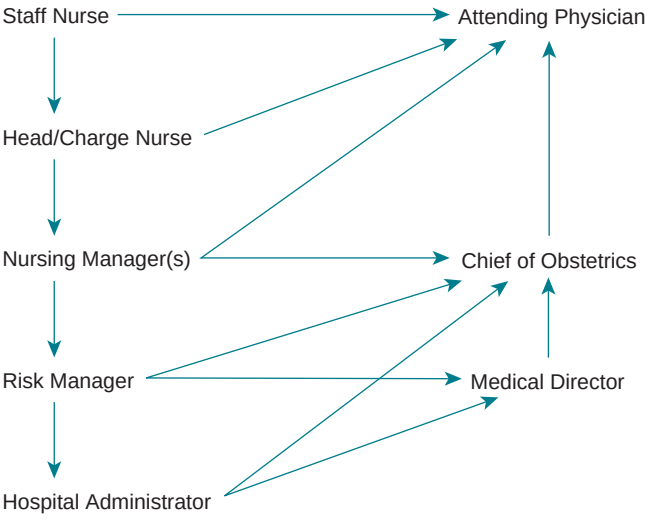

Figure 14.3. Sample chain of communication model. (From Tucker SM, Miller LA, Miller DA. *Fetal monitoring: a multidisciplinary approach*. 7th ed. Mosby, St. Louis: in press; used with permission)

Case Example No. 2

A 23-year-old G1P0000 at term called her physician complaining of labor. The physician sent the patient to the hospital where she was sent to a waiting area for over 2 hours. When placed on the monitor, the nurse could not find FHTs with the external Doppler, though she and another nurse attempted for over 20 minutes. There was no in-house physician to perform artificial rupture of membranes (AROM), although the patient's exam would have allowed AROM by a qualified clinician. The attending physician was called, and while in route, the patient's membranes ruptured spontaneously revealing meconium. The nurse then placed a fetal scalp electrode that revealed bradycardia (Fig. 14.4) and the patient was taken to the operating room (OR) for an emergency cesarean delivery. Cord gases revealed metabolic acidemia and the infant required full resuscitation including medications. Following a long course in the neonatal intensive care unit, the infant was discharged and eventually diagnosed with cerebral palsy. Litigation was initiated. The patient testified that she had told the nurse on arrival that she was in labor and had not felt her baby move for several hours. The nurse testified that the patient had not told her of the decreased fetal movement upon arrival, but was unable to explain the extended time in the waiting room prior to evaluation. Plaintiff's allegations included delay in initial monitoring and delay in obtaining FHR via internal method, with the overall resulting delay in delivery as proximate cause. A confidential settlement was reached for both the hospital and physician.

Obstetric triage is an area that deserves attention from a risk management perspective. The Guidelines for Perinatal Care state that "a pregnant woman who comes to the labor and delivery area should be evaluated in a timely fashion" (10). Although the term "timely" is not specifically defined, it is unlikely that a delay of 2 hours could be considered reasonable in any patient complaining of labor. And while it is appropriate for nursing staff to initially assess obstetric triage patients, if a nurse is unable to find a FHR in a term patient in a short period of time, an obstetric provider should be notified. This case illustrates the importance of departmental policies for obstetric triage and a systematic approach to dealing with difficulty obtaining FHR data. Many hospitals have implemented the use of an obstetric hospitalist, also known as a laborist, or an in-house call system so that labor and delivery has an obstetrician available for emergencies, and the American College of Obstetricians and Gynecologists (ACOG) has endorsed this approach (11,12). Although it

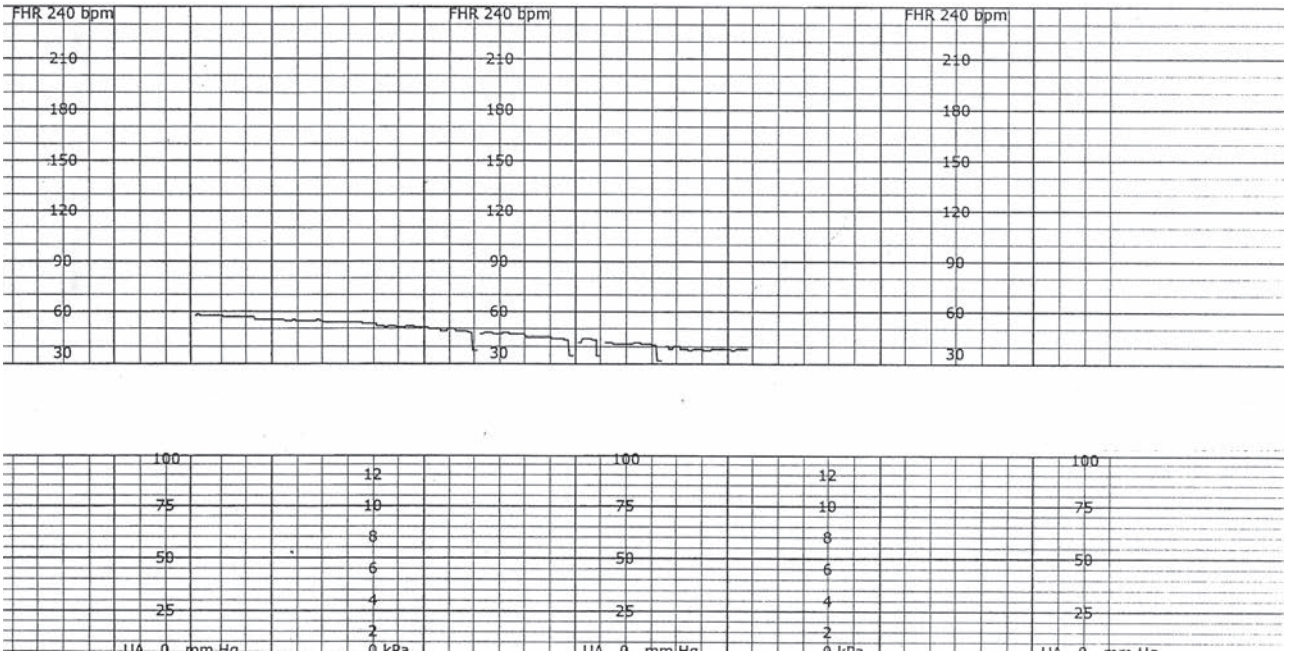

Figure 14.4. Internal fetal electrode application reveals bradycardia.

was published several years after the aforementioned case example, the Association of Women's Health, Obstetric, and Neonatal Nurses (AWHONN) has a position statement (13) that supports nursing placement of an internal fetal scalp electrode through intact membranes in situations similar to the one described, and hospitals should provide education and training, as well as competence validation, for staff nurses so that they may avoid delay in obtaining FHR data when a physician or midwife is not immediately available. Additionally, nurses can follow AWHONN guidelines for training and competency in limited ultrasound (14), which would also allow documentation of FHR in situations where it may be difficult to find with external Doppler, such as obesity or multiple gestation.

Case Example No. 3

Case example no. 3 involves nursing, midwifery, and obstetric care during the second stage of labor. A 23-year old G1P0000 had an uneventful prenatal course followed by a nurse-midwife who was part of a hospital-owned group practice of physicians and midwives. Labor was augmented with oxytocin due to a protracted active phase following epidural placement. The patient was complete and plus one station at 4:00 PM with a baseline FHR of 145 beats per minute (BPM), moderate variability, no accelerations, and (initially) no decelerations (Fig. 14.5). Although uterine activity appeared adequate and the patient has reached the second stage, the nurse continued to increase the oxytocin, now to 16 mU per minute (Fig. 14.6). The patient begins pushing, and prolonged decelerations soon become evident (Fig. 14.7). The nurse calls the nurse-midwife to the bedside, and the nurse-midwife increases the oxytocin to 17 mU per minute and begins active pushing using the closed-glottis technique (Fig. 14.8). The decelerations become recurrent and progressively deeper, accompanied by a rising baseline and loss of moderate variability (Fig. 14.9). The physician is not notified until 6:50 PM, at which time the nurse discontinues Pitocin, instructs the patient to stop pushing, and begins intrauterine resuscitation with position change, IV fluid bolus, and supplemental oxygen (Fig. 14.10). The patient is moved to the OR where an emergency cesarean section is performed under general anesthesia. Delivery occurs at 7:10 PM. Cord gases are obtained, with an arterial cord pH of 6.80 and the base excess reported as "unable to calculate—out of analyzer range." The initial neonatal diagnosis is hypoxic ischemic encephalopathy (HIE), and the infant is eventually diagnosed with cerebral palsy of the spastic quadriplegic type. Case review revealed all of the essential criteria necessary for establishment of a causative link between intrapartum events and cerebral palsy as discussed in Chapter 3, including metabolic acidemia in arterial cord blood and early onset of severe neonatal encephalopathy. Litigation and a mediation process resulted in a structured settlement.

This case illustrates a number of potential liability areas related to EFM, including basic FHR tracing interpretation, management of oxytocin and uterine activity, and management of the second stage of labor. The nurse and nurse-midwife in this case continued to encourage active pushing

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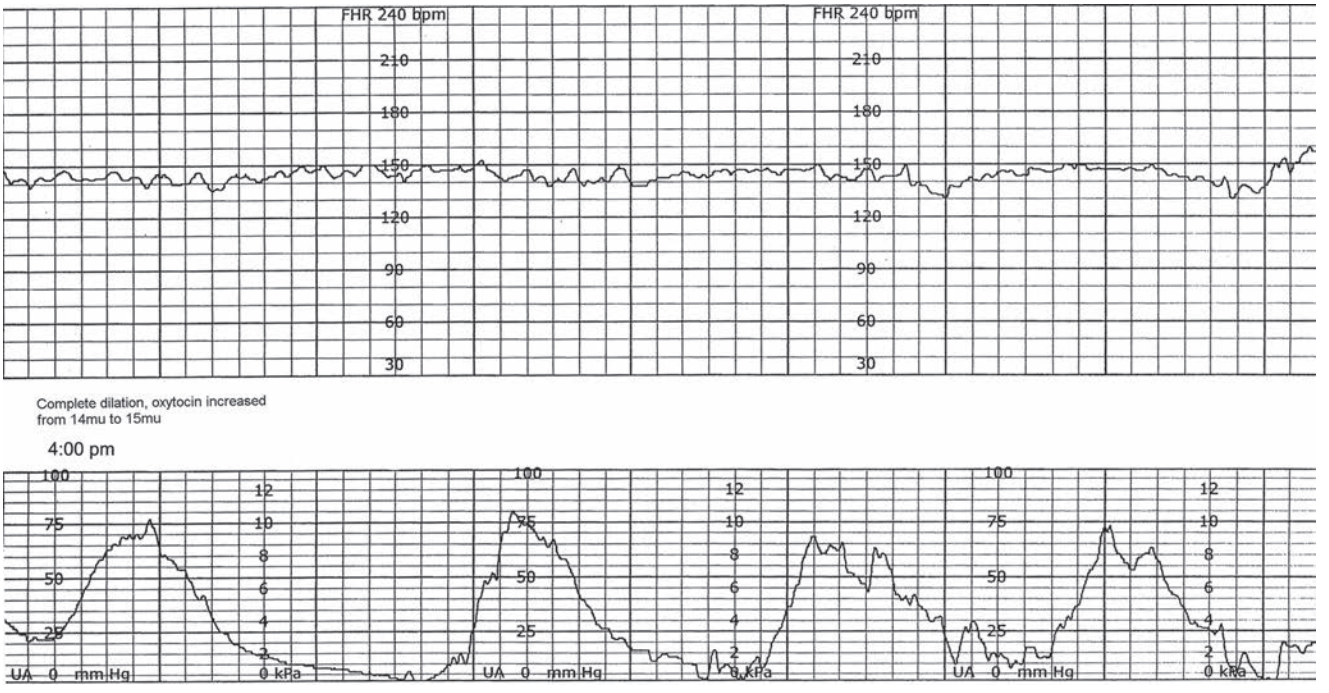


Figure 14.5. Complete dilation, +1 station.

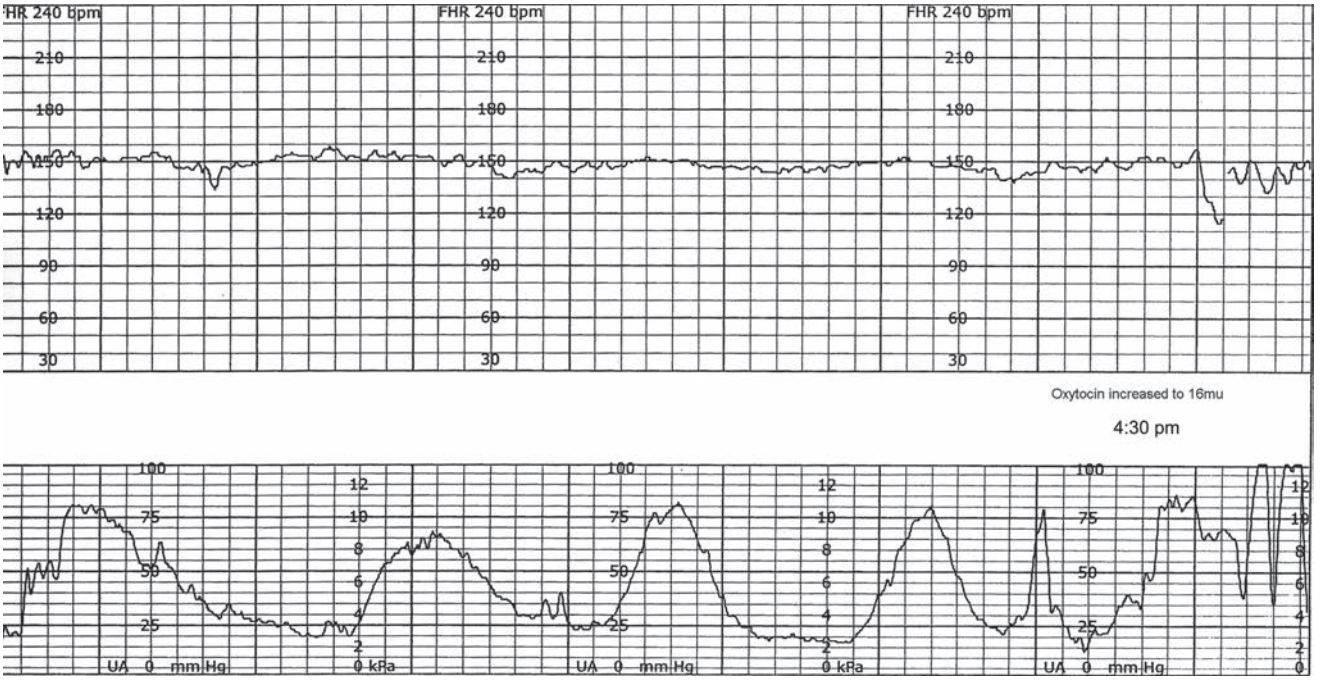


Figure 14.6. Oxytocin inappropriately increased.

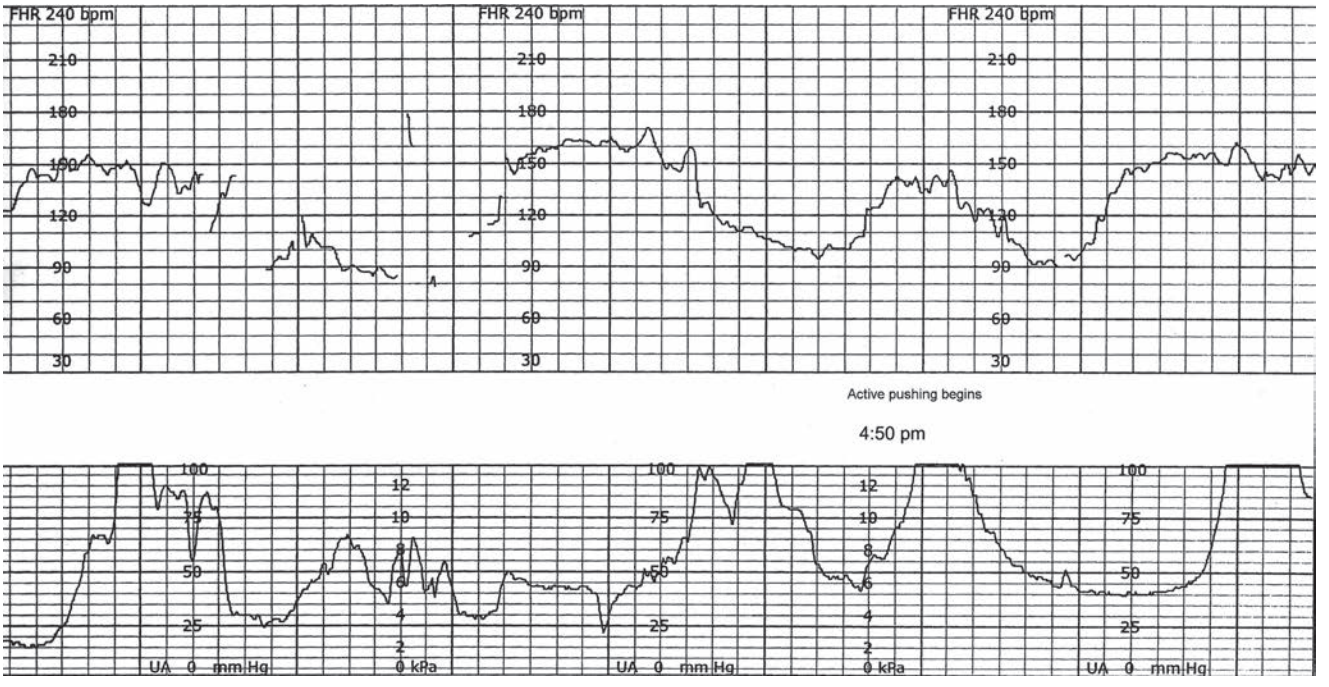


Figure 14.7. Active pushing begins.

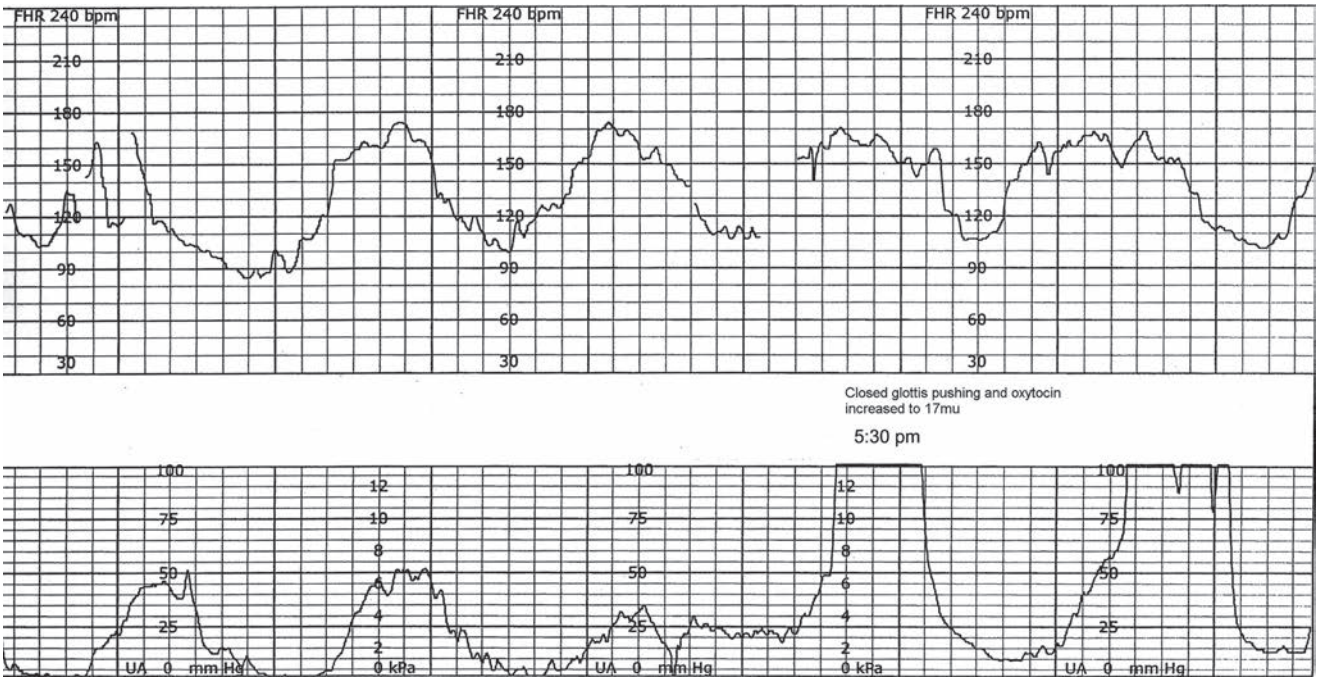


Figure 14.8. Nurse-midwife increases oxytocin and begins closed-glottis pushing.

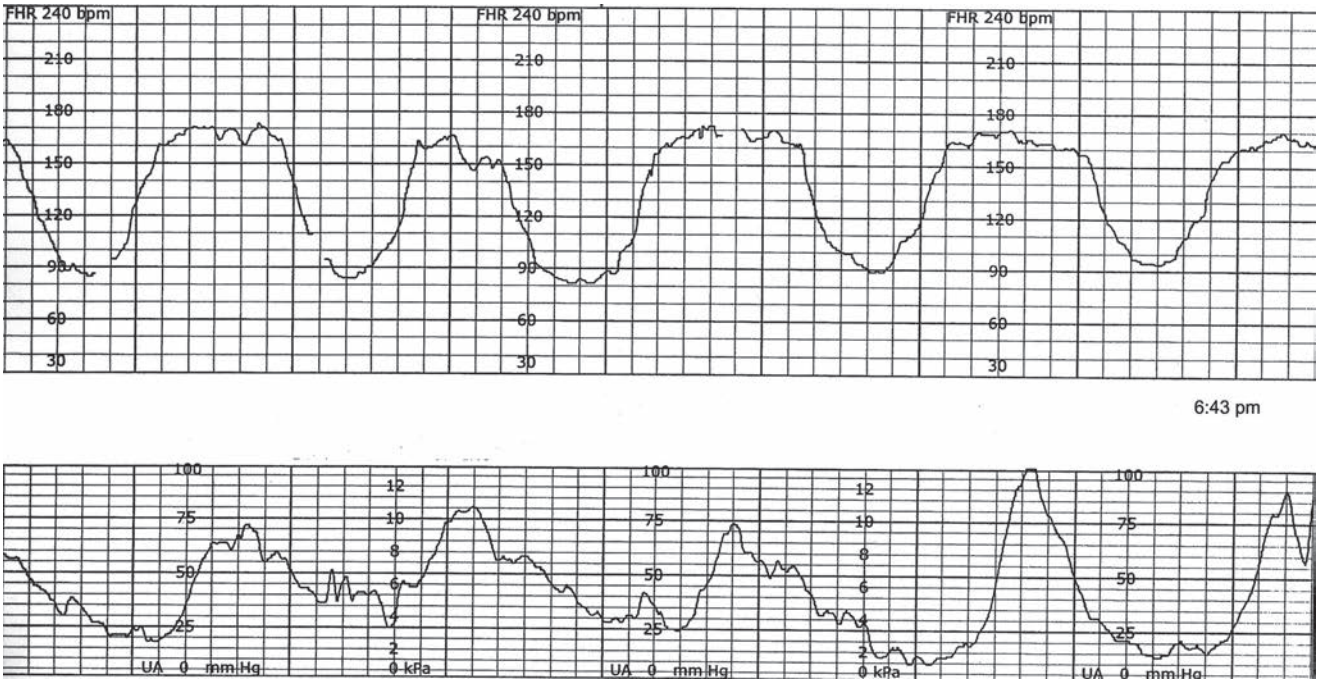


Figure 14.9. Recurrent decelerations with a rising baseline and decreasing variability.

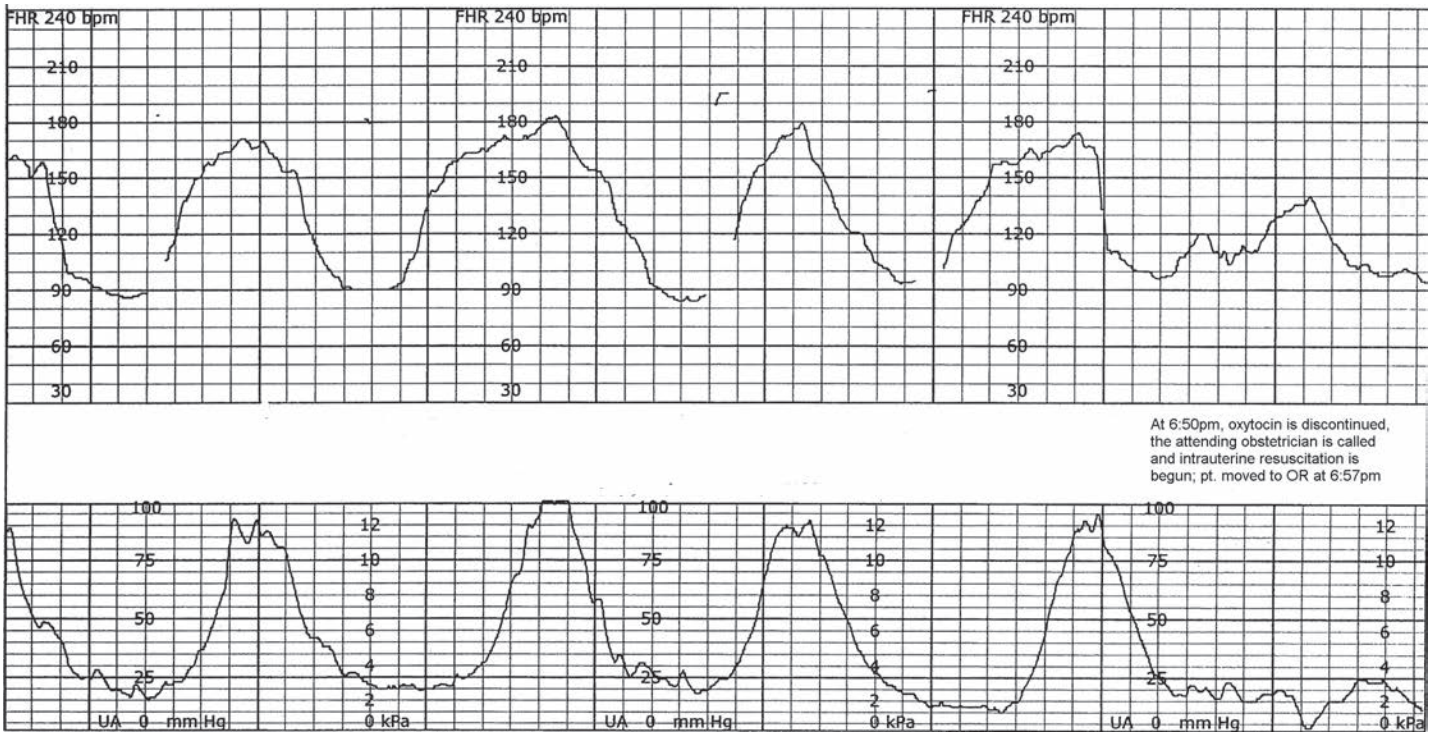


Figure 14.10. Immediately prior to transfer to operating room.

and continued to increase oxytocin even as the FHR tracing deteriorated. Understanding the mechanisms of different deceleration patterns and the progression from hypoxemia to metabolic acidemia is key to effective clinical practice. Clinicians must recognize that moderate variability and/or FHR accelerations are indicative of the absence of fetal metabolic acidemia at the time of observation (15–17) and be able to apply this knowledge to tracing evaluation as well as articulate this understanding. The nurse and nurse-midwife in this case failed to recognize the potential for deterioration of fetal acid-base status as the decelerations increased and the variability decreased.

Compounding the problem of tracing interpretation in this case is the continuation of oxytocin. Tachysystole is currently defined as greater than five contractions in a 10-minute window, averaged over 30 minutes, and this definition applies to spontaneous as well as stimulated labors (15,16). The National Institute of Child Health and Human Development (NICHD) workshop panel specifically stated that frequency of contractions (the basis for identification of tachysystole) was only a partial assessment of uterine activity, and the panel noted the importance of other factors, such as duration, strength, and relaxation time, in the clinical evaluation of uterine activity (16). Oxytocin has been designated as a high-alert medication by the Institute for Safe Medication Practice (18). Clinicians must be able to clearly articulate their assessments of uterine activity and, in cases involving the use of oxytocin or other uterine stimulants, their management (and rationale for management) of tachysystole when it does occur. The NICHD panel also recommended further clarification as to the presence or absence of FHR decelerations in association with periods of tachysystole. Clinical recognition of the significance of late, variable, and prolonged decelerations, as well as the *absence* of clinically significant decelerations, will assist clinicians in their interpretation of uterine activity. Use of a multidisciplinary, standardized protocol that includes a standardized checklist for oxytocin management has been described by Clark et al. (6), resulting in improved outcomes and a significant reduction in claims of obstetric liability.

Second-stage labor management is another issue in this case example. Evidence suggests the use of open-glottis pushing techniques and/or allowing passive descent in the second stage to support fetal oxygenation (19). Improvement of FHR tracings in the second stage may result by simply redirecting or discontinuing pushing efforts, decreasing or discontinuing uterine stimulants if indicated, and maternal position changes and intravenous fluid support. Figure 14.11 illustrates a second-stage pattern where the patient is on her back with legs drawn up and using coached closed-glottis pushing repeatedly during contractions. Figure 14.12 shows the rapid response of the FHR tracing to the following simple nursing interventions: taking a break from pushing, turning the patient to the left side, and providing an intravenous fluid bolus.

Finally, this case again exemplifies the importance of team communication. The physician was not made aware of the FHR tracing until there was no other option than emergent cesarean. Had the nurse and/or nurse-midwife provided ongoing updates related to the deteriorating tracing and the lack of progress, earlier intervention and resuscitation may have resulted in a different outcome.

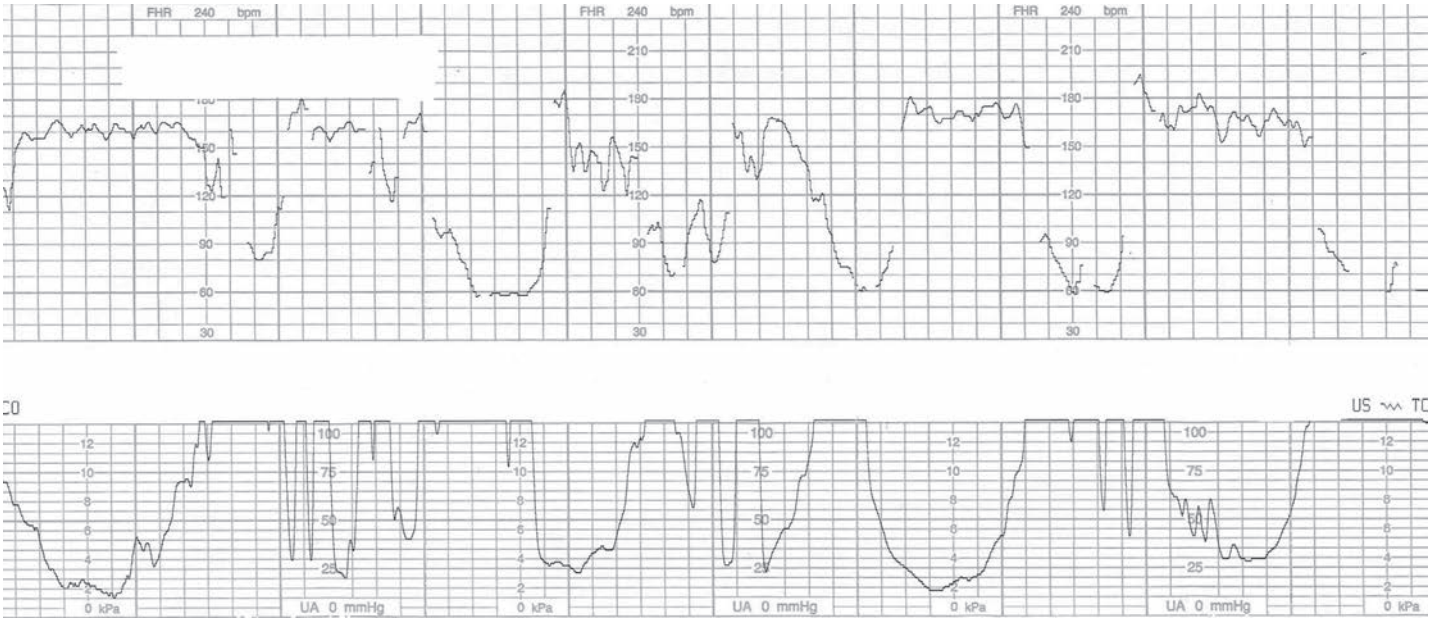


Figure 14.11. Second-stage fetal heart rate pattern with patient pushing on her back and using long Valsalva pushing efforts.

