devices can give us biparietal and other diameters with accuracy. Can we, however, equate biparietal diameter (BPD) or fetal size in general with pulmonary surfactant maturity? Clearly, we cannot. Witness the macrosomic infant of a diabetic mother who develops the respiratory distress syndrome (RDS) with a BPD greater than 95 mm.

We think that the best current method for assessing the fetus's ability to breathe adequately after birth is the assessment of surfactant phospholipids in the amniotic fluid, the lecithin/sphingomyelin ratio, and, particularly, phosphatidylglycerol (PG). In a series now exceeding 500 amniocenteses, we have had only two infants develop RDS when PG was present in the amniotic fluid. One of these was a septic baby, and the other was the premature infant of a mother with lupus nephritis who was on a regimen of steroids in large doses.

We challenge the statement that amniocentesis near term carries a 1% risk to the fetus; it is certainly much lower than that, particularly, as noted by the authors, when performed under ultrasonic guidance.

We agree with the authors that rupture of a previous uterine scar with labor is a rare event. In a recent review of 80,183 deliveries, 10,691 of which were cesarean sections, only six cesarean hysterectomies were performed for uterine rupture, only three of which involved rupture of a previous cesarean section scar. There was asymptomatic significant dehiscence of a previous section scar in eight additional cases. When assessment of pulmonary surfactant phospholipids is not available or amniocentesis fails, we await the onset of labor as the safest policy for the baby, provided that the mother has prompt access to the hospital. If the previous cesarean section was for a nonrecurring cause, we might even elect an attempt at vaginal delivery and expect to succeed in 50% of cases.

Whether one allows labor or determines surfactant levels in amniotic fluid, the thing one must not condone is the delivery of premature babies who develop RDS because of elective cesarean section at some arbitrary gestational age. We congratulate the authors on bringing this problem to the attention of the specialty in this age of doggedly increasing cesarean section rates.

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Reply to Dr. Plauché

To the Editors:

We appreciate Dr. Plauché's comments and agree both that gestational age is difficult to estimate with certainty and that fetal size does not necessarily correlate with pulmonary maturity. Furthermore, even "term" infants who are delivered by elective repeat cesarean section are at risk for the respiratory distress syndrome. Indeed, in our series, 29 affected infants (41%) were judged to have been delivered at term.

Determination of amniotic fluid phosphatidylglycerol is clearly a useful method of evaluating fetal pulmonary maturity. We believe that awaiting the onset of labor at term is a reasonable alternative to invasive testing.

In choosing between these approaches, one must balance their associated risks. Amniotic fluid phosphatidylglycerol is a relatively new test. As with most new diagnostic tests, as the spectrum of patients to whom the test is applied broadens, the percentage of false positive results is likely to increase.[1] There is also the problem of laboratory error and the possibility that the specimen will be lost. Although the overall risk of serious injury to the fetus from amniocentesis is probably less than 1%, there clearly is a risk. In addition, some women refuse to undergo the procedure.

The frequency of uterine rupture when a woman who has had a previous low transverse cesarean section is allowed to undergo labor is approximately 0.5%, which is about the same as the frequency of uterine rupture at the time of scheduled cesarean section.[2] Other potential risks which may be associated with allowing these women to initiate labor spontaneously include an additional anesthetic risk in a patient who may have recently eaten and a possible increased frequency or duration of postpartum fever. These potential risks have not been adequately studied. The monetary cost of this latter approach is clearly far less than that of performing amniocentesis with ultrasound and phospholipid testing.

We agree with Dr. Plauché that either approach is acceptable, and that elective cesarean delivery at an arbitrary gestational age, without prior evidence of fetal pulmonary maturity, is to be avoided.

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Importance of sinusoidal fetal heart rate pattern

To the Editors:

In their recent article, "Sinusoidal fetal heart rate pattern: Its definition and clinical significance" (AMERICAN JOURNAL OF OBSTETRICS AND GYNECOLOGY 142:

Table I. Intrapartum SFHR patterns associated with fetal or neonatal death

Author	*Obstetric complications*	*Associated FHR changes*	*Delivery*	*Apgar scores*	*Birth weight (gm)*	*Morbidity/mortality*
Sacks (1980)	Postdates, meconium	Prolonged bradycardia	Cesarean section	0/0	4,564	Stillborn (FD)
Sibai	Vaginal bleeding	Prolonged bradycardia	Cesarean section	0/0	3,170	Acidosis, anemia, seizures (ND)
	Postdates, polyhydramnios	Late decelerations, beat-to-beat variability	Cesarean section	2/4	—	Hydrops, acidosis, anemia (ND)
Goldstein (1981)	Decreased fetal movements, term	Prolonged bradycardia, variable decelerations	Cesarean section	0/0	—	Pulmonary edema, aspiration of amniotic fluid (ND)
Cetrulo (1976)	Postdates, meconium	Prolonged bradycardia	Cesarean section	0/0	4,400	Stillborn (FD)
Baskett (1974)	Postdates, pregnancy-induced hypertension, meconium	None	Cesarean section	0/0	4,080	Disseminated intravascular coagulation, meconium aspiration (ND)

FD = Fetal death; ND = neonatal death.

Table II. Number of cases reported and number of illustrations for each

Author	*No. of cases*	*Figures reviewed*
Kubli	12	2
Rochard	20	1
Gray	17	2
Johnson	31	5*

*Three figures from same case.

1033, 1982), Drs. Modanlou and Freeman reviewed 23 publications that had appeared since 1972 and that contained 41 tracings that had been defined as sinusoidal fetal heart rate (SFHR) patterns. According to the authors, 27 tracings were confirmed as true sinusoidal rhythms while 11 did not meet their criteria; three tracings were reported as equivocal. They concluded that SFHR patterns indicate significant fetal and/or neonatal jeopardy because, in the 27 cases which they interpreted as "true" sinusoidal patterns, 24 were associated with fetal or neonatal death and/or severe morbidity.

We have conducted a comprehensive review of the literature and from the information we have obtained we wish to question their conclusions. Our review included a total of 188 cases of SFHR patterns that appeared in the English literature since 1972. We excluded one of the articles reported by Modanlou and Freeman, since that case included sinusoidal patterns only during the neonatal period (McReid and associates[1]). Seventy-three percent of the cases reported ended with live-born infants, and the majority of these infants had minimal morbidity. There were 52 fetal/neonatal deaths, but only seven of these occurred when the pattern was initially diagnosed during labor. In each of these there were significant additional clinical factors that may have accounted for the poor outcome (see Table I). On the other hand, 55% of pregnancies with a SFHR pattern noted during antepartum testing resulted in deliveries complicated by fetal/neonatal death, and more than 95% of cases with the same patterns first observed in labor ended with live-born infants.

Since Modanlou and Freeman reviewed only 28% of the reported cases and since they made generalized conclusions from reviews of very short tracings in articles that did not present figures or tracings from all (Table II) cases, we feel that their conclusions cannot be sustained. SFHR patterns noted during the antepartum period with either nonstress testing or oxytocin challenge testing have a poor prognosis, especially if the patients are rhesus isoimmunized. The same conclusion does not hold when the pattern is first detected in labor; those cases noted during intrapartum electronic fetal monitoring and without associated clinical abnormalities are almost always benign.

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REFERENCE

1. McReid, M., Jenkins, J., and McClure, G.: Sinusoidal heart rate rhythms in severe neonatal hypoxia, Arch. Dis. Child. **54**:432, 1979.

Reply to Drs. Sanchez-Ramos, Robertson, and Beydoun

To the Editