Blazer S, Zimmer EZ (eds):
The Embryo: Scientific Discovery and Medical Ethics.
Basel, Karger, 2005, pp 291–308

Fetal Surveillance Tests

Ido Solt[a] Michael Y. Divon[b,c]

[a]Department of Obstetrics and Gynecology, Rambam Medical Center, Haifa, Israel; [b]The Albert Einstein College of Medicine, Bronx, and [c]Department of Obstetrics and Gynecology, Lenox-Hill Hospital, New York, N.Y., USA

Not long ago, the fetus was considered a passenger, not a separate patient. It was believed that nothing could be done to improve fetal health inside the uterine 'black-box'. Perhaps the first written record we have of a mother and baby surviving a cesarean section comes from Switzerland in 1500 when a sow gelder, Jacob Nufer, performed the operation on his wife. After several days in labor and help from 13 midwives, the woman was unable to deliver her baby. Her desperate husband eventually gained permission from the local authorities to attempt a cesarean. The mother lived and subsequently gave birth normally to 5 children, including twins [1]. Peter Chamberlen presumably invented the obstetric forceps and kept it as a family secret [2]. These interventions were intended to save the parturient life during an obstructed labor, but they also indirectly improved the survival chances of the fetus. The concept of 'the fetus as a patient' has evolved in the twentieth century and now it is fundamental and firmly established in obstetric care.

'Fetal monitoring' in a wide sense means fetal surveillance but, practically, it is an indirect way to measure fetal well-being or the adequacy of fetal oxygenation and, as such, it is an integral part of the concept of 'the fetus as a patient'. The primary goal of fetal monitoring is a healthy newborn with a healthy mother.

Table 1.

Methods of fetal monitoring

Fetal movement assessment
Periodic fetal heart rate (FHR) auscultation (fetoscope)
Continuous electronic FHR monitoring (NST, CST)
Fetal biophysical profile scoring (BPP)
Stimulation techniques: scalp stimulation and vibroacoustic stimulation
Fetal amniotic fluid analysis
Fetal blood evaluation
Fetal pulse oximetry
Doppler velocimetry

There are several methods of antepartum and intrapartum fetal monitoring (table 1). This chapter will focus mainly on the 'continuous electronic fetal heart rate monitoring' (CEFHRM), biophysical profile and Doppler velocimetry which are currently the main clinical assessment techniques.

History of Fetal Heart Rate Monitoring

Direct fetal heart rate (FHR) auscultation with the examining ear placed on the maternal abdomen has been known for a centuries [3], Marsac heard fetal heart beats in the seventeenth century. Mayor [4], a Swiss surgeon, first reported direct FHR auscultation in 1818. Jean Alexandre Le Jumeau and Viscount de Kergaradec first described auscultation of the fetal heart with a Laennec stethoscope or with a wooden fetoscope in 1821 [5]. Le Jumeau, who was not an obstetrician, reported hearing fetal heart beats in 8 pregnant women. Le Jumeau suggested that fetal auscultation could be used to detect pregnancy as well as to identify twin gestation, fetal lie and even fetal health [6]. Le Jumeau was the pupil of Laennec, the founder of medical auscultation. Kennedy, in his monograph from 1833 [7] postulated that 'since newborn hearts beats longer compared to adult hearts when excised, they can better tolerate a hypoxic environment'. Kennedy described the effects of head and cord compression on the fetal heart rate and related a slow return of FHR after a bradycardia to 'fetal sufferance'. This monograph was ahead of its time by including an appendix containing legal notes! Kilian, in 1849, published on 'stethoscopical indications for forceps operation'. He suggested that forceps

delivery should be applied without delay when the FHR is less than 100 bpm or more than 180 bpm, when the fetal heart sounds lose their purity of tone or when only one tone could be clearly heard [8]. Kehrer [9], in 1867, described for the first time the association between fetal bradycardia and fetal head compression. Schwartz [10], in 1870, proposed counting the FHR as often as possible between and during the uterine contractions because FHR might change due to fetal 'asphyxic intoxication'. Schatz [11], in 1885, described the mechanism of FHR deceleration due to umbilical cord compression. Ernesto Pestalozza of Pavia described phonocardiography in 1891. In 1893, Winckel [12] suggested criteria for defining fetal distress, among them, FHR under 120 bpm or above 160 bpm. In 1903, Seitz [13] suggested three types of FHR decelerations, two related to enhanced vagal activity and one related to paralysis of all extracardiac nervous activity. He proposed that the changes in the FHR pattern reflect fetal oxygenation. In the same year, in the first edition of Williams Obstetrics [14], J. Whitridge Williams stated: 'The rate of fetal heart is subject to considerable variations which afford us a fairly reliable means of judging as to the wellbeing of the child. As a general rule, its life should be considered in danger when the heartbeats fall below 100 or exceed 160'.

Cremer recorded the first fetal electrocardiogram (ECG) in 1906, only 5 years after the first adult ECG registration by Einthoven in 1901. In 1922, Sach tried to use abdomino-vaginal and abdomino-rectal leads. In 1938, Bell had recorded fetal P waves. In 1958, Hon [15], 'the father of the modern FHR monitor', developed a method for continuous FHR recording. He described three patterns of FHR decelerations: early, variable and late which were related to head compression, cord compression and uteroplacental insufficiency, respectively. In 1963, Hon improved the quality of FHR recordings by introducing a fetal scalp electrode. In 1966, Hammacher [16] indicated that neonates who had FHR late decelerations prior to delivery, had lower Apgar scores after delivery and higher stillbirth rate. Six years later, Ray et al. [17] confirmed the observation of Hammacher in the first prospective blinded trial in the USA. In 1969, Hammacher linked FHR accelerations to fetal wellbeing. Continuous electronic FHR monitoring (CEFHRM) was commercially introduced in 1968; initially it was used for complicated and 'high-risk' pregnancies, but gradually it penetrated into the 'low-risk' pregnancies as well. By 1978, CEFHRM was used in nearly two thirds of deliveries in the USA [18]. By 1998, 84% of laboring women were monitored by CEFHRM [19] and today it is the most prevalent obstetrical monitoring technique, almost universally practiced in the Western world [3, 20].

FHR as an Indirect Measure of Fetal Brain Activity

Continuous recording of the fetal oxygenation would be the ideal method to assess fetal well-being. However, no such technique is presently available. Fetal heart rate monitoring was therefore suggested as an indirect measure of fetal brain oxygenation. Many physiological factors have an impact on the pacemakers of the fetal heart. Most important is the autonomic nervous system (sympathetic and parasympathetic nervous systems). Stimulation of the sympathetic nervous system leads to the release of catecholamines. These hormones increase the fetal cardiac output by increasing the FHR and myocardial contractility. Stimulation of this system occurs in cases of stress while during rest periods the heart rate is controlled by a tonic sympathetic stimulus.

The parasympathetic nervous system is dominated by the vagus nerve, which is the tenth cranial nerve and originates from the medulla oblongata. The vagal fibers supply the heart pacemakers and the sinoatrial and atrioventricular nodes. Vagal stimulation leads to the release of acetylcholine, which decreases the heart rate. There are two main vagal influences on the FHR. The first is a constant tonic stimulus that decreases the normal intrinsic FHR and, the second, an oscillatory stimulus that is responsible for most of the variability in the FHR. The vagal influence increases with gestational age. At 20 weeks' gestation, the average FHR is 155 bpm, at 30 weeks it is 144 bpm and at term it is 140 bpm. Variations of up to 20 bpm above or below the average FHR are regarded as normal. In well-oxygenated fetuses, FHR changes reflect different behavioral states. During active sleep and wakefulness, there are fluctuations in the baseline rate with a clearly visible beat-to-beat variability and heart rate accelerations during fetal movements. During quiet sleep there are only a few or no heart rate accelerations, and the beat-to-beat variability is reduced to some extent.

Hypoxic periods are characterized by an increased activity of both the sympathetic and parasympathetic nervous systems. Initially, the chemoreceptors are activated, leading to α-sympathetic stimulation that constricts the vascular beds of the gastrointestinal tract, liver, spleen, lungs, carcass, kidneys and skin (which are nonessential organs for immediate survival of the fetus) and thus cause hypertension and preferential increase in blood flow to the vital organs, i.e. the brain, heart, adrenals and placenta. At this stage of hypertension, the baroreceptors are activated with afferent link to the brain stem, leading to vagal stimulation and slowing of the heart rate. The decrease

in heart rate presents as fetal heart rate deceleration. In cases of severe hypoxia, the fetus becomes acidotic, the brain stem is less responsive, the autonomic reflexes are blunted and there is a direct myocardial depression, which worsens the heart rate deceleration and lead to severe bradycardia.

The various FHR patterns observed in well oxygenated or hypoxic fetuses serve as the basis for the FHR testing.

The Nonstress Test

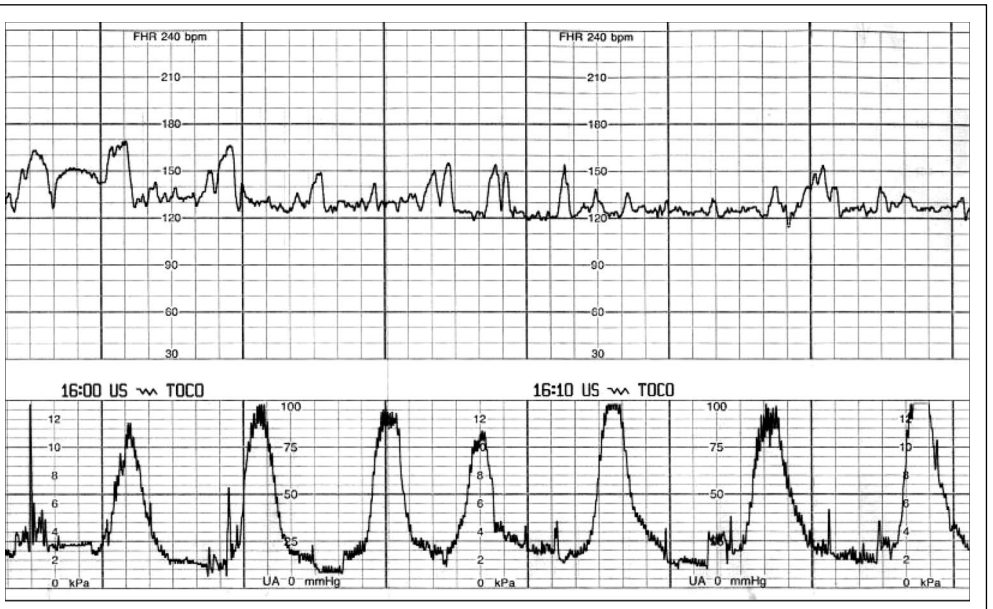
The nonstress test (NST) is a primary fetal surveillance tool. It is noninvasive, inexpensive, and simple and has no contraindications. Continuous wave Doppler transducers applied to the maternal abdomen record the FHR. The American College of Obstetricians and Gynecologists has defined a normal, i.e. reactive, NST as a tracing with two or more FHR accelerations that peak at 15 bpm or more above baseline, each lasting 15 s or more, and all occurring within 20 min of testing (fig. 1). The absence of two FHR accelerations within 20 min qualifies the tracing as nonreactive; however, in order to conclude that fetal reactivity is absent, at least 40 min of recording are required since most non-reactive tracing are due to fetal sleep. The perinatal mortality within a week of a reactive NST is 2–5/1,000. While a reactive NST is highly predictive of fetal well-being, the opposite is not true as most fetuses with a nonreactive NST are not compromised. Overall, the NST has a specificity of more than 90% and a sensitivity of 50%. The negative predictive value is 90% and the positive predictive value is less than 50%.

Most obstetricians perform a visual interpretation of the FHR tracing. However, the accuracy of this method is questionable and large inter- and intra-observer variations have been noted. The introduction of computerized analysis systems has, therefore, been suggested, but is not widely practiced.

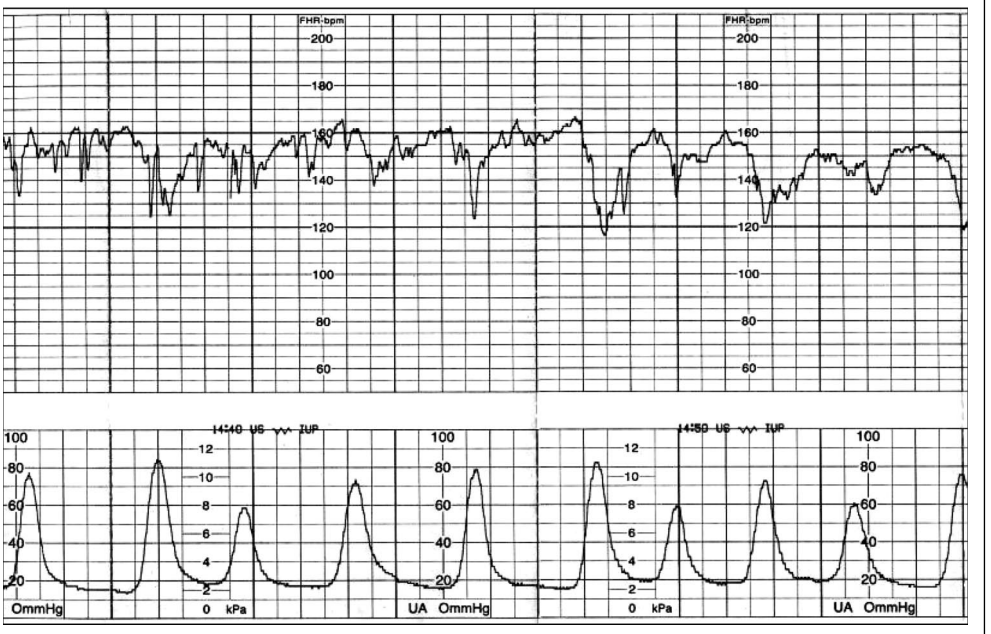
The high false-positive rate of the NST may lead to unnecessary interventions. Most investigators therefore believe that additional testing is needed to confirm the results of the NST.

The Contraction Stress Test

During the late 1960s, as the experience with intrapartum electronic fetal monitoring became more widespread, investigators focused on the contraction


FHR 240 bpm
210
180
150
120
90
60
30
16:00 US TOCO
16:10 US TOCO
12
10
8
6
4
2
0 kPa
100
75
50
25
UA 0 mmHg

a


FHR-bpm
200
180
160
140
120
100
80
60
14:40 US TUP
14:50 US TUP
100
80
60
40
20
OmmHg
12
10
8
6
4
2
0 kPa
UA OmmHg

b

stress test (CST) as an antepartum test of fetal well-being. The hypothesis underlying the CST evolved from animal physiologic observations suggesting that blood flow to the intervillous space is decreased during uterine contractions. The rationale of the CST is to induce uterine contractions in order to uncover cases of uteroplacental insufficiency. Uterine contractions are induced by intravenous administration of oxytocin and the FHR reactivity is assessed. In order to get sufficient data, three 40–60 s contractions should be recorded within 10 min. The CST may induce labor and is therefore contraindicated in cases of a previous classical cesarean section, placenta previa, placental separation and premature rupture of membranes. The test should also be performed cautiously in cases of multiple pregnancy, hydramnios and incompetent cervix.

Fetuses whose oxygenation is suboptimal due to 'placental insufficiency' are likely to display late fetal heart rate decelerations when faced with a further decrease in their oxygenation during uterine contractions. The CST is considered positive when late decelerations occur following 50% or more of uterine contractions. A detailed description of the CST is beyond the scope of this editorial. In general, most authors have demonstrated a favorable perinatal outcome following a negative CST. In 1978, Evertson et al. [21] summarized 14 observational clinical reports that revealed seven fetal deaths among 1,739 high-risk patients within 1 week of a negative CST, for a noncorrected incidence of antenatal fetal death of 4/1,000. In most of these cases fetal demise resulted from 'non-predictable' etiologies such as umbilical cord accidents, severe congenital anomalies or placental abruption. A prospective multi-institutional study by Freeman et al. [22] included 4,626 patients who underwent weekly CST. The study demonstrated a corrected false negative rate of 0.4/1,000.