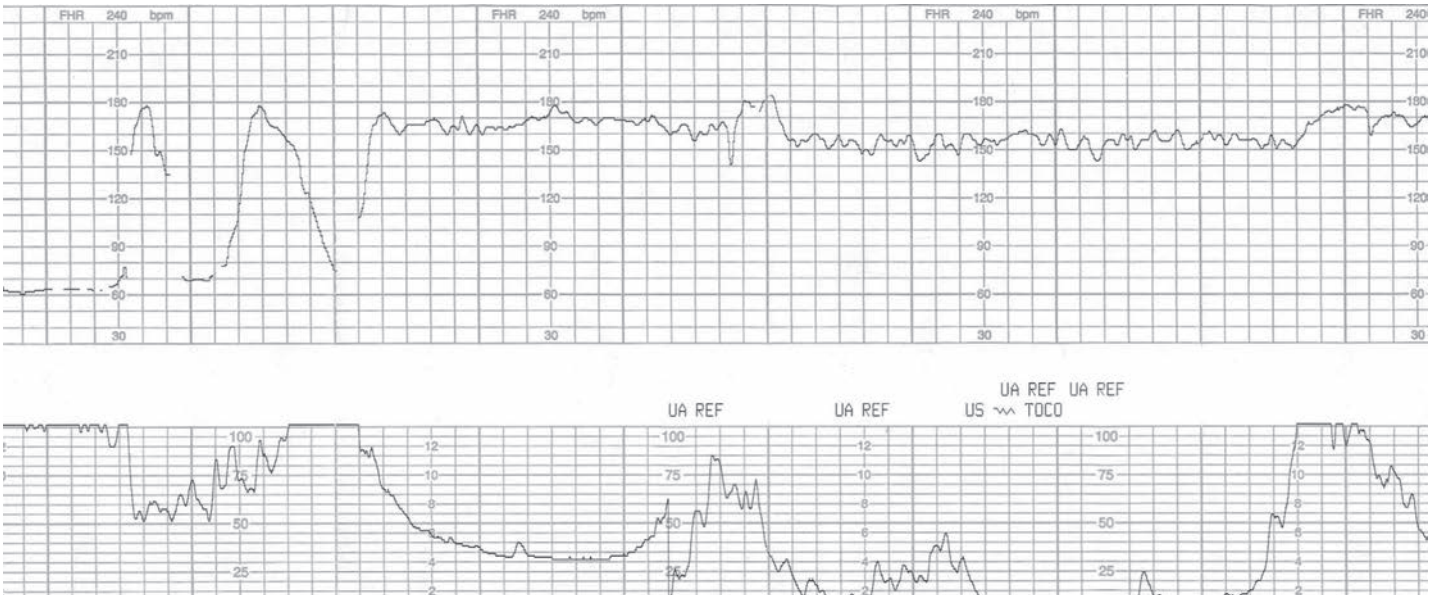


Figure 14.12. Note rapid improvement of fetal heart rate tracing following cessation of pushing, left side-lying position, and intravenous fluid bolus.

Case Example No. 4

Figures 14.13–14.19 illustrate another potential problem seen during labor, often in the second stage. This patient had been admitted for an induction of labor due to postdate pregnancy. She progressed to complete and had a somewhat prolonged second stage, but clinicians were unconcerned with the FHR tracing during active pushing, noting that there were "accelerations" during pushing efforts. The infant delivered vaginally, with Apgar scores of 0-0-0 and no response to full resuscitation. Most of the second-stage FHR tracing was actually a recording of maternal heart rate (MHR). The sudden shift in baseline from 150 to 120, along with the disappearance of commonly seen variable decelerations, and the unusual appearance of what appeared to be FHR accelerations but were really accelerations of maternal pulse may be obvious in hindsight but are easily missed in actual practice.

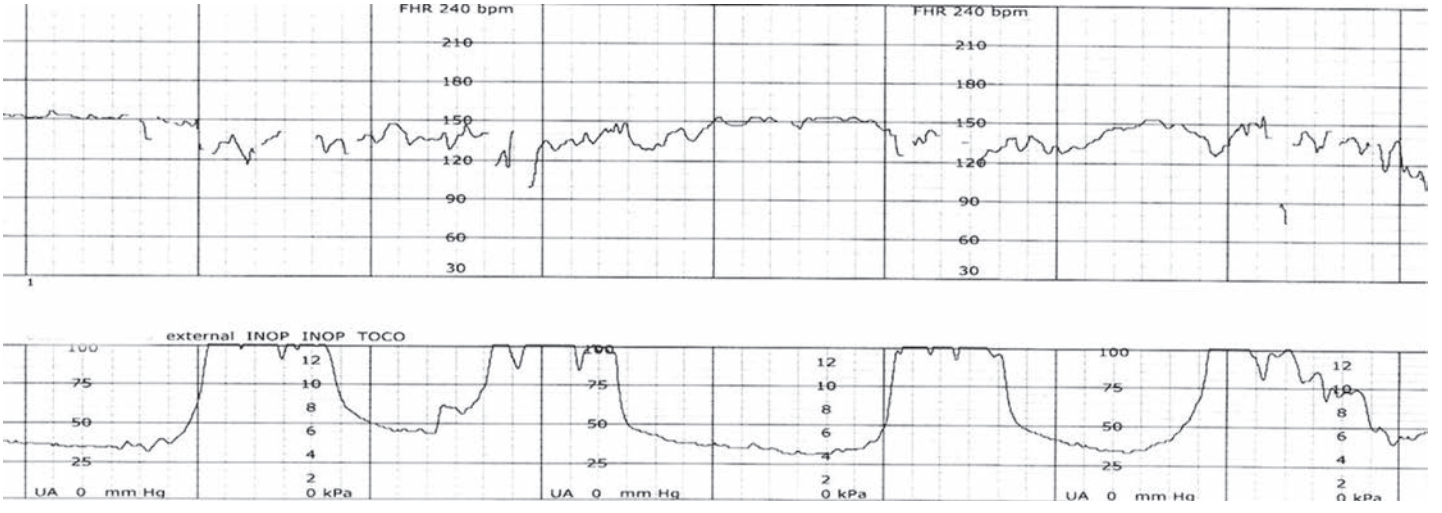


Figure 14.13. Beginning of second stage, note sporadic loss of signal.

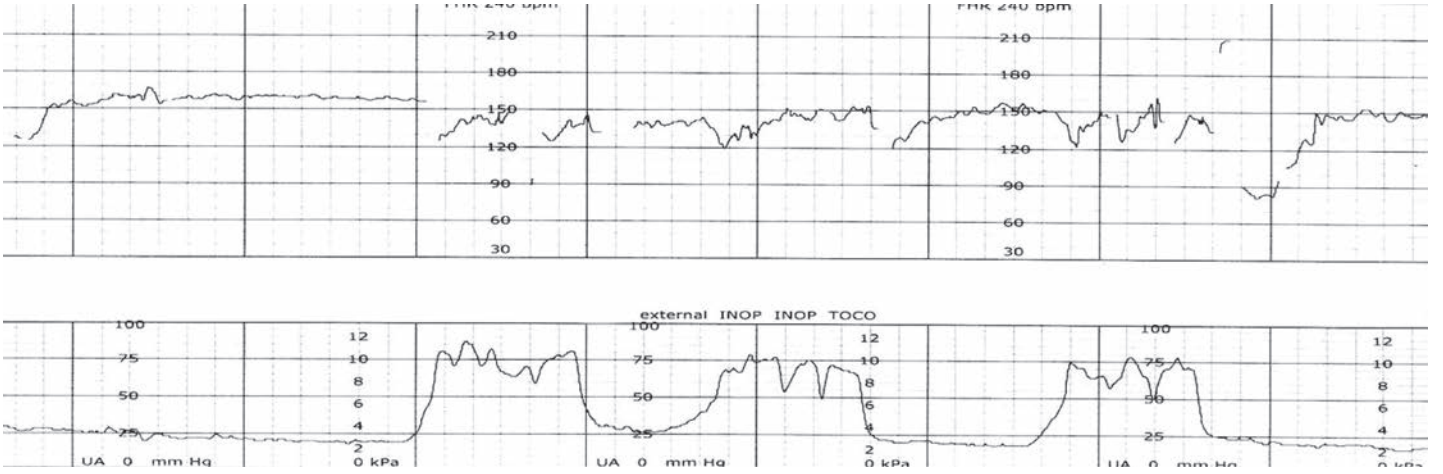


Figure 14.14. Second stage continues, note baseline of 150 with variable decelerations near end of tracing.

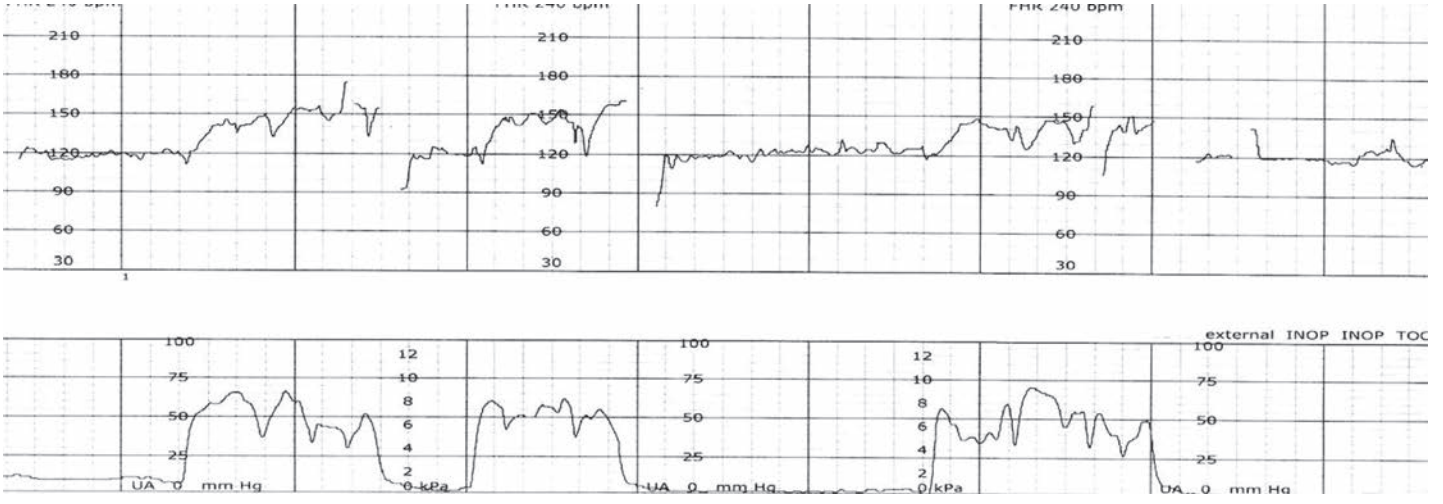


Figure 14.15. Note change in "baseline" to 120 BPM with "accelerations" during pushing.

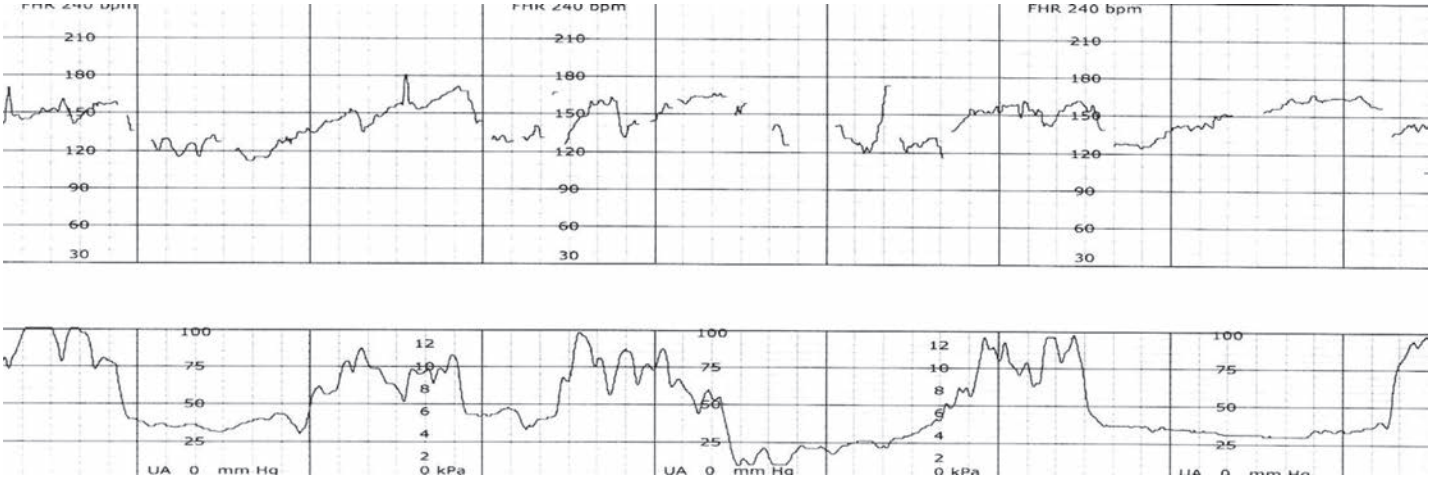


Figure 14.16. Pushing continues, this is maternal heart rate recorded as fetal.

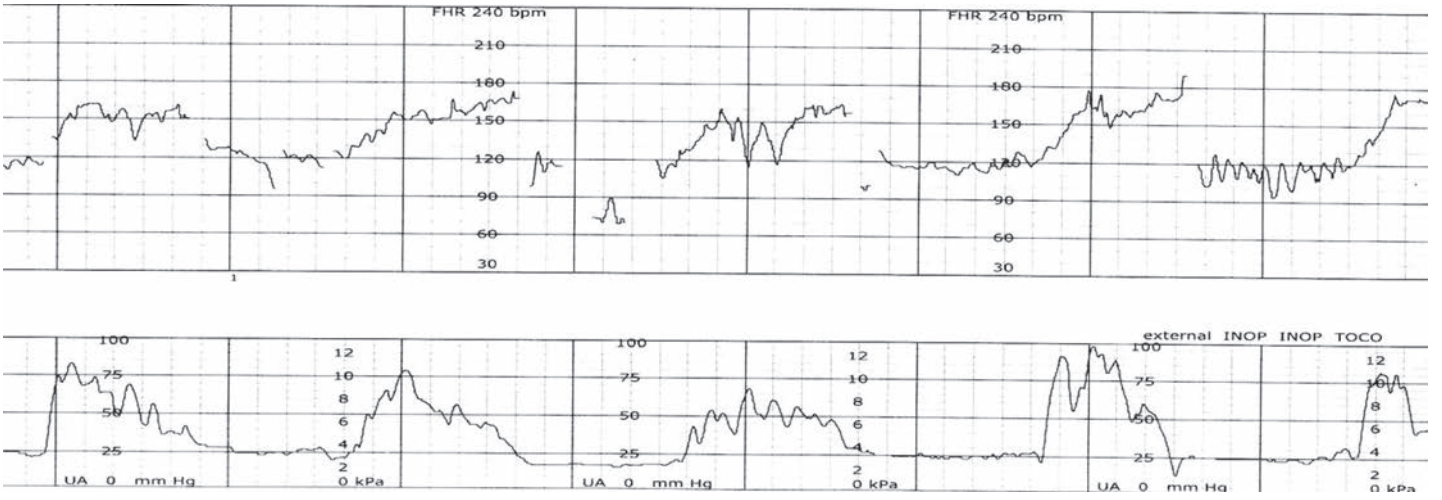


Figure 14.17. Pickup of maternal heart rate continues, last known fetal heart rate was at beginning of second stage.

In addition to signal ambiguity with MHR, signal coincidence with multiple gestations can also be problematic. Identification and clinical management of both of these issues is detailed in Chapter 4. Regardless of the etiology, clinicians should confirm the validity of the FHR signal every time they make an assessment of the tracing, as shown in the standardized management model described later in this chapter. Documentation of recognition of signal coincidence and/or signal ambiguity, along with corrective actions taken by nursing or medical staff, is a crucial part of record keeping and will assist the clinicians when questions arise regarding validity of the FHR data.

Loss of contiguous tracing and gaps in FHR tracings also occur commonly in the second stage and may be seen frequently in labor during placement of epidural anesthesia (Fig. 14.20). When the patient is repositioned and the FHR tracing is again recording, it is not uncommon to see late or prolonged decelerations due to maternal hypotension (Fig. 14.21). While the majority of these FHR changes are easily resolved by correction of maternal hypotension, clinicians should not hesitate to place an internal fetal electrode if the tracing continues to be questionable. Use of intermittent monitoring during periods of ambulation, during use of alternate measures for pain relief such as the birthing ball or a warm bath/shower, or even simply removing the patient from the monitor to use the bathroom can also lead to allegations regarding adequacy of monitoring. Plaintiffs may attempt to portray these

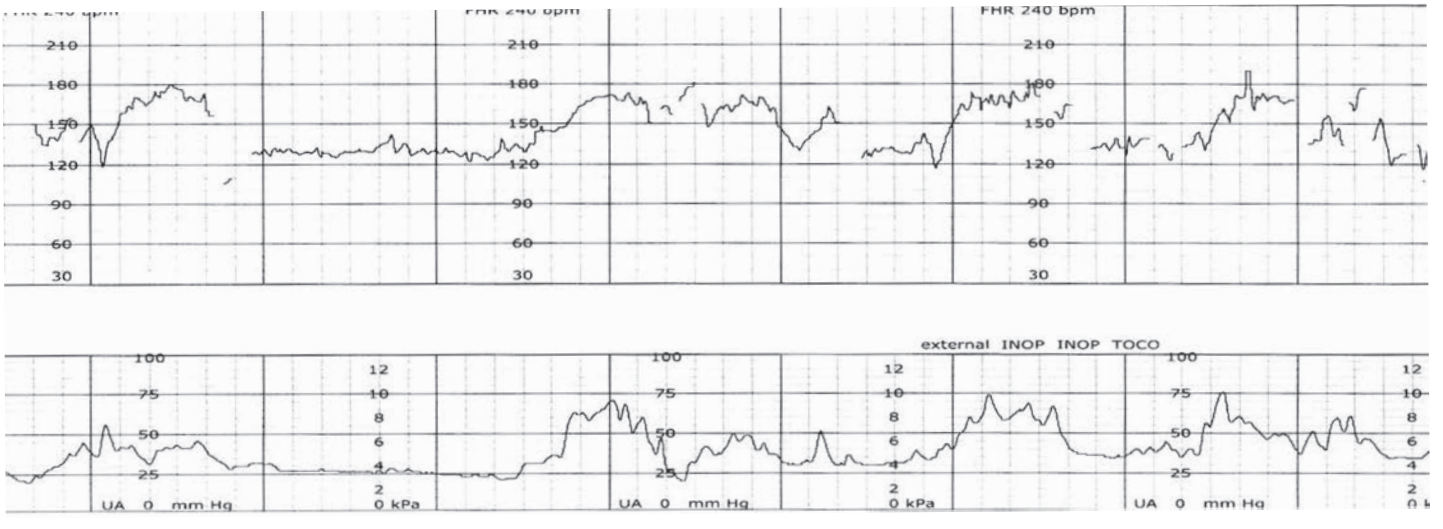


Figure 14.18. Note the broad accelerations during pushing, consistent with maternal pulse that increases with pushing efforts.

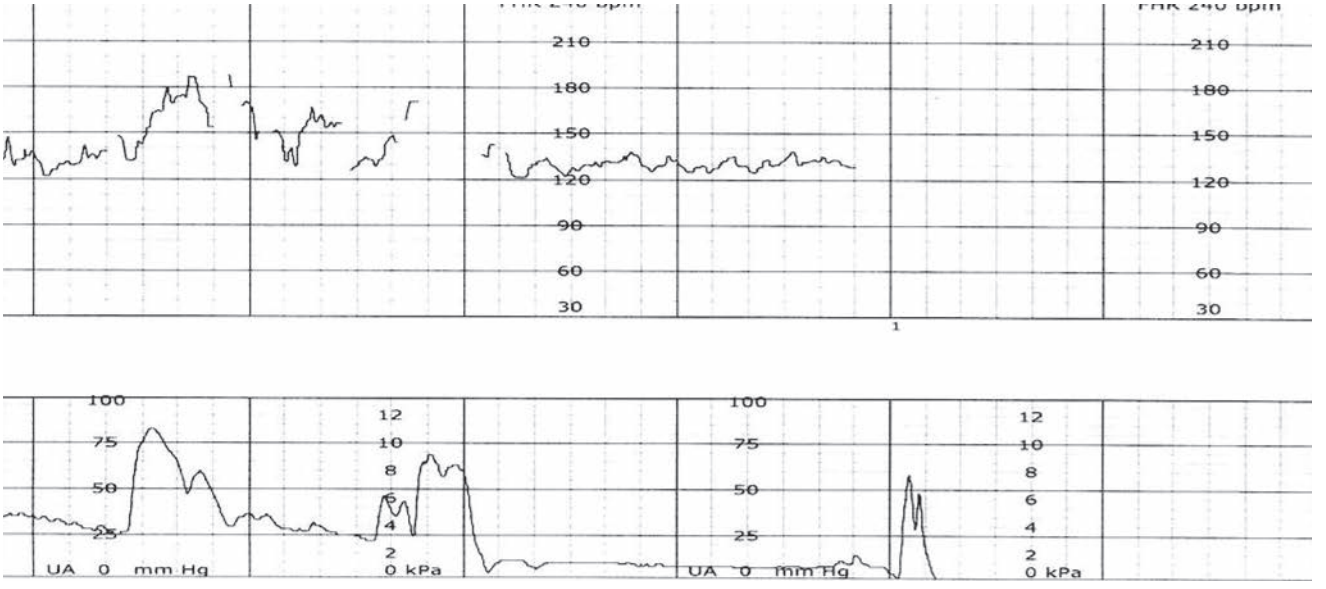


Figure 14.19. Spontaneous delivery of infant with 0-0-0 Apgar score.

commonly occurring monitoring lapses as "inadequate monitoring" or even seek experts to speculate that the gaps may hide critical FHR decelerations that clinicians should have attended to with resuscitation. Nursing documentation that shows recognition of the gaps and adjustment of the monitors to obtain an adequate tracing is essential. To avoid problems, reduce risk, and provide care that is both appropriate and defensible, clinicians should have clear guidelines or policies for intermittent monitoring that identify appropriate patient selection, frequency and length of intermittent monitoring, and utilization of auscultation of FHTs per professional guidelines (17,20,21) for periods in which continuous EFM is not being employed.

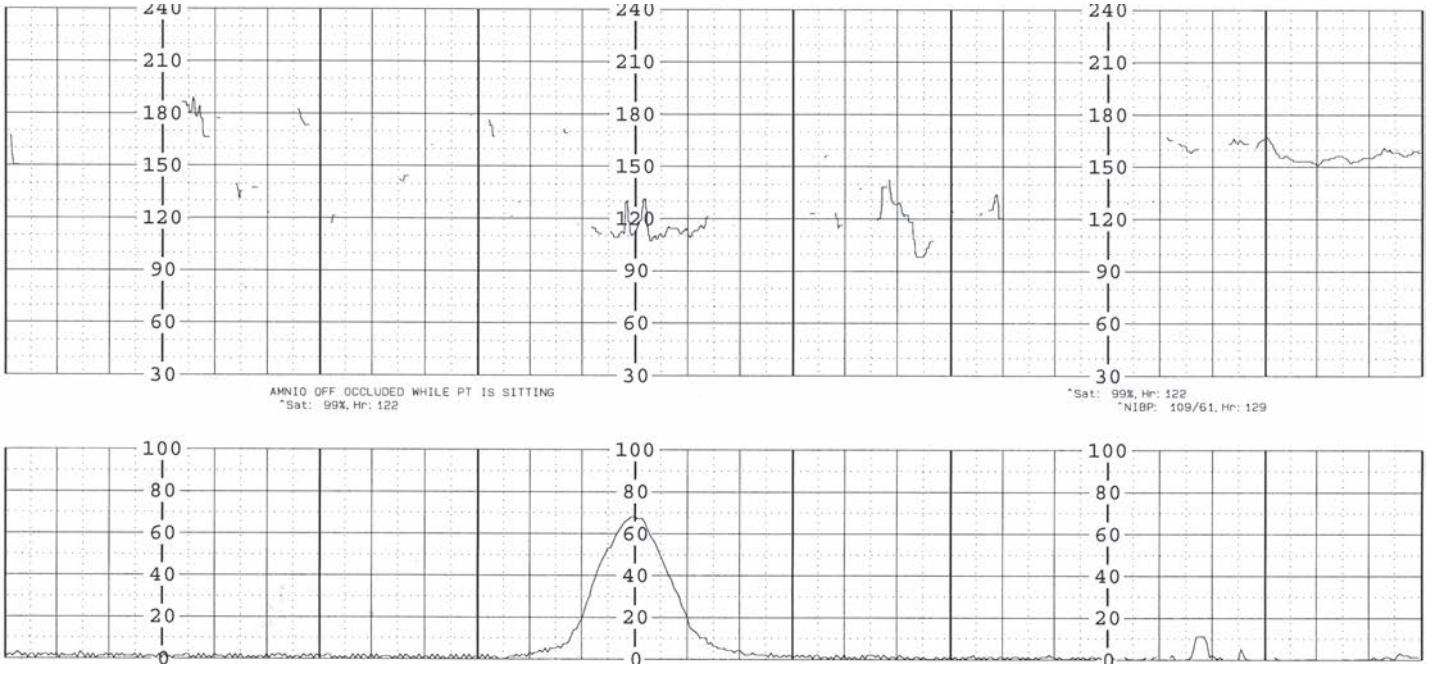


Figure 14.20. Loss of signal with external Doppler during epidural placement.

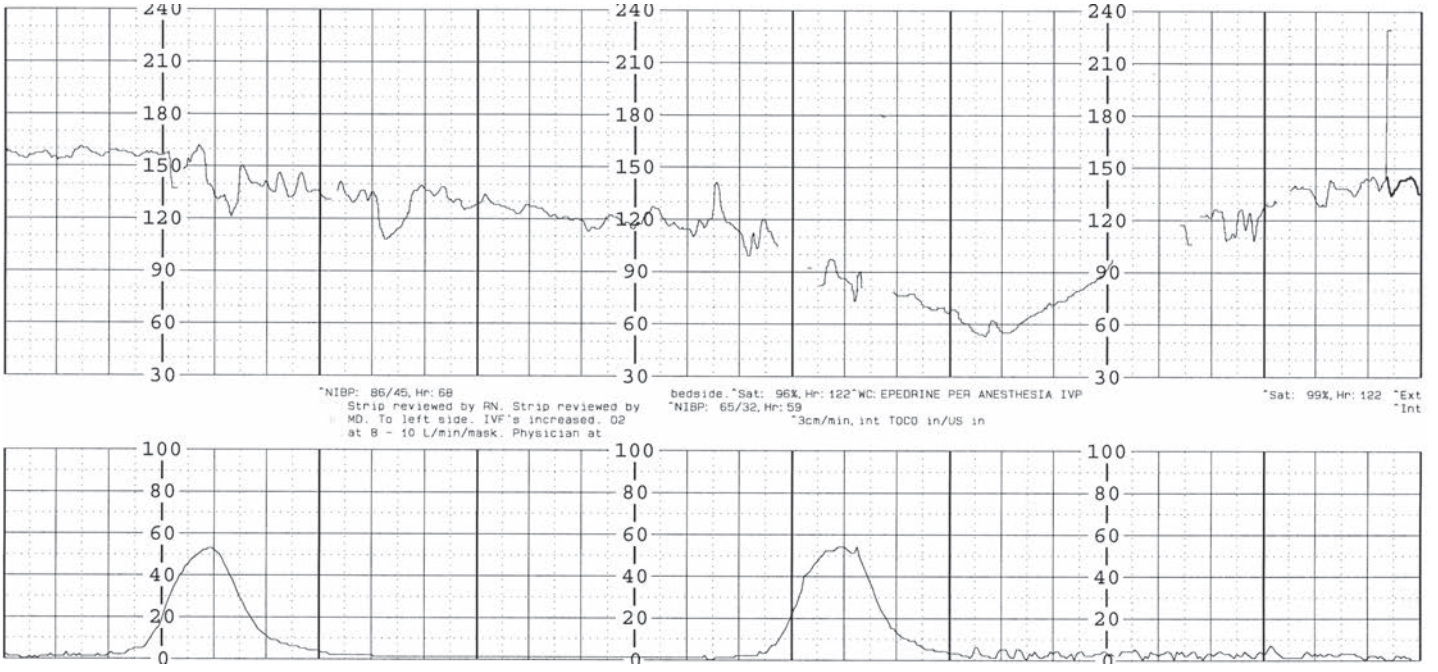


Figure 14.21. Prolonged deceleration due to maternal hypotension following epidural. Note nursing documentation of strip review and interventions. Also note placement of internal fetal electrode near end of tracing.

Case Example No. 5

A 21-year-old G3P2002 with a history of cerclage placement at 29 weeks was admitted at 41 weeks' gestation for induction of labor. She developed a fever of 102.8 and the FHR tracing became tachycardic with periods of decreased variability. In addition to EFM, fetal scalp sampling was obtained (Figs. 14.22 and 14.23). She received antibiotics during labor and was delivered by cesarean section. Apgar scores were 1, 3, and 4 at 1, 5, and 10 minutes, respectively. Cord gases were obtained with the following results: Arterial pH was 7.11, with a pCO_2 of 62, and HCO_3 of 20; venous pH was 7.30, with a pCO_2 of 42, and HCO_3 of 21. Placental pathology revealed chorioamnionitis and funisitis. The initial diagnosis by the admitting neonatologist was HIE even though the cord gas data and later course made fetal inflammatory response syndrome (FIRS) the more likely diagnosis. The infant went on to develop spastic quadriplegia and litigation ensued. Although all defense experts agreed that this was not related to intrapartum events and that the initial diagnosis of HIE was incorrect, the plaintiff was able to show the records of 14 subsequent treating physicians that included and referenced the original diagnosis of HIE. The case was settled during trial.

Like this case, the management of the patient who presents on admission with either a Category III tracing or a Category II tracing that lacks accelerations and/or moderate variability is a rare but challenging situation for the obstetric team. Prompt evaluation, corrective measures, and a decision regarding expedited delivery by the physician are the appropriate response. The inclusion of fetal stimulatory testing may be helpful in the clinical decision-making process. Should a depressed infant be delivered, a frank discussion with the parents at some point following delivery attempting to explain the potential causes of the clinical outcome is very important (22). In this situation, the disclosure process is not about medical or nursing error but rather serves to assist the family in understanding the outcome. Evaluation of the initial tracing, corrective measures, notification of various team members, and decisions regarding either expedited delivery or ongoing observations should be clearly documented in order to support evidence of a timely response. When infection is suspected, clear communication with neonatal clinicians by the obstetrician is important, as well as postdelivery communication between the treating neonatal team and the family.