Later, Freeman et al. [23] combined results from 16 studies and reported a corrected false negative of 1.2/1,000. These figures suggest that the false-negative rate of CST is much lower than the stillbirth rate of 8/1,000 commonly reported in the general obstetrical population. CSTs are commonly performed in high-risk populations, which are by definition expected to have a higher fetal death rate than the general population.

Fig. 1.

Fetal heart rate (FHR) tracings. **a** Normal FHR tracing: The upper panel shows FHR variability and accelerations. The lower panel shows uterine activity. **b** An abnormal FHR tracing: The upper panel shows FHR decelerations. The lower panel shows uterine activity.

Therefore, it is commonly concluded that the use of the CST results in a dramatic decrease in antepartum fetal deaths. However, there are no prospective randomized trials (i.e. tested vs. nontested populations) documenting the presumed benefits of CST in either low-risk or high-risk patient populations.

The false-positive rate for the CST averages about 30%, with a range of 8–57%. This implies that approximately 30% of interventions due to a positive CST were deemed unnecessary following the intervention. In some studies true positives were defined by predicting questionable outcome variables such as thick meconium, 'need for cesarean section for fetal distress' or the presence of fetal growth restriction. Thus, the true false-positive rate of the CST is probably unknown.

As already stated, randomized studies of the CST versus no fetal testing are yet to be performed. However, in a non-randomized study of 1,542 high-risk patients undergoing weekly non stress testing (NST) and 4,626 patients undergoing CST, Freeman et al. [22] have shown that the rate of intervention for an abnormal test is higher for the CST group (4.5 vs. 2.9%, respectively) whereas the NST group had significantly higher incidence of respiratory distress syndrome, intrauterine growth restriction, birth weight <2,500 g and 5 min Apgar score of <7. The false-negative rates for the CST and NST in the prediction of fetal demise were 0.4/1,000 and 3.2/1,000, respectively.

It is generally believed that the CST is superior to the NST in the prevention of fetal death.

Biophysical Profile

In the early 1980s Manning and co-workers [24] introduced the concept of the fetal biophysical profile (BPP). This profile is somewhat similar to the commonly used neonatal Apgar score and consists of combined use of the NST and four biophysical activities observed with ultrasound: (1) fetal heart rate accelerations (NST); (2) fetal breathing movements; (3) fetal body movements; (4) fetal tone, and (5) amniotic fluid volume. Two points are assigned for each normal variable while no points are assigned for each abnormal variable. Thus, the BPP score ranges from zero to ten. The BPP reflect the integrity of the fetal central nervous system. In the original prospective blinded study, Manning and co-workers evaluated 216 high-risk pregnancies and observed no perinatal deaths when all five variables were normal.

Perinatal mortality was 60% when all variables were abnormal. Intermediate score values yielded intermediate perinatal loss rates. Recently, an observational study of 89,184 pregnancies that underwent BPP testing was reported by Dayal et al. [25]. The final BPP results were normal in 86,955 (97.5%) pregnancies (including 84,771 fetuses observed in Manitoba's fetal assessment program and 2,184 fetuses from Columbia Presbyterian's program). The false-negative death rate varied between the two institutions: in the Manitoba population there were 60 fetal deaths among 84,771 fetuses within 1 week of a normal BPP, yielding a false-negative rate of 0.71 per 1,000. In contrast, in the Columbia-Presbyterian study, 5 of 2,184 fetuses died within a week of a normal BPP, yielding a false-negative rate of 2.29 per 1,000. However, when these ratios were compared with the gross perinatal mortality in each center, the ratio of the false-negative rate to the total perinatal mortality rate showed no statistical significance (0.093 and 0.104 for Manitoba and Columbia Presbyterian, respectively) [25].

In 1993, Manning [26] conducted a prospective observational study in order to determine the relationship between the fetal BPP and umbilical venous pH obtained by cordocentesis. Cordocentesis was performed immediately after the BPP. A total of 493-paired observations were made. A biophysical score of zero was associated with significant fetal acidemia (pH <7.20), whereas a normal score (i.e. 8–10) was associated with a normal pH. An equivocal test – a score of 6 – was a poor predictor of an abnormal pH. Other investigators also examined this correlation and concluded that the association of the BPP and umbilical vein pH is strongest at the extremes of the BPP score [27–29].

In a subsequent study, Manning et al. [30] evaluated the association between final BPP score before delivery and the incidence of cerebral palsy (CP). A highly significant inverse exponential relationship was observed in a retrospective study of 26,288 high-risk pregnancies. The authors suggested that antenatal asphyxia is an important and potentially avoidable cause of CP.

In a prospective blinded study, 735 high-risk patients were randomly assigned to either a fetal BPP (375 patients) or a NST (360 patients). Although the sensitivity, specificity, and accuracy were higher with BPP, these differences did not reach statistical significance [31].

In a randomized prospective trial, Platt and co-workers [32] studied a total of 652 patients (1,628 tests were preformed). 279 pregnancies were managed by BPP and 373 were managed by NST. The results of this study suggest that the biophysical profile is more predictive of abnormal outcome

than the NST. However, because the groups were too small statistical significance could not be established and the investigators recommended that additional studies be performed.