Although in the preceding case example the normal cord gases at birth were not sufficient to counter the multiple neonatal and pediatric records referring to HIE, clinicians should consider obtaining cord gases for any delivery where resuscitation is provided. ACOG has identified a number of clinical situations where umbilical cord blood gases should be obtained, including any cesarean delivery for fetal indications, abnormal FHR tracings, low 5-minute Apgar scores, and intrapartum fever (23). Clinicians should obtain both venous and arterial samples. While the arterial is the critical sample to diagnose metabolic acidemia at birth, the venous sample is important in that it allows confirmation of the accuracy of the arterial sample. Umbilical cord gases and fetal acid-base concepts are discussed in Chapter 7. Cord gases can be particularly helpful in postdelivery review and assessments related to causation and

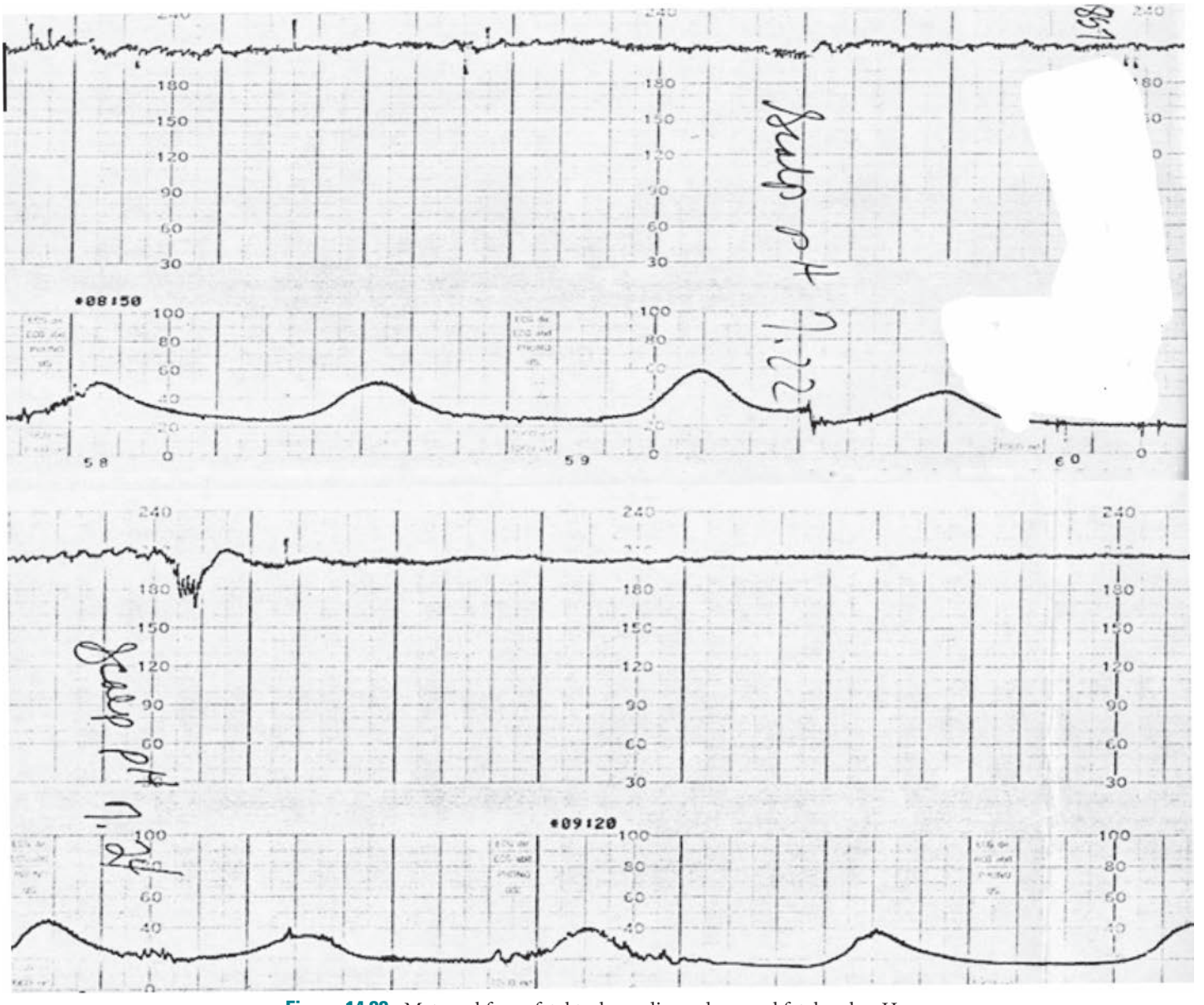


Figure 14.22. Maternal fever, fetal tachycardia, and normal fetal scalp pH.

may be valuable information in the immediate neonatal management.

Placental pathology can also be a helpful tool in the evaluation of causation. Examination of the placenta can provide valuable information regarding the etiology of neurologic injury, including associated factors such as FIRS (24). Having a policy for identifying placentas that should be sent for further examination may be beneficial. At one large academic institution, all placentas are delivered to the pathology department with basic clinical and identifying information and are separated into two groups: those for examination based on indication and the remainder that are placed on a 7-day hold. The placentas on hold are kept in a locked refrigerator and discarded unless the neonate is admitted (or readmitted) to neonatal intensive care or the mother is readmitted with a complication, either of which will trigger a full examination (25). Table 14.1 provides a sample list of indications for placental examination. Although this approach does generate costs associated with the triage and storage of placentas, the cost savings should even one case be averted due to placental pathology may easily make any economic argument against such a policy moot.

The previous case example illustrated the issue of causation in brain-damaged neonatal cases. Clinicians must understand the causes of neonatal encephalopathy, and the overwhelming data showing that HIE is rarely related to intrapartum events alone. Umbilical cord gases and placental pathology are two of the important factors to consider when evaluating neonatal encephalopathy in the context of intrapartum events. Chapter 3 provides a detailed review of HIE and the critical factors

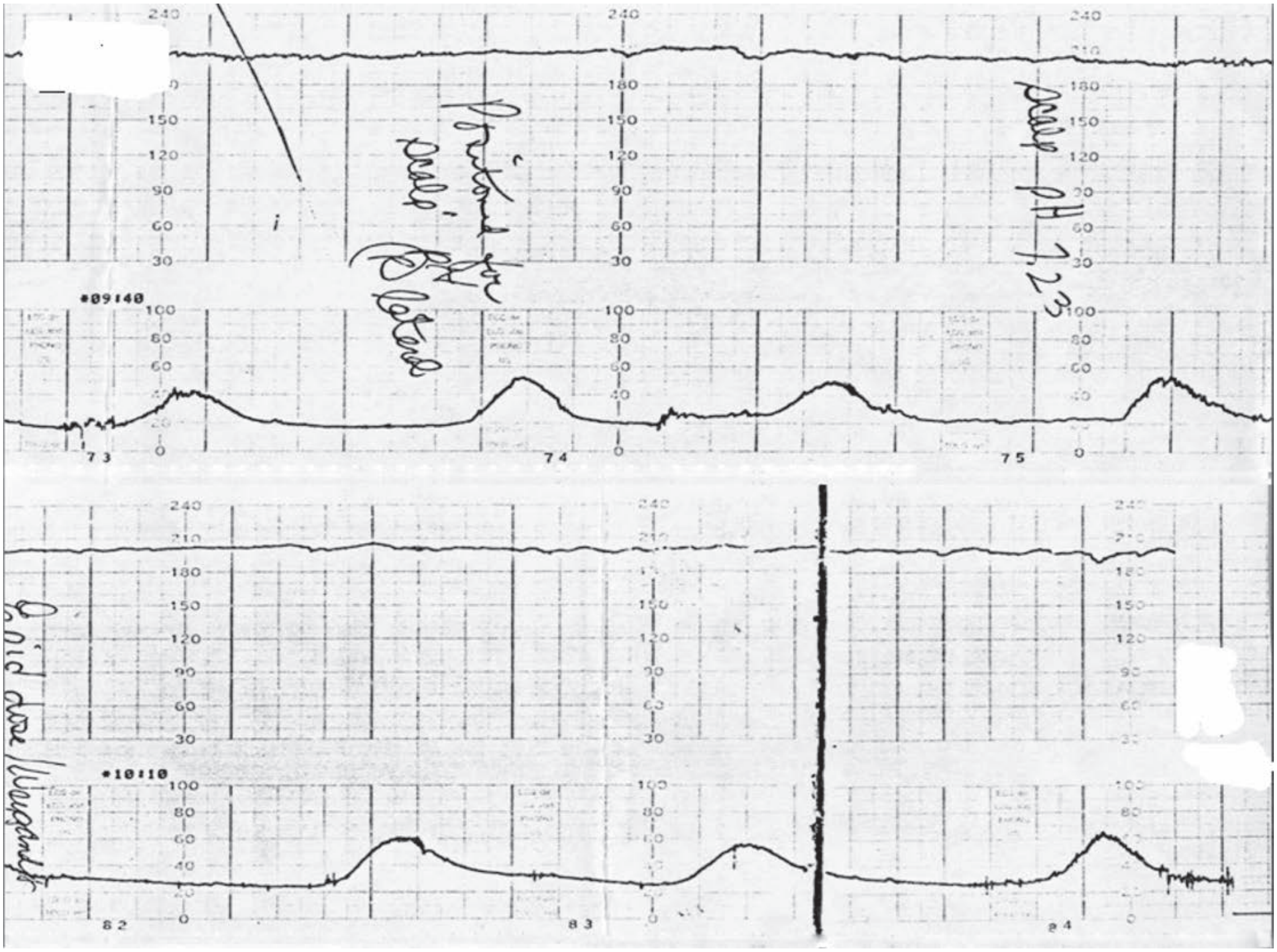


Figure 14.23. Continued tachycardia with normal scalp pH immediately prior to delivery.

related to causation, including early brain imaging studies and the importance of pediatric neurology when examining issues of causation in brain-damaged neonatal cases.

Case Example No. 6

Thirty-year-old G2P1001 with a complaint of decreased fetal movement at 39 2/7 weeks' gestation was seen in labor and delivery. The nurse documented "questionable sinusoidal tracing" (Figs. 14.24 and 14.25) and notified the physician. The physician performed an ultrasound at the bedside, which revealed an AFI of 22, no fetal movement, and no fetal breathing, for a biophysical profile (BPP) score of 4 (2 for fluid, 2 for tone). Patient was counseled and consent obtained for primary cesarean section. Physician's progress note included recognition of the sinusoidal FHR tracing, abnormal BPP, and documentation of her discussion with the patient and family as well as involvement of the neonatal team for delivery. Infant delivered was pale but had Apgar scores of 8 and 9. Initial newborn hemoglobin and hematocrit were 4 and 13, respectively. A Kleihauer-Betke was obtained from maternal blood and revealed 5% fetal cells, consistent with a fetomaternal hemorrhage. The infant was given transfusions and followed in neonatal intensive care. Infant was discharged on day three with no apparent sequelae, follow-up by neonatology was normal at 6, 12, and 18 months. Following the delivery, both the neonatologist and the obstetrician met with the patient and her family and explained the mechanism of the fetal anemia as well as the response to the sinusoidal tracing.

Although this case did not involve litigation, it illustrates the importance of teamwork, communication, timely decision making, and appropriate documentation. When the nurse initially viewed the tracing, she suspected a sinusoidal pattern even though the patient was essentially low risk. Rather than wait, she immediately notified the physician, who responded

TABLE 14.1 Indications for placental pathology

Obstetric
Intrauterine fetal demise
Intrapartum fever
Abnormal cord gases
Abnormal fetal heart rate tracing
Maternal infection
Maternal medical complications
Maternal obstetric complications
Previous history of poor pregnancy outcome
Amniotic fluid abnormalities
Abnormal appearance of placenta at time of delivery
Neonatal
Prematurity
Fetal growth restriction
Admission to neonatal intensive care unit
Low Apgar scores
Congenital anomalies (known or suspected)
Infection

by performing a bedside evaluation, including ultrasound examination. When the physician made the diagnosis of a sinusoidal FHR pattern and an abnormal BPP, she informed the patient and family, obtained consent for cesarean section, and worked with nursing staff to facilitate mobilization of the response team. The decision to proceed to cesarean was made only after the physician had counseled the patient and obtained informed consent, and although some of the team mobilization was occurring concurrently, the actual decision time followed the full discussion with the patient. This was also the time noted in the physician documentation. Although there is no law or statute that designates a 30-minute time frame from "decision to incision," the Guidelines for Perinatal Care note that "the consensus has been that hospitals should have the capability of beginning a cesarean delivery within 30 minutes of the decision to operate" (26) and clinicians may find it difficult to defend cases where this time frame was not met. A confirmed sinusoidal pattern is a Category III FHR pattern that requires expedited delivery if uncorrected, which is exactly what the obstetric team did in this case. There will always be situations where there are valid reasons for safely delaying the beginning of a cesarean section, and in those cases the attending physician should consider a progress note outlining the decision to wait and the status of the patient.

While the primary nurse stayed with the patient, the charge nurse notified the rest of the team including anesthesia and neonatology. Following the delivery, the primary nurse and obstetric physician performed a routine "debriefing" to review the events, interventions, and timeline prior to completing their delivery summary documentation. This case is an example of a high reliability perinatal team in action and provides valuable lessons on some common liability issues.

Perinatal units that operate successfully with reduced error and injury have been described as high reliability organizations (HROs) (27). Recently, Knox and Simpson have updated the elements of perinatal HROs and differentiated high reliability from quality improvement. They cite

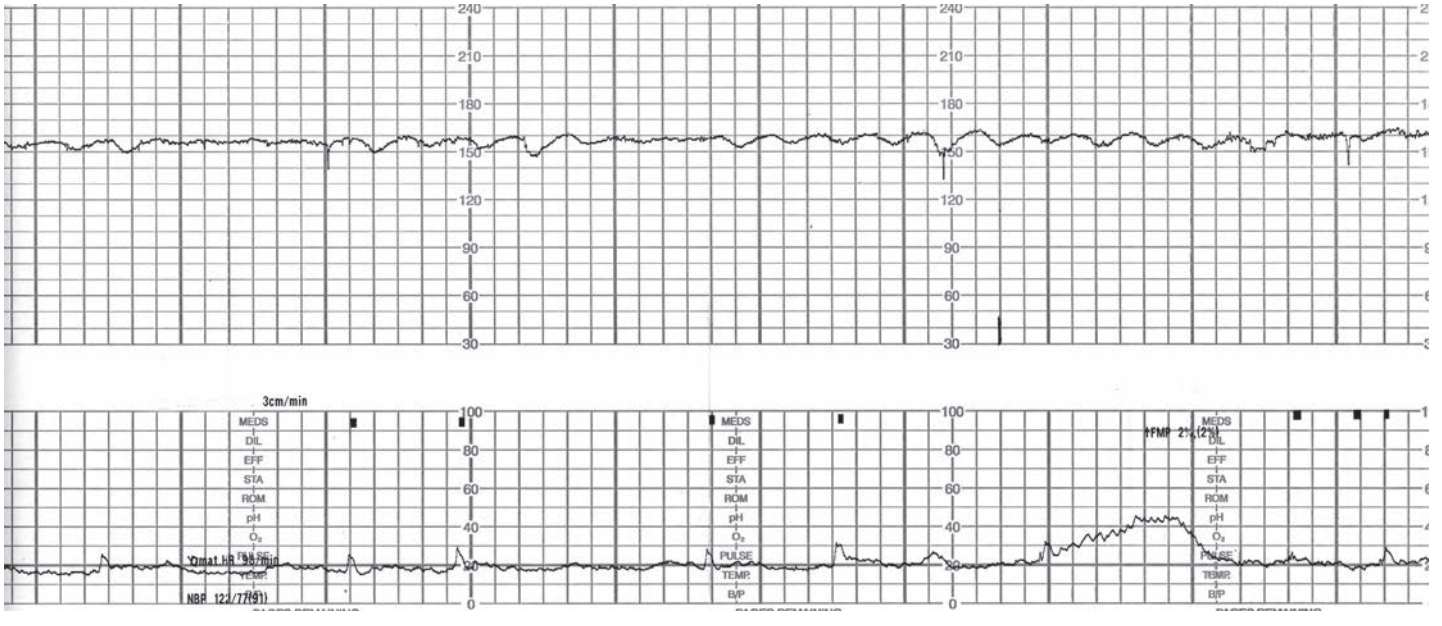


Figure 14.24. Initial admission fetal heart rate tracing, nurse notifies MD of "questionable sinusoidal tracing."

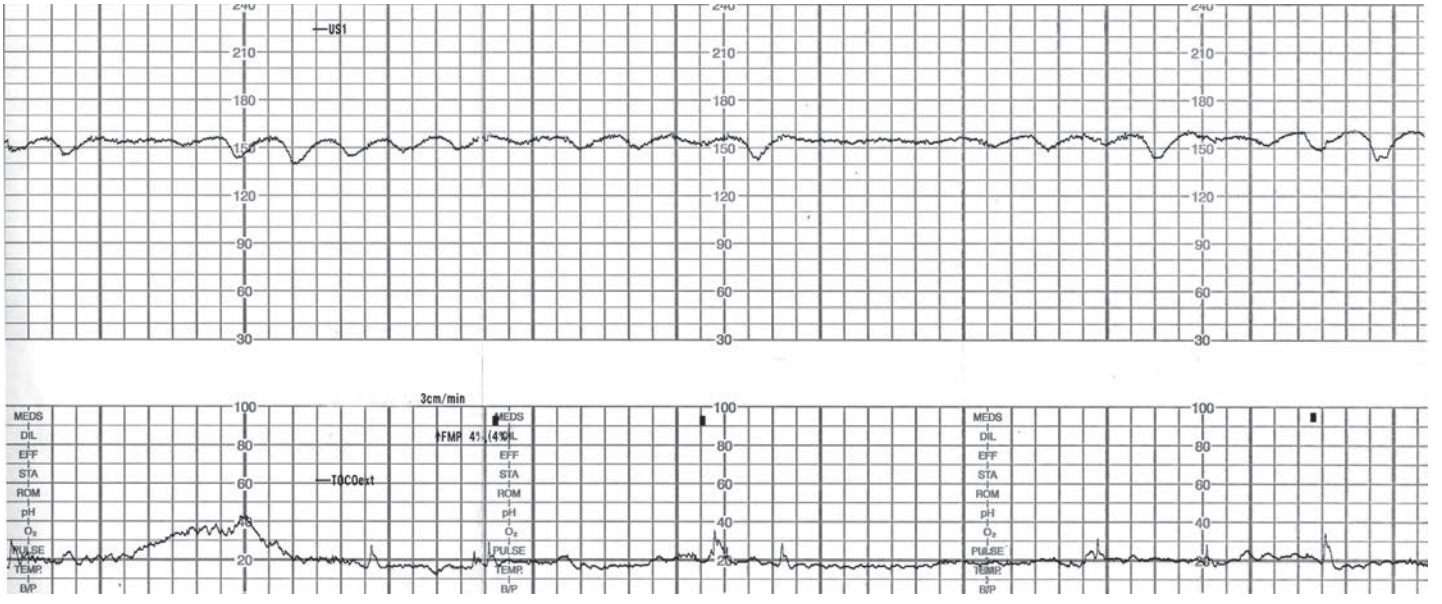


Figure 14.25. Physician at bedside evaluating tracing with nurse.

five fundamental concepts as essential for success: heightened awareness to operations and processes, avoiding of simplification of failure causation, interdisciplinary review of near-miss occurrences, leadership deference to expertise and elimination of hierarchy, and appropriate response and staff support when dealing with failures. They summarize high reliability as the "creation of a culture and processes that radically reduce system failures and effectively respond when failures do occur" (28). In the preceding case example, the nurse and obstetrician worked in concert with the rest of the perinatal team to identify, evaluate, and confirm an abnormal FHR tracing and provide timely and appropriate interventions. The physician was certainly the team leader, but the overall culture reflected a peer-to-peer rather than hierarchical approach. The utilization of a postevent debriefing allowed both process review and ensured lack of conflict between nursing and medical documentation related to the events.