A modified fetal BPP based on two of the five original variables – the NST and amniotic fluid volume – has been reported [33, 34].

In a prospective observational study of 15,482 high-risk pregnancies, a false-negative rate of 0.8/1,000 and a false-positive rate of 60% were reported [35].

The American College of Obstetrics and Gynecologists has concluded that the modified BPP is an acceptable modality for antepartum fetal surveillance [36].

Alfirevic et al. [37] randomized 145 women with singleton, uncomplicated pregnancies at or beyond 42 weeks of gestation to either BPP comprising of computerized NST, amniotic fluid index and assessment of fetal breathing, tone and gross body movements (n = 72) or the standard NST and a measurement of the amniotic fluid volume as defined by the depth of the largest pocket of amniotic fluid (n = 73). There were significantly more abnormal results in the BPP group. There were no differences in cord blood gases, neonatal outcome or in outcomes related to labor and delivery between the two groups, but a trend towards more obstetric interventions in the BPP group was noted. Another randomized controlled trial compared CST to modified BPP in 5,444 pregnancies. In this population, the frequency of adverse perinatal outcome following a negative modified BPP was no greater than that following a negative CST [38].

Alfirevic and Neilson [39] reviewed the literature concerning BPP and modified BPP in order to determine whether this is an effective and safe test for the assessment of fetal well-being in high-risk pregnancies.

Four randomized controlled studies were included in their analysis. They concluded that biophysical profile testing showed no obvious effect (either beneficial or deleterious) on pregnancy outcome when compared to other fetal testing modalities. However, they have noticed that although many reports of observational studies have been published, less than 3,000 pregnancies have been enrolled into randomized trials. Thus, they concluded that there is insufficient evidence from randomized trials to evaluate the use of BPP as a test of fetal well-being in high-risk pregnancies.

The logic for the clinical use of the BPP is similar to the logic applied for justifying the use of the NST or the CST, i.e. that the use of BPP in high-risk populations is associated with a low antepartum fetal death rate when

compared to either the nontested low-risk population or to historical controls. No randomized controlled trials of tested versus nontested are available to substantiate this conclusion.

Doppler Evaluation of the Fetus

Doppler ultrasound is a noninvasive technique used to evaluate blood flow in maternal and fetal vessels. The shift in frequency of the sound wave caused by moving red blood cells enables determination of the blood flow velocity by waveform analysis.

The umbilical artery is the easiest organ to visualize for Doppler studies. The most simple and most common index to be evaluated is the systolic-diastolic ratio (S/D ratio). Normally, there is a forward flow throughout diastole and an S/D of less than 3 is reassuring in the third trimester. In cases of uterine and placental pathology, there is an increase in vascular resistance and decrease in diastolic flow, resulting in an elevated S/D ratio. In severe cases, absence of the diastolic flow or even a reversed diastolic flow may be noted (fig. 2). Randomized controlled trials have shown that abnormal umbilical artery velocity waveforms are valuable in identifying the growth-restricted fetus [40]. A meta-analysis of studies on pregnancies complicated by suspected fetal growth restriction revealed that the use of Doppler evaluation was associated with a significant improvement in prenatal outcome and a reduction of 38% in perinatal mortality [40].

The use of Doppler velocimetry in low risk pregnancies is much less promising. The current data reveal that routine Doppler ultrasound in low-risk or unselected populations does not confer benefit on mother or newborn. The American College of Obstetricians and Gynecologists therefore recommends using Doppler studies in adjunct to other fetal evaluation modalities but not as a screening test for fetal well-being [41].

Pitfalls and Dilemmas in the Interpretation of Continuous Electronic FHR Monitoring

The introduction of continuous electronic FHR monitoring (CEFARM) created a utopian belief that pregnancy and delivery are finally safe and that a perfect outcome could be guaranteed in all cases. There was an

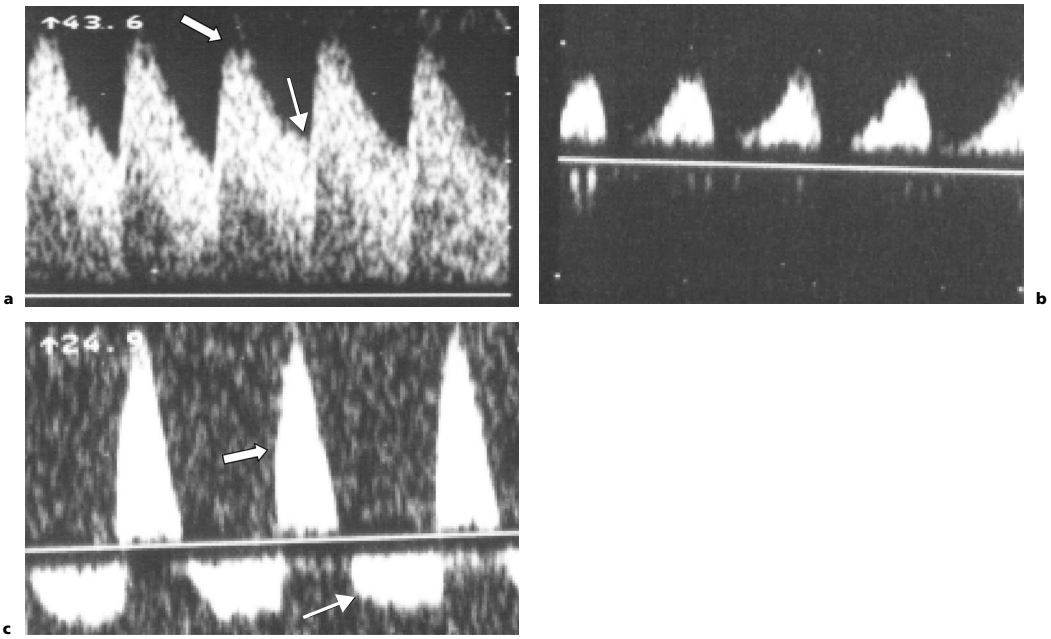


Fig. 2.