Routine debriefing is another process seen in HROs and can be especially helpful following deliveries complicated by shoulder dystocia. Although generally unrelated to fetal monitoring, two specific liability issues seen in shoulder dystocia cases warrant succinct attention: (i) nursing preoccupation with FHR once the vertex is delivered and (ii) conflicting information regarding events in nursing versus medical documentation. In shoulder dystocia situations, once the vertex has been delivered and there is a delay with the delivery of the shoulders, the focus of the team must be on maneuvers to resolve the shoulder dystocia and mobilization of support team members for assistance and possible neonatal resuscitation. Continued attempts to find or record the FHR once the vertex is delivered are unnecessary and could delay important nursing actions such as patient positioning or suprapubic pressure. Following delivery, it is important that nursing and medical documentation accurately reflect the events, including maneuvers employed and time frames. A multidisciplinary checklist-style delivery note for use in shoulder dystocia cases has been evaluated (29), and implementation of this type of delivery summary provides an excellent template for debriefing and ensuring accuracy and completeness in delivery note documentation.

Case Example No. 7

This case involves a 22-year-old G2P1001 admitted for a trial of labor after a previous cesarean section. Continuous fetal monitoring was employed, and in the first stage, the FHR tracings were unremarkable (Fig. 14.26). During the second stage, FHR changes including prolonged decelerations with a rising baseline and loss of moderate variability were noted, and the decision for cesarean section was made (Fig. 14.27). External fetal monitoring was discontinued and the patient was moved to the OR. A delay occurred in the OR secondary to difficulties with the patient's epidural. No FHR was recorded or auscultated during the delay, which was approximately 20 minutes. Although no uterine rupture was noted, the infant was born depressed and cord gases were consistent with metabolic acidemia. Prompt neonatal resuscitation prevented any neonatal complications, and the infant was able to room-in with the mother postrecovery. Although this case did not result in litigation, it still provides valuable lessons for patient management. The hospital policy was to review all

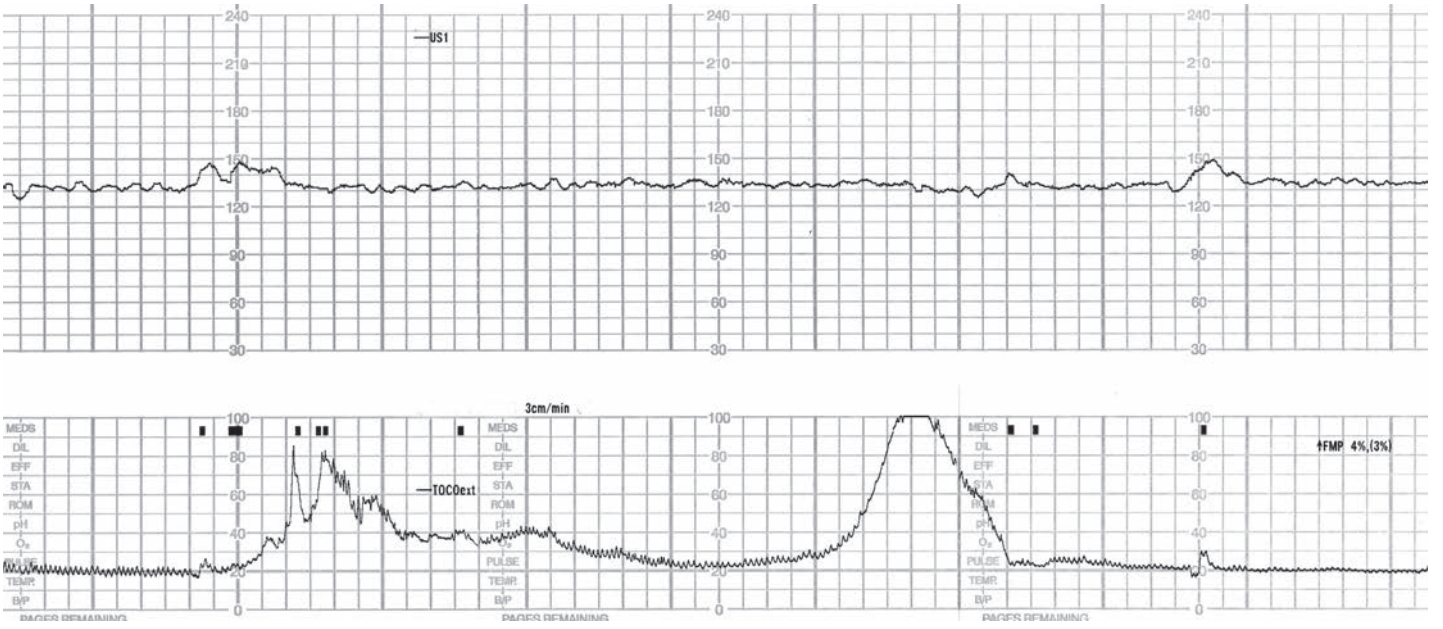


Figure 14.26. First-stage electronic fetal monitoring reveals normal fetal heart rate baseline, moderate variability, presence of accelerations, and no clinically significant decelerations.

nonscheduled cesarean sections as part of an ongoing safety program, and several issues arose during the review, including a lack of documentation regarding scar type on the inpatient record (no prenatal record had been made available until after delivery); failure to follow hospital guidelines regarding documentation of informed consent for vaginal birth after cesarean (information also contained in the prenatal record but not available until later); and the lack of fetal monitoring in the OR during the delay. Had this case resulted in hypoxic-ischemic damage to the neonate it would have been difficult to defend. Routine review of "near-miss" situations like the one this case presents allows the obstetric team to correct

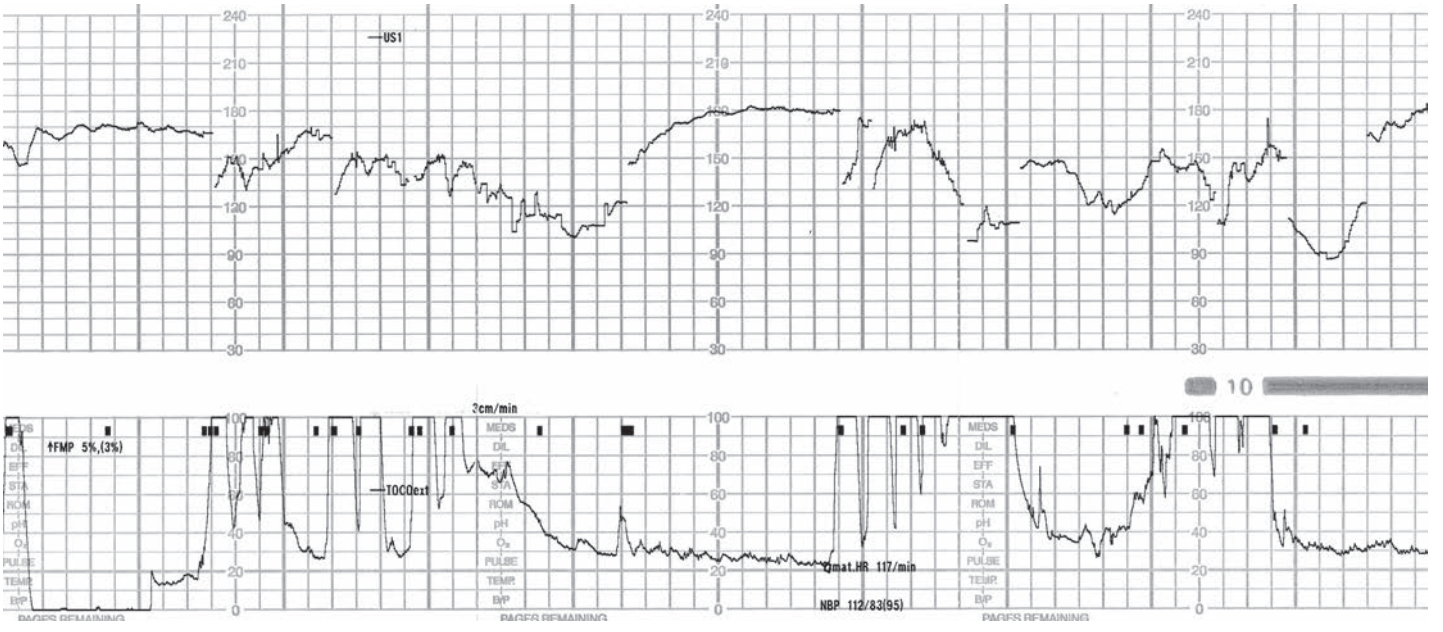


Figure 14.27. Fetal heart rate tracing in second stage: rising baseline with minimal variability and prolonged decelerations; decision is made to proceed to cesarean section.

potential errors in processes before a poor outcome occurs. Following the in-house case review several steps were taken related to trial of labor after cesarean (TOLAC). Nurses and physicians were reminded of the need for documentation on informed consent and scar type upon admission to labor and delivery, and nurses were provided with a review of the hospital policy on obtaining FHR data in the OR.

In summary, TOLAC is considered a highly litigious area. But fetal monitoring for patients undergoing TOLAC is not remarkably different than for any patient in whom heightened surveillance is indicated. Continuous EFM is indicated, although there is no evidence that internal monitoring is preferable to external monitoring when external monitoring is adequate. Nursing and medical staff must be aware that the appearance of clinically significant decelerations (late, variable, or prolonged) and bradycardia are the principal signs of impending uterine rupture (30). Nurses should request physician evaluation of decelerations that are increasing in frequency, depth, or duration. Even in situations where prompt response and emergency delivery are provided, severe metabolic acidosis and its sequelae may not be avoidable (31,32). Documentation of response times and interventions should be part of both the medical and nursing records, and utilization of debriefing following emergency cesarean section is important to avoid inadvertent conflicts in the record. Use of a multidisciplinary delivery summary for documentation may also be considered and may be beneficial for all patients, versus limiting such tools to emergency situations.

Case Example No. 8

A 17-year-old G2P0010 is admitted and progresses rapidly to complete dilation following epidural anesthesia. Over the following 30 minutes, the physician attempts to correct the tracing by repeatedly performing scalp stimulation during decelerations (Figs. 14.28 and 14.29). The tracing continues to deteriorate, and after 45 minutes, the patient is taken for cesarean section (Fig. 14.30). The infant is delivered 20 minutes later, and full resuscitation is provided. No cord gases are obtained, but the initial newborn blood gas reveals metabolic acidemia. Eventual diagnosis is HIE, and during litigation, the plaintiff's attorney asks both the physician and nursing staff questions about fetal scalp stimulation and receives conflicting answers. Although the nurses were uncomfortable with the repeated episodes of scalp stimulation, they did not know how to address the issue with the physician. At deposition, the nurses testified that they were aware of the proper use of scalp stimulation as a test, not a treatment, and could not explain why the physician was repeatedly using it. The physician maintained that scalp stimulation was indicated and appropriate, although was unable to cite any supporting literature. This conflict, as well as the delay in the decision to deliver and a number of other factors related to second-stage management, likely contributed to the ultimate outcome of the case, which was settlement by both the hospital and physician.

In the management of FHR tracings, one of the most misunderstood tools of fetal assessment is that of fetal

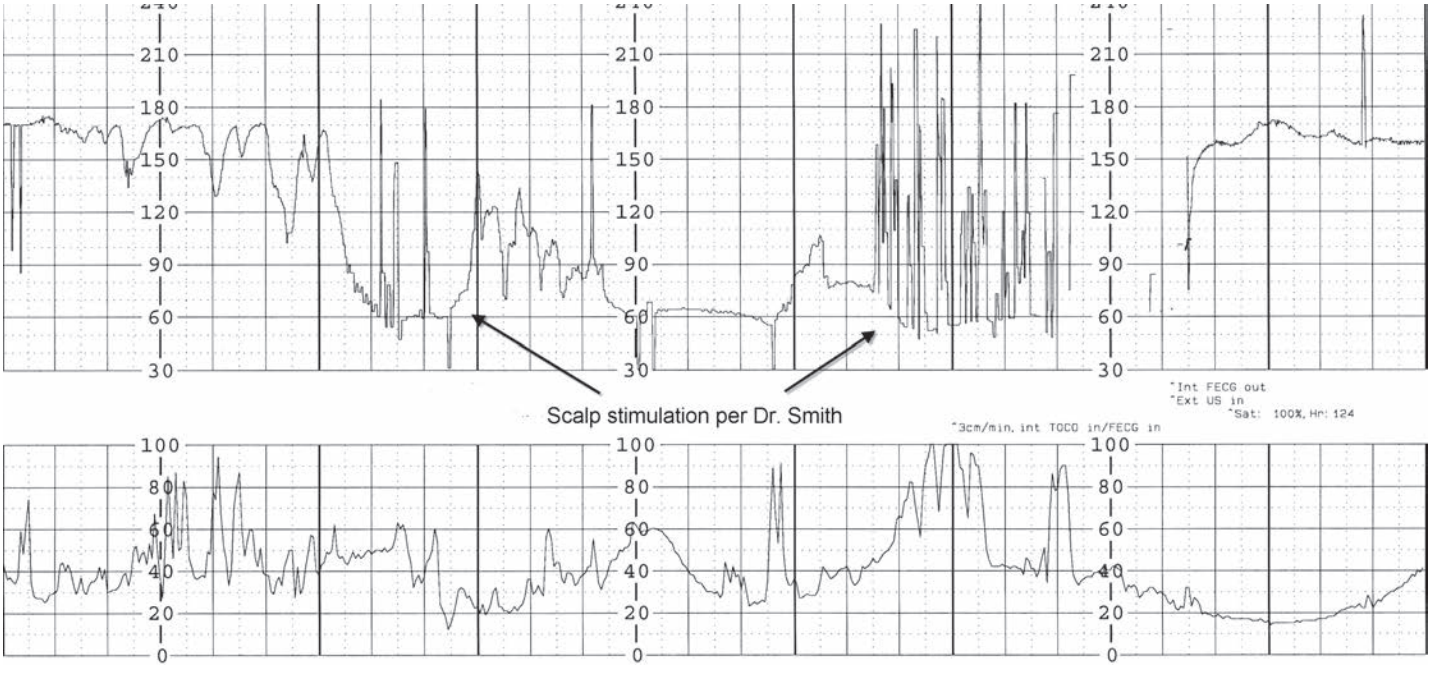


Figure 14.28. Scalp stimulation performed during a deceleration.

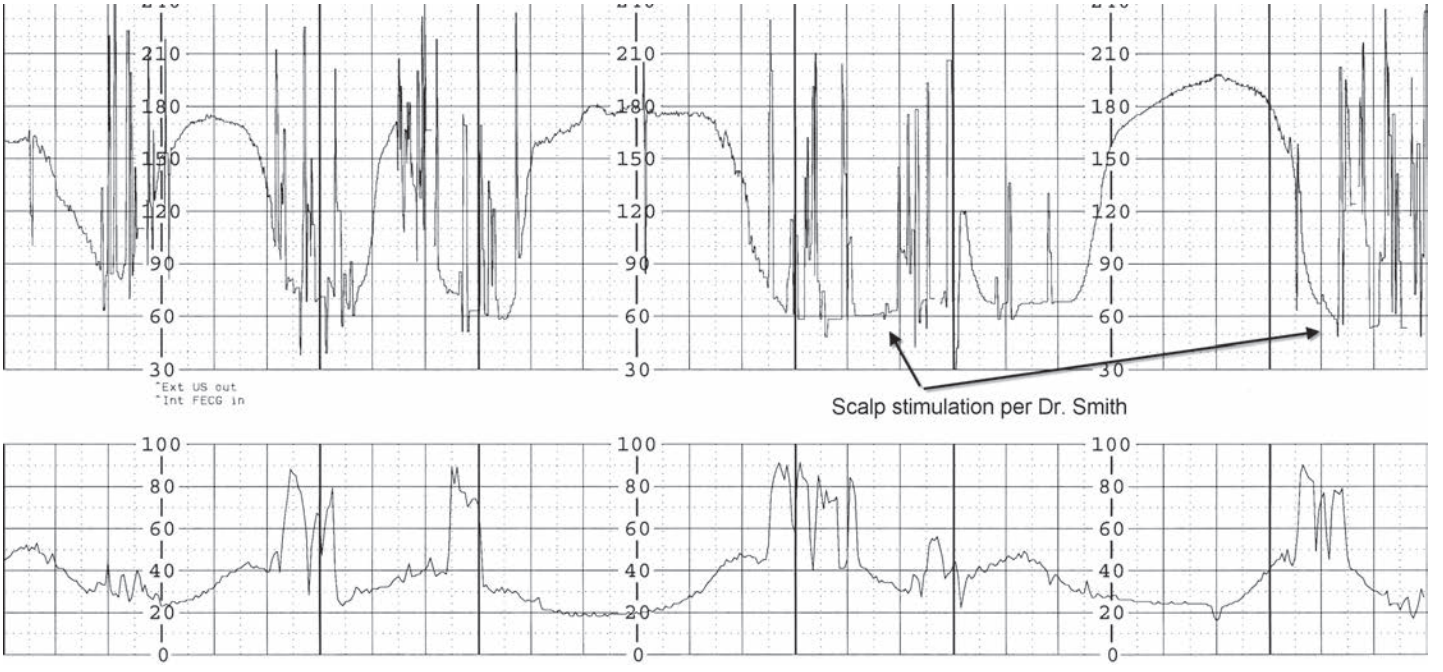


Figure 14.29. Repeated misapplication of scalp stimulation technique.

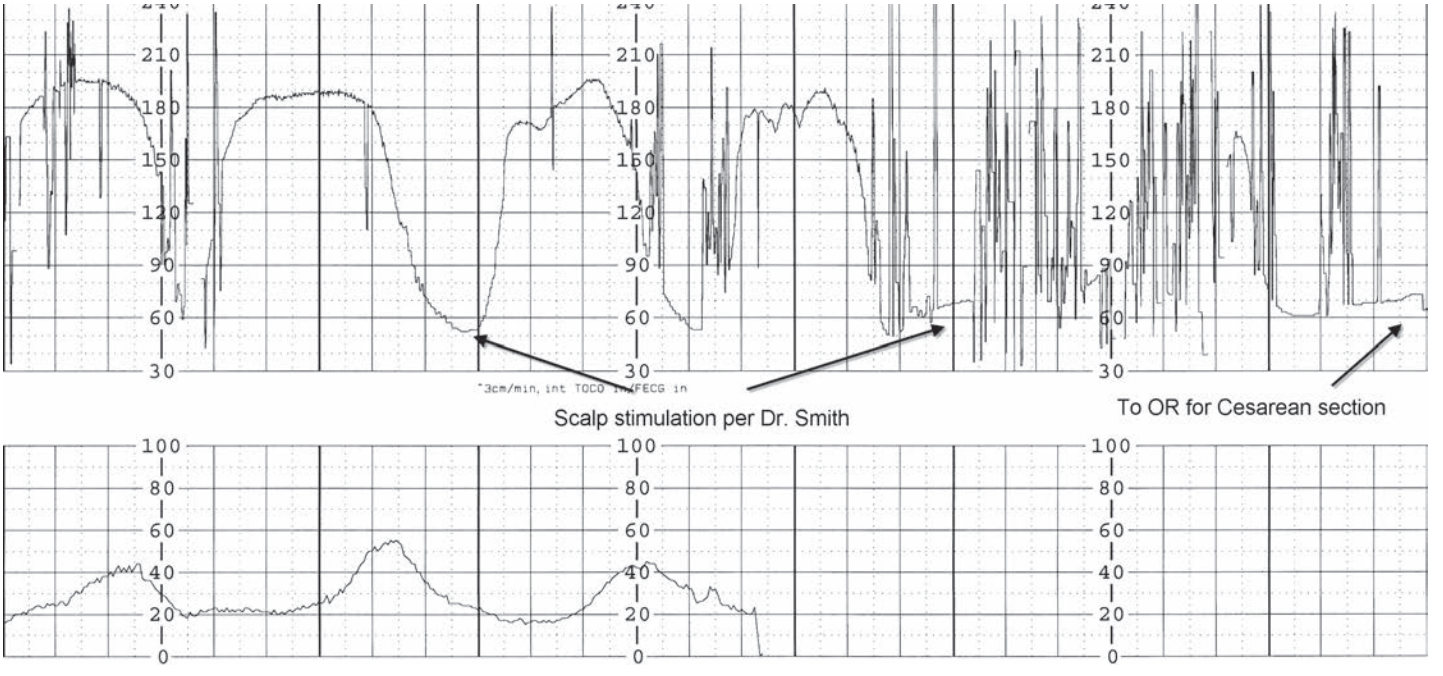


Figure 14.30. Following over 30 minutes of recurrent decelerations and scalp stimulation, patient is moved to the operating room for cesarean section.

stimulation, either via scalp stimulation or use of vibroacoustic stimulation. During periods of absent or minimal variability without FHR accelerations, either scalp stimulation or vibroacoustic stimulation may be used to rule out the concurrent presence of fetal metabolic acidemia. The stimulation should be done during periods of baseline FHR, not during episodes of deceleration or bradycardia. Clinicians must recognize that both scalp and vibroacoustic stimulation are tests, not treatments, and their role in the intrapartum setting is limited to *evaluation* of fetal acid-base, and they are not corrective measures for suspected hypoxemia or deceleration patterns (33). Further, while an acceleration of the FHR in response to fetal stimulation rules out fetal acidemia at that point in time, the absence of acceleration does not necessarily establish the presence of acidemia.

RISK MANAGEMENT AND PATIENT SAFETY

All of the aforementioned cases reflect areas of risk that require and deserve attention from a collaborative safety approach. The application of risk management can be divided into two major components: the avoidance of preventable adverse outcomes and the reduction of liability exposure following an adverse outcome (34). Both are valid goals but involve different approaches. Safety approaches to reduce the incidence of obstetric accidents include systems analysis, standardization, and improved inter- and intradisciplinary communication, while documentation strategies and advances in the area of disclosure and mediation can be implemented to limit liability risk following an adverse outcome.

Standardization

Standardization in perinatal care has been shown to improve outcomes and reduce the incidence of malpractice claims (5–8). Standardization of clinical practice in EFM has been difficult to achieve, in part due to a historical lack of national consensus between disciplines but also due to wide variance in fetal monitoring education and a lack of multidisciplinary education. These problems create fertile ground for system errors in fetal monitoring (35,36). However, adoption of standardized nomenclature by the American College of Obstetricians and Gynecologists (ACOG), the Association of Women's Health, Obstetric, and Neonatal Nurses (AWHONN), and the American College of Nurse-Midwives (ACNM) in recent years has resulted in availability of a common EFM language for clinical use (37). Initially developed by a panel of experts convened under the auspices of NICHD in 1997, standardized definitions were reaffirmed and expanded in 2008, when a second panel of NICHD experts was convened. The 2008 panel provided further clarification of the 1997 NICHD definitions as well as a three-tiered category system for FHR tracing evaluation and a clarification regarding summary terms for uterine activity (16). The NICHD terminology is discussed in Chapter 6.

EFM definitions that are standardized and consistent among and between disciplines are only the first step. Education of all obstetric team members, including nurses, midwives, residents, and all physicians, should include not only familiarization with the NICHD nomenclature but training in teamwork and communication as well as standardized approaches to interpretation and management of FHR tracings. Education should be multidisciplinary and must be ongoing and repetitive to result in retention (38).

Communication

In 2004, the Joint Commission on Accreditation of Healthcare Organizations (JCAHO) cited both communication issues and inadequate fetal monitoring as underlying causes of perinatal morbidity and mortality (38). As mentioned earlier, a more recent sentinel event alert by JCAHO highlighted the deleterious effect of disruptive behavior on patient safety (9). Both alerts highlight the need for enhanced and respectful communication processes between and among clinicians. Several training programs have been developed to enhance inter- and intradisciplinary communication, many include utilization of specific scripted communication templates, such as SBAR (Situation, Background, Assessment, Recommendation), used in patient handoff situations (39,40); and five-step assertiveness used in crew resource management (41).

Multidisciplinary Interpretation and Management

Following the adoption of the standardized NICHD nomenclature and implementation of improved methods of communication, standardization of interpretation and management of FHR tracings during labor becomes a rational next step in reducing risk and promoting safety. Although initially considered negatively as "cook-book" medicine, the use of protocols, guidelines, and checklists in medical and nursing care has become commonplace and allows for the development of shared mental models, a key factor for successful team performance (42). In 2010, ACOG published an algorithm for the management of intrapartum FHR tracings that was based upon the three categories identified in the 2008 NICHD workshop report (43). Similar to the ACOG algorithm, but providing more details related to interpretation concepts and specific management checklists, is the model published by Miller in 2011 (15,44). Miller identifies three principles of interpretation that can be applied to any tracing, by any member of the

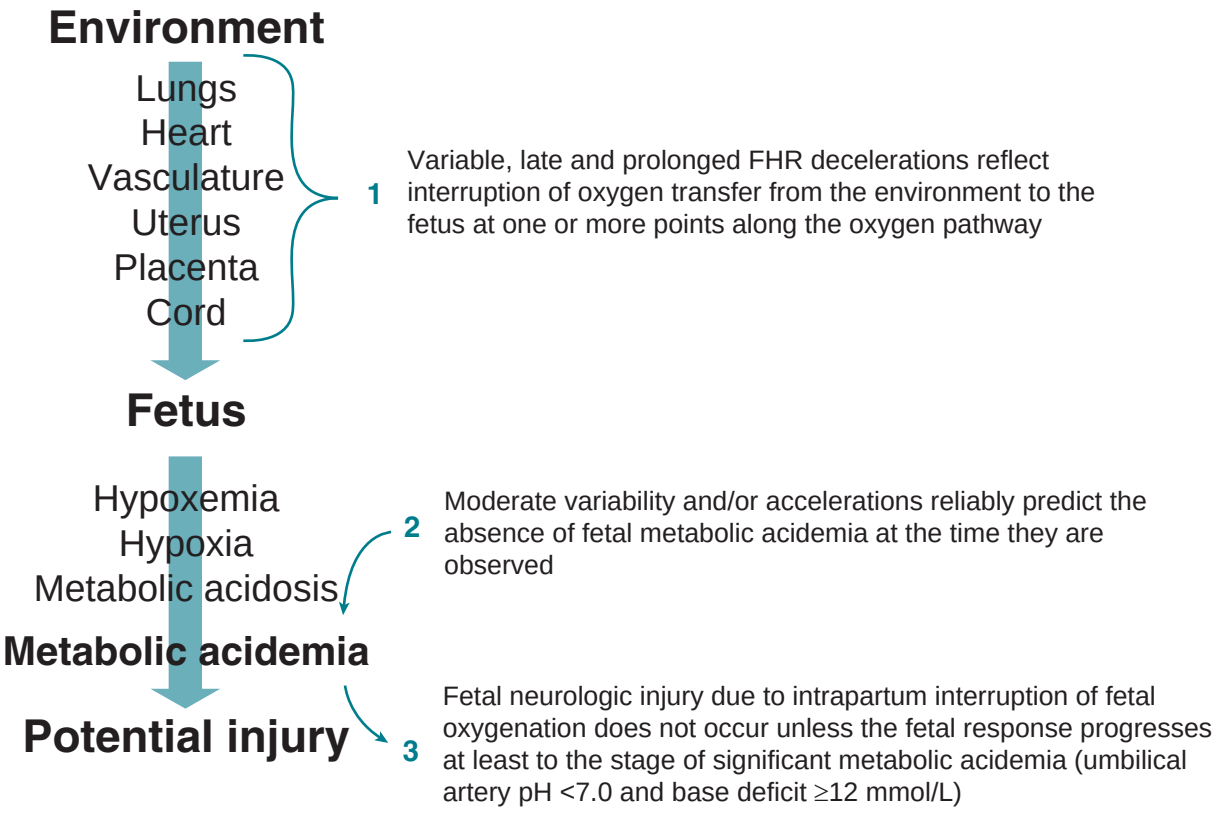


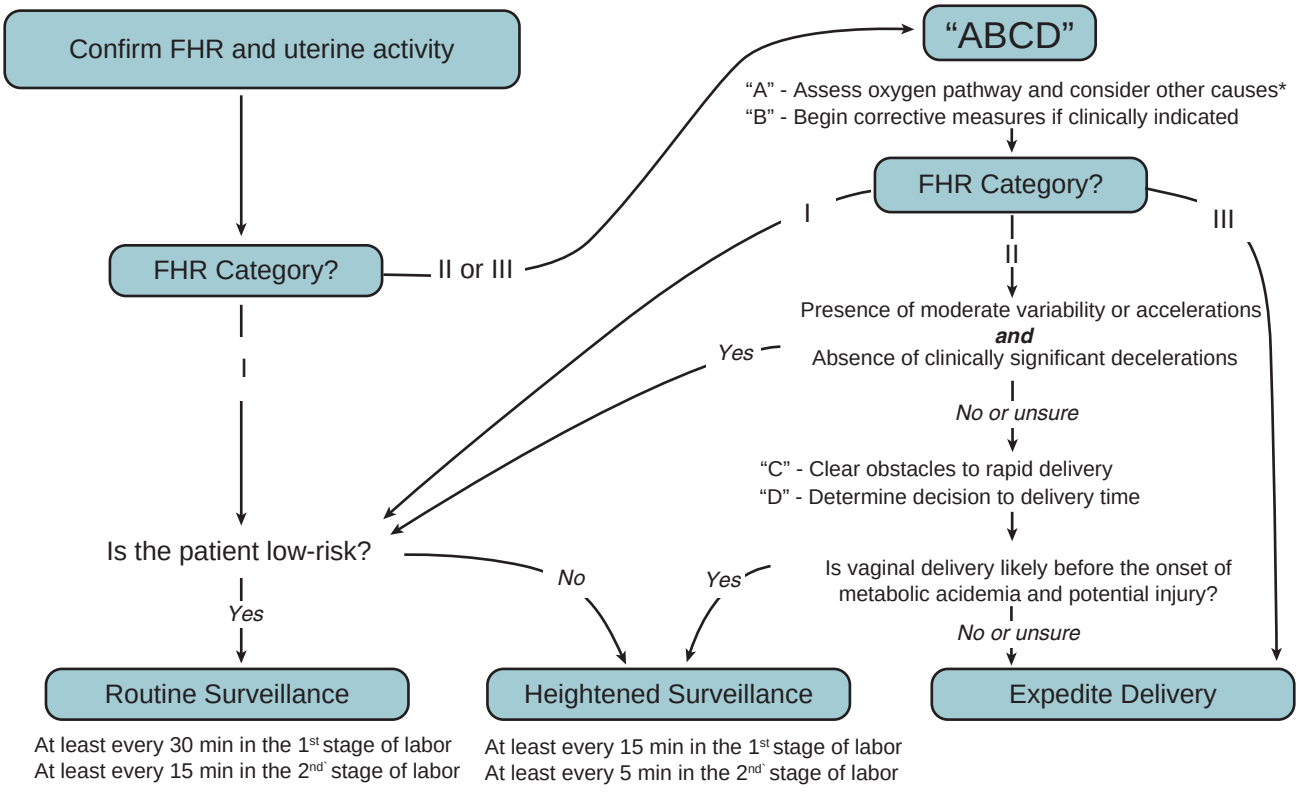
Figure 14.31. Three central concepts of intrapartum fetal heart rate interpretation. (Courtesy of David Miller, MD.)

health care team, to help the clinician evaluate fetal oxygenation and rule out the possibility of fetal metabolic acidemia. The three principles are illustrated in Figure 14.31.The associated management model is based on the application of standardized interpretation and utilizes an approach to management that includes assessing the pathway of oxygen transfer, beginning corrective intrauterine resuscitative measures when indicated, considering resources and clinical context related to vaginal versus operative delivery, and decision support for mode and timing of delivery (44) (Fig. 14.32). As obstetric teamwork improves and the concept of a multidisciplinary approach to FHR evaluation, interpretation, and management becomes the clinical norm, it is clear that standardization will play an ongoing role in FHR monitoring. The importance of uniformity in EFM training for nurses, physicians, and midwives cannot be overemphasized and has been shown to decrease liability and improve outcomes (6,7).