Doppler velocimetry in the fetus. **a** Normal flow, the systolic flow is marked by thick arrow. The diastolic flow is marked by a thin arrow. **b** Absent diastolic flow, only the systolic flow is observed. There is no diastolic flow. **c** Reverse diastolic flow, the systolic flow is marked by thick arrow. There is reverse diastolic flow marked by a thin arrow.

expectation that all perinatal damage or death will be prevented. In 1994, Symonds [42] found that about 70% of lawsuits related to neonatal brain damage and neurological disabilities were based on a nonreassuring CEFHRM. An assumption that was never proved is that fetal compromise develops in a gradual, progressive fashion which provides an 'intervention opportunity window' to improve the perinatal outcome. However, it is obvious that not all fetuses 'expire slowly' and that sudden deterioration such as in cases of placental abruption may occur.

Cesarean Section Rate and Neonatal Outcome. In almost all of the randomized controlled trials, CEFHRM was associated with an increase in the cesarean section rate without a significant improvement in neonatal outcome. Metabolic acidosis complicates up to 2% of all deliveries.

However, the rate of cesarean sections due to no reassuring tracing is well over 10%. There is, therefore, an urgent need for improving our ability to detect the hypoxic fetus. Techniques such as fetal ECG and pulse oximetry have been suggested. However, the clinical utility is still unproven.

CEFHRM and Cerebral Palsy. There has been no appreciable decline in the incidence of cerebral palsy (2/1,000) during the last 30 years since CEFHRM was introduced into obstetrical practice. However, there are other factors which influenced the rate of cerebral palsy during this period. The constant gradual improvement in the neonatal intensive care units, the introduction of the surfactant treatment, high-frequency ventilation and other techniques all dramatically improved the survival rates of the preterm and very low-birth-weight infants, which accounts for most of the cerebral palsy cases. The wide use of assisted reproductive technology has resulted in an increased incidence of multiple pregnancy and subsequent prematurity. The increased survival rate of asphyxiated term newborns has also added to the number of cases of cerebral palsy. Thus, the protective effect of CEFHRM might have been obscured by these improvements in survival rates.

Much effort has been devoted to identifying FHR patterns associated with neonatal brain damage. Todd et al. [43] examined the outcome of 2 year-old infants who were delivered electively before 34 weeks with an abnormal NST or Doppler study. Poor cognitive progress was more commonly noted with an abnormal NST compared to an abnormal Doppler. It was suggested that by the time a nonreassuring fetal status is diagnosed by antepartum testing, fetal brain damage has already become irreversible.

Other studies have shown that 75–92% of cerebral palsy cases are not related to perinatal asphyxia and occur during pregnancy or early in labor before CEFHRM has been applied [44, 45]. It seems, therefore, that these cases are not preventable by early obstetric intervention. In 1996, a committee opinion of the American College of Obstetricians and Gynecologists [46] addressed the relationship between perinatal asphyxia and the newborn neurological disability. It was concluded that the following four criteria must be present before a link can be made between perinatal asphyxia and cerebral palsy:

1 Umbilical artery acidemia with pH less than 7.0.
2 Apgar score 0–3 for longer than 5 min.

3 Neonatal neurological sequelae (hypotonia, seizures, coma).
4 Multiorgan system dysfunction.

Does FHR Monitoring Improve Perinatal Outcome?

The fundamental question in fetal monitoring remains whether any of these modalities improves perinatal outcomes.

Thacker and Berkelman [47] reviewed 600 articles to assess the accuracy and efficacy of the NST, CST and recording of fetal movements. Both the CST and the NST generally demonstrated low sensitivity and high rates of false-positivity. Only four randomized controlled trials have addressed these questions and none was of sufficient size to demonstrate whether there is a significant difference in outcome following use of the CST or the NST. A year later, Platt et al. [48] reviewed the impact of fetal testing of more than 200,000 pregnancies; almost 17,000 of these underwent various types of fetal surveillance tests during a 15-year period between 1971 and 1985. They concluded that fetal testing is beneficial since fetal death rate in selected high-risk patients was significantly lower when antepartum testing was utilized.

Mohide et al. [49] used the Oxford database of perinatal trials. The studies that were appropriately designed failed to show that the use of antepartum fetal testing is associated with improved perinatal outcome. Enkin et al. [50], in their 'guide to effective care in pregnancy and childbirth', had declared that antepartum tests are 'forms of care likely to be ineffective or harmful'.

Conclusion

Initially, there were great expectations from CEFHRM. The primary goal of fetal monitoring was, in a broad sense, to improve the short- and long-term perinatal outcome. It was hoped that fetal monitoring will enable an early and accurate diagnosis of fetal distress and will reduce unnecessary obstetric interventions.