Documentation

The second component of risk management is mitigation of liability. While institutional changes that promote a focus on patient safety can reduce liability by improving outcomes, not all adverse outcomes in perinatal care are preventable, and liability issues can occur even in HROs. When questions arise in litigation regarding obstetric care, nursing and provider documentation in the medical record will be closely scrutinized by experts for both the plaintiff and the defendants. Documentation must accurately reflect communications between caregivers as well as events to avoid the "dueling defendants" scenario. If documentation is inadequate, caregivers may later recall communications and events differently and inadvertently create the appearance of conflict. Any appearance of conflict between caregivers, regardless of whether it is related to the standard of care, can create questions in the minds of jurors, making the defense of even the most appropriate care extremely difficult.

Although there are other methods of offering evidence of clinical care, such as sworn testimony regarding clinician recollections and routine or common clinical practices (45), case law demonstrates that documentation will be crucial to the outcome of litigation and may thwart frivolous liability claims (46). Inadequate or conflicting documentation by nursing and medical staff may be a problem. Specifically evaluating adherence to fetal monitoring guidelines, one study found that deviations from practice occurred more



	A Assess oxygen pathway	B Begin corrective measures if indicated[a]
Lungs	Airway Breathing	Supplemental oxygen
Heart	Heart rate and rhythm Cardiac output	
Vasculature	Blood pressure Volume status	IV fluid bolus Maternal position changes Correct hypotension
Uterus	Contraction strength Contraction frequency Baseline uterine tone Uterine relaxation time Exclude uterine rupture	Stop or reduce stimulant Consider uterine relaxant
Placenta	Placental separation Vasa previa	
Cord	Vaginal exam Exclude cord prolapse	Consider amnioinfusion

	C Clear obstacles to rapid delivery	D Determine decision to delivery time
Facility	OR availability Equipment	Response time
Staff	Consider notifying • Obstetrician • Surgical assistant • Anesthesiologist • Neonatologist • Pediatrician • Nursing staff`	Consider staff • Availability • Training • Experience
Mother	Informed consent IV access Anesthesia options Laboratory tests Blood products Urinary catheter	Surgical considerations (prior abdominal or uterine surgery) Medical considerations (obesity, hypertension, diabetes)
Fetus	Confirm • Estimated fetal weight • Gestational age • Presentation • Position	Consider • Estimated fetal weight • Gestational age • Presentation • Position
Labor	Confirm • Accurate monitoring • Adequate uterine activity	Consider • Arrest disorder • Protracted labor • Remote from delivery • Poor expulsive efforts

Examples of clinical factors to be considered in a systematic fashion. Institutions may modify according to individual circumstances.
[a]Conservative corrective measures should be guided by clinical circumstances. For example, amnionfusion may be appropriate in the setting of variable decelerations but would not be expected to result in resolution of late decelerations.

Figure 14.32. **A, B:** Standardized management model for intrapartum fetal heart rate monitoring. (Courtesy of David Miller, MD.)

frequently related to documentation rather than evaluation or management (47).

Nursing, midwifery, and physician documentation must provide support for the clinician being questioned at deposition or trial, and while styles and methods of charting may properly differ, the basic principles of documentation remain unchanged by specialty. Use of the acronym "CLEAR" has been suggested as a basis for charting, the letters representing five core concepts (45):

Contemporaneous
Logical
Explicit
Accurate
Readable

Entries in the nursing and medical record should be *contemporaneous* or they may be open to speculations regarding credibility. Appropriate late entries can be used following an emergency or if documentation of an intervention or observation was delayed, and should include both the time of recording as well as the actual time of the event. While there is no specific legal standard regarding the definition of contemporaneous, institutions would do well to identify reasonable time frames for documentation. For example, documentation that is within 30 to 60 minutes of an event may be considered reasonable, but a lengthy note written following an emergency should be appropriately labeled as a late entry. Some institutions have instituted the use of specific response teams to obstetric emergencies, where one of the team members functions as a recorder, allowing contemporaneous documentation of emergency interventions and team member actions (48).

Documentation should be *logical* and provide an accurate picture of the patient's condition, along with the team's plan of care or management. Use of the SOAP (Subjective, Objective, Assessment, Plan) note designed by Weed (49) continues to be favored by many providers, and creates an opportunity for clinicians to demonstrate a link between assessment and plan, which can assist in deposition or at trial by refreshing recollections of the obstetric team's approach. The SOAP note also assists in chart audits for quality assurance processes, by providing evidence of the thought processes of clinicians. It is also important that documentation be *explicit*, as any vague or ill-defined terminology will be subject to attack in litigation, and may have the unintended consequence of creating the appearance of lack of knowledge or conflict among team members. Litigation can result in the need for a clinician to answer questions regarding fetal monitor tracings many years after the fact, sometimes in situations where the actual FHR tracing is missing or lost. When documenting FHR findings, clinicians are better served by recording the actual components of baseline rate, baseline variability, accelerations, and decelerations rather than the simplified categories. While the FHR components can always be used to determine a category, the category alone will provide little to no help in recalling the individual components. The same is true for summary terms related to uterine activity; "normal" and "tachysystole" provide a picture of frequency of contractions alone, which do little to help when queried on specifics. The clinician is encouraged to document the components of uterine activity, which include frequency, duration, and strength.

Accuracy is also extremely important to avoid allegations of spoliation, the deliberate or negligent alteration, hiding, or falsification of records. Entries must reflect actual events and the times related to the events. Use of standardized nomenclature, short debriefings to confirm event times and team responses following obstetric emergencies, and inclusion of documentation review as part of the overall quality assurance program can improve accuracy. The last component of the CLEAR acronym requires that documentation be *readable*. Closely related to accuracy, notes must be readable to reviewers, whether the review relates to litigation or is part of a quality or safety initiative. The introduction of electronic medical records to labor and delivery suites has been shown to positively affect both readability and comprehensiveness of documentation without impinging upon direct patient care (50). There is also evidence showing increased satisfaction and improved communication with the adoption of electronic medical records and computerized entries by obstetric physicians (51). Regardless of the method of documentation, paper or electronic, the use of abbreviations can lead to confusion and affect readability. At trial, documentation must be interpretable and understandable by all readers, not merely the author (46). Obstetric teams should agree on a standardized set of abbreviations for clinical usage, which should be available to all clinicians and/or included in electronic documentation systems.

Documentation frequency can be a challenging issue for clinicians, and frequency of documentation related to EFM assessment rightly varies among different members of the health care team. Physicians looking to ACOG will find a recommendation for "periodic" documentation of FHR tracing review (17). Again, a reasonable approach (in addition to admission and delivery notes) would be to consider writing a note, using the SOAP or a similar format whenever the patient is examined for progress, following any significant changes in condition, and whenever the plan of management or support is altered. Patients whose condition requires multiple day stays for observation may require less frequent documentation than those in active labor or those with acute complications. Nursing documentation, often aided by the use of preprinted or electronic flow sheets, generally occurs on a more frequent basis due to the nature of nursing care. Frequency of nursing documentation is usually based on hospital policy rather than legal or regulatory requirements (52), and many hospital policies confuse guidelines on the frequency

of assessment with standards for documentation frequency (37,45). While it is entirely appropriate to have nurses at the bedside in the second stage of labor, and to expect them to assess the fetal monitor findings and uterine activity as often as every 5 minutes in at-risk patients (53), it is not rational to expect them to chart with such frequency. In fact, policies that place onerous documentation burdens upon nurses may create situations where nurses will feel obligated to "fill in the blanks" by back-charting, an invitation for allegations of spoliation (40). Regardless of the obstetric team member undertaking documentation, frequency should be based on individual patient acuity and reasonable expectations rather than arbitrary or misguided time frames.

Deposition Testimony

Given the fact that not all poor outcomes are preventable, it is likely that nurses, midwives, and physicians who practice for any significant length of time will be involved in litigation, either as a named defendant or as a witness. It is imperative that clinicians understand and be prepared for providing sworn testimony in such situations, whether this occurs as part of the discovery process in deposition, or in front of a jury at trial. Although an in-depth discussion of deposition or trial preparation is outside the scope of this text, EFM is the single most common obstetric procedure and therefore frequently becomes a central issue in brain-damaged newborn cases. It is important that clinicians understand the implications of their testimony and be appropriately prepared.

All too often, physicians and nurses have difficulty clearly articulating their assessments of FHR tracings and the obstetric team's plan of management. Physicians, nurses, and midwives must be able to demonstrate, through their answers to deposition questions, that they acted in a reasonable and prudent manner in providing care to the mother and fetus. When questions arise related to fetal monitoring, clinicians who are able to provide accurate definitions of FHR terminology, discuss the underlying physiology of deceleration patterns, and explain the plan of intervention and management will be well positioned to defend against allegations related to violations of the standard of care. Documentation, discussed earlier, becomes crucial to deposition performance, as it provides a record of events and will serve as an aid to memory. But even the best documentation will not serve a clinician who fails to be able to correctly answer basic questions on EFM terminology or FHR physiology. Multidisciplinary education, standardization, and repetition of fetal monitoring concepts in the clinical setting are the best tools to guarantee deposition performance accurately reflects the skill and knowledge of the obstetric team. Additionally, these tools preclude the plaintiff from creating the appearance of conflict by ensuring that team members (whether new nurse or experienced physician) are consistent with their use of terminology and approach to interpretation and management. Deposition testimony will set the stage for trial testimony and will be instrumental in decisions regarding settlement. The majority of cases involving allegations of birth-related injuries will settle prior to or during trial if not dismissed during the discovery period. There are many reasons for this, including the costs of trial weighed against perceived defensibility, strength of expert and defendant testimony, and insurer's tolerance for risks associated with jury trials. Defendant clinicians should recognize that settlement is not a reflection of fault or negligence but rather a decision made based on many factors outside of the clinician's control including fears related to disproportionally large jury verdicts in many obstetric cases when compared to malpractice verdicts in other areas of medicine. Recognition of this disparity, along with dissatisfaction with the current tort system, has led to proposals for alternate methods of dispute resolution.

ALTERNATIVES TO THE CURRENT TORT SYSTEM

In the United States, only Florida and Virginia currently have alternatives to the tort system for handling claims related to neurologically impaired infants. These programs provide compensation to families with infants who have specifically defined types of birth-related injuries and do not require evidence of negligence as is required by traditional malpractice actions under tort law. Both the Florida and Virginia programs include case review by a medical panel, and while both have significant limitations they have been shown to decrease malpractice claims (54). These programs function similarly to worker's compensation plans, and do not provide dispute resolution or evaluation of the health care provided relative to the injury. Recently, there have been efforts to pilot administrative health courts for dispute resolution and compensation as an alternative to traditional malpractice adjudication (55–57). Composed of specially trained judges, able to identify junk science, and providing access to all patients, regardless of the perceived monetary value of the claim, the difference administrative health courts offer over no fault compensation programs is twofold: They provide dispute resolution while offering opportunities for practice improvement through dissemination of the findings of unbiased expert panels. A sample schematic for an administrative health court process is illustrated in Figure 14.33. In addition to appropriate compensation, the health court findings would include remedial action for clinicians when warranted by the review of neutral experts.

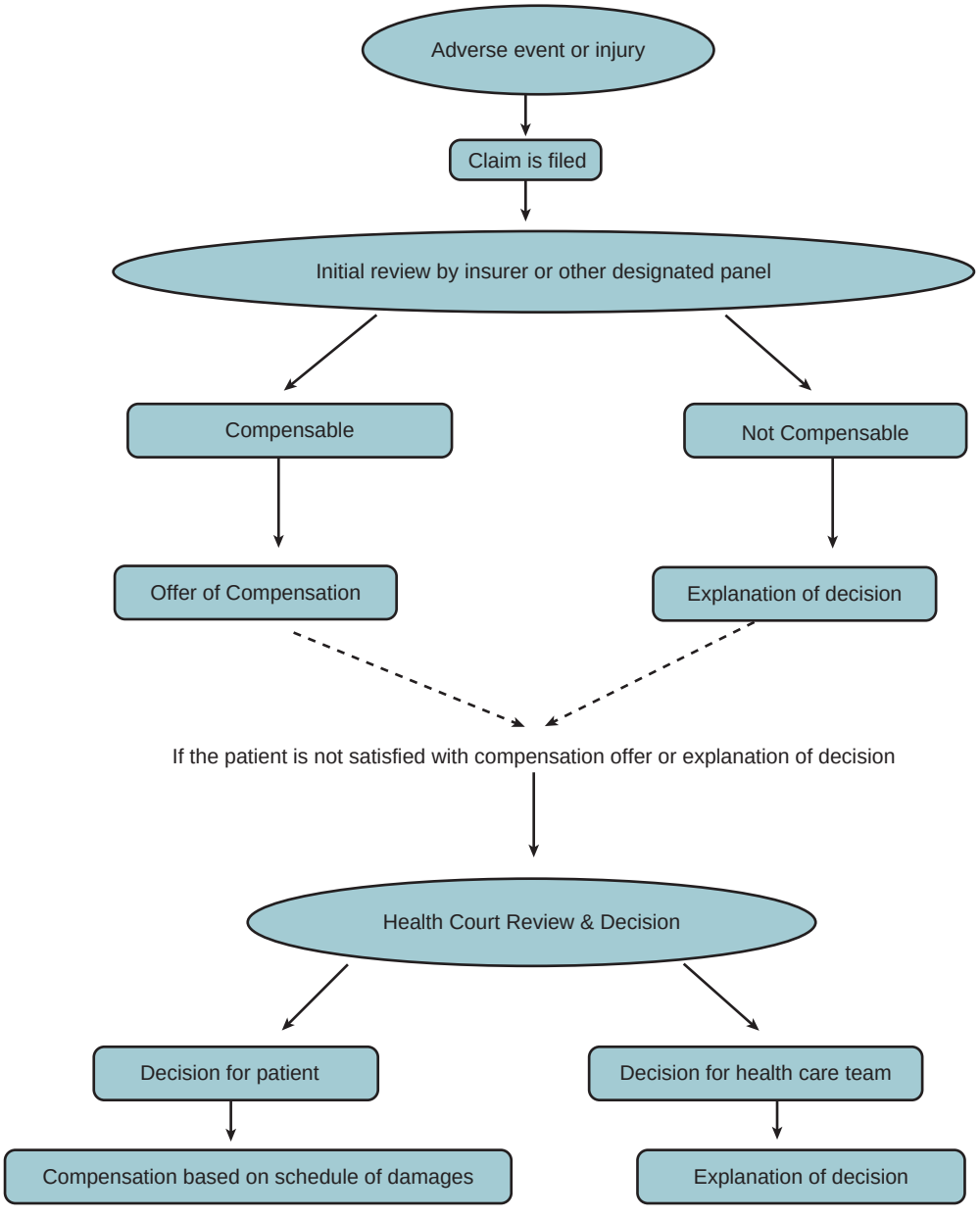


Figure 14.33. Model for alternative health court. (From Miller LA: Health courts: an alternative to traditional tort law. *J Perinat Neonat Nurs* 25(2),99–102, 2011, with permission.)

SUMMARY

In a detailed overview of quality improvement in obstetrics and gynecology, Gambone and Reiter (58) point out that "[g]ood people working within a flawed system will produce less than optimal outcomes". Patient safety requires ongoing evaluation and process adaptation as new evidence, consensus, and innovations in clinical practice arise. In relationship to EFM, several measures can be undertaken to promote patient safety. Multidisciplinary education in EFM, standardization of nomenclature, interpretation and management, use of obstetric hospitalists, and teamwork and communication training are all avenues to potentially improve practice. Knowledge of liability and risk areas related to EFM allows clinicians to create systems that minimize the frequency of unintended outcomes, while understanding the role of documentation and interdisciplinary communication aids in both patient care and in the defense of alleged negligence.

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