In the late 1970s, the lack of randomized controlled trials and a steep rise in the cesarean delivery rate without a concomitant improvement in neonatal outcomes has started the debate about the effectiveness of the

routine use of CEFHRM. In 1987, the American College of Obstetricians and Gynecologists recommended that auscultation was an acceptable alternative to CEFHRM in low risk deliveries. The debate continued and provocative statements such as *'Has it been an absurd dream... a kind of game for unoccupied physicists and obstetricians?'* or *'Are we ready to throw it out and go back with confidence to the 175-year-old methodÿof clinical auscultation?'* [51] continued to be heard among obstetricians.

Banta and Thacker [52] discussed the limited evidence-based benefit of CEFHRM compared to the possible additional harm and cost. They conclude that: 'the technical advances required in the demonstration that reliable recording could be done seem to have blinded most observers to the fact that this additional information will not necessarily produce better outcomes. The current daily practice therefore reveals a discrepancy between the evidence-based data, which did not show a significant benefit of CEFHRM, and the widespread use of this technology. However, following 30 years of experience with this technology it seems that it's well-spread use is irreversible at the present time.'

References

1 Douglas RG, Stromme WB: Operative Obstetrics. New York, Appleton-Century-Crofts, 1957, pp 413–430.
2 Da KN: Obstetric Forceps: Its history and Evolution. Mosby, St. Louis, 1929.
3 Gultekin-Zootzmann B: The history of monitoring the human fetus. J Perinat Med 1975;3:135–144.
4 Mayor H: Biblioth Univ. de Geneve, November 9, 1818, quoted by Thomas H: Classical Contributions to Obstetrics and Gynecology. Springfield, Thomas, 1935.
5 Le Jumeau JA (de Kergaradec) (1): Memoire sur l'Auscultation. Applique a l'Etude de la Grossesse, Paris, 1822.
6 Le Jumeau JA (de Kergaradec) (2): Ann Med Physiol 1822;1:344.
7 Kennedy E: Observations of Obstetrical Auscultation, with an Analysis of the Evidence of Pregnancy and an Inquiry into the Proofs of the Life and Death of the Foetus in Utero. Dublin, Hodges & Smith, 1833.
8 Kilian, quoted by Jaggard WW: in Hirst BC (ed): A System of Obstetrics. Philadelphia, Lea Broth, 1888.
9 Kehrer FA, quoted by Fischer WM: in Fischer WM, Halberstadt E, Ruttgers G (eds): Kardiotokographie. Stuttgart, Thieme, 1976.
10 Schwartz S: Arch Gynaekol. 1870;1:361.
11 Schatz F: Arch Gynaekol. 1885;25:159.
12 Winckel F: Lehrbuch der Geburtshilfe. Leipzig, Veit, 1893.
13 Seitz L: Die Fetalen Herztone Unter der Geburt, Munchen, 1903.

14 Cunningham FG, Gant NF, Leveno KJ, Gilstrap LC, Hauth JC, Wenstrom KD: Antepartum assessment; in: Williams Obstetrics, ed 21. New York, McGraw-Hill, 2001, pp 1095–1110.
15 Hon EH: The electronic evaluation of the fetal heart rate (preliminary report). Am J Obstet Gynecol 1958;75:1215–1230.
16 Hammacher K: Früherkennung intrauteriner Gefahrenzustände durch Electrophonocardiographie und Fotographie; in Elert R, Hates KA (eds): Prophylaxe frühkindlicher Hirnschäden. Stuttgart, Thieme, 1966, pp 120–132.
17 Ray M, Freeman RK, Pine S, Hesselgesser R: Clinical experience with the oxytocin challenge test. Am J Obstet Gynecol 1972;114:1–9.
18 Banta HD, Thacker SB: Assessing the cost and benefits of electronic fetal monitoring. Obstet Gynecol Surv 1979;34:627–642.
19 Ventura SJ, Martin JA, Curtin SC, Mathews TJ, Park MM: Births: Final data for 1998. Natl Vital Stat Rep 2000; 48:1–100.
20 Goodlin R: History of fetal monitoring. Am J Obstet Gynecol 1979;133:323–352.
21 Evertson LR, Gauthier RJ, Collea JV: Fetal demise following negative contraction stress tests. Obstet Gynecol 1978;51: 671–673.
22 Freeman RK, Anderson G, Dorchester W: A prospective multi-institutional study of antepartum fetal heart rate monitoring. II. Contraction stress test versus nonstress test for primary surveillance. Am J Obstet Gynecol 1982;143:778–781.
23 Freeman RK, Garite TJ, Nageotte MP: Fetal Heart Rate Monitoring, ed 2. Baltimore, Williams & Wilkins, 1991.
24 Manning FA, Platt LD, Sipos L: Antepartum fetal evaluation: Development of a fetal biophysical profile. Am J Obstet Gynecol 1980;136:787–795.
25 Dayal AK, Manning FA, Berek DJ, Mussalli GM, Avila C, Harman CR, Menticoglou S: Fetal death after normal biophysical profile score: An eighteen-year experience. Am J Obstet Gynecol 1999; 181:1231–1236.
26 Manning FA, Snijders RJ, Harman CR, Nicolaides K, Menticoglou S, Morrison I: Fetal biophysical score. VI. Correlation with antepartum umbilical venous fetal pH. Am J Obstet Gynecol 1993;169: 755–763.
27 Ribbert LMS, Snijders RJ, Nicolaides KH, Visser GH: Relationship of fetal biophysical profile score and blood gas values at cordocentesis in severely growth-retarded fetuses. Am J Obstet Gynecol 1990; 163:569–571.
28 Salvesen DR, Freeman J, Brudenell JM, Nicolaides KH: Prediction of fetal acidemia in pregnancies complicated by maternal diabetes by fetal biophysical profile scoring and fetal heart rate monitoring. Br J Obstet Gynecol 1993;100: 227–233.
29 Yoon BH, Romero R, Roh CR, Kim SH, Ager JW, Syn HC, et al: Relationship between the fetal biophysical profile score, umbilical artery velocimetry, and fetal blood gas status determined by cordocentesis. Am J Obstet Gynecol 1993;169: 1586–1594.
30 Manning FA, Bondji N, Harman CR, Casiro O, Menticoglou S, Morrison I, et al: Fetal assessment by fetal biophysical profile score. VIII. The incidence of cerebral palsy among tested and non-tested perinates. Am J Obstet Gynecol 1998;178: 696–706.
31 Manning FA, Lange IR, Morrison I, Harman CR: Fetal biophysical profile score and the non-stress test: A comparative trial. Obstet Gynecol 1984;64: 326–331.

32 Platt LD, Walla CA, Paul RH, Trujillo ME, Loessor CV, Jacobs ND, et al: A prospective trial of the fetal biophysical profile versus the non-stress test in the management of high-risk pregnancies. Am J Obstet Gynecol 1985;153:624–633.
33 Clark SL, Sabey P, Jolley K: Non stress testing with acoustic stimulation and amniotic fluid volume assessment: 5973 tests without unexpected fetal death. Am J Obstet Gynecol 1989;160:694–697.
34 Nageotte MP, Towers CV, Astra T, Freeman RK: Perinatal outcome with the modified biophysical profile. Am J Obstet Gynecol 1994;170:1672–1676.
35 Miller DA, Rabello YA, Paul RH: The modified biophysical profile: Antepartum testing in the 1990s. Am J Obstet Gynecol 1996;174:812–817.
36 American College of Obstetricians and Gynecologists: Antepartum Fetal Surveillance. ACOG Technical Bulletin 188. Washington, ACOG, 1994.
37 Alfirevic Z, Walkinshaw SA: A randomized controlled trial of simple compared with complex antenatal fetal monitoring after 42 weeks of gestation. Br J Obstet Gynecol 1995;102: 638–643.
38 Nageotte MP, Towers CV, Asrat T, Freeman RK, Dorchester W: The value of a negative antepartum test: Contraction stress test and modified biophysical profile. Obstet Gynecol 1994;84:231–234.
39 Alfirevic Z, Neilson JP: Biophysical profile for fetal assessment in high-risk pregnancies (Cochrane Review). The Cochrane Library, Issue 4. Oxford, Update Software, 1999.
40 Alfirevic Z, Neilson JP: Doppler ultrasonography in high-risk pregnancies: Systematic review with meta-analysis. Am J Obstet Gynecol 1995;172: 1379–1387.
41 American College of Obstetricians and Gynecologists: Intrauterine growth restriction. Practice Bulletin No. 12, 2000.
42 Symonds EM: Fetal monitoring: Medical and legal implications for the practitioner. Curr Opin Obstet Gynecol 1994;6: 430–433.
43 Todd AL, Trudinger BJ, Cole MJ, Cooney GH: Antenatal tests of fetal welfare and development at age 2 years. Am J Obstet Gynecol 1992;167:66–71.
44 Stanley FJ, Blair E: Why have we failed to reduce the frequency of cerebral palsy? Med J Aust 1991;154:623–626.
45 Phelan JP, Ahn MO, Korst L, Martin GI: Is intrapartum fetal brain injury in the term fetus preventable? Am J Obstet Gynecol 1996;174:318.
46 American College of Obstetricians and Gynecologists: Use and abuse of the Apgar score. Committee opinion No. 174, January 1996.
47 Thacker SB, Berkelman RL: Assessing the diagnostic accuracy and efficacy of selected antepartum fetal surveillance techniques. Obstet Gynecol Surv 1986;41:121–141.
48 Platt LD, Paul RH, Phelan J, Walla CA, Broussard P: Fifteen years of experience with antepartum fetal testing. Am J Obstet Gynecol 1987;156:1509–1515.
49 Mohide PT, Keirse MJ: Biophysical assessment of fetal wellbeing; in Chambers I, Enkin M, Keirse MJ (eds): Effective Care Pregnancy and Childbirth, vol I. Pregnancy. New York, Oxford University Press, 1991, pp 477–510.
50 Enkin M, Keirse MJ, Renfrew M, Neilson J: A Guide to Effective Care in Pregnancy and Childbirth, ed 2. New York, Oxford University Press, 1995, pp 410–422.

51 Sureau C: Historical perspectives: Forgotten past, unpredictable future. Baillières Clin Obstet Gynaecol 1996; 10:167–184.

52 Banta DH, Thacker SB: Historical controversy in health technology assessment: The case of electronic fetal monitoring. Obstet Gynecol Surv 2001;56:707–719.

Michael Y. Divon, MD

Department of Obstetrics and Gynecology, Lenox Hill Hospital,
130 E 77th Street, New York, NY 10021 (USA)
Tel. +1 212 434 2160, Fax +1 212 434 2180, E-Mail Mdivon@lenoxhill.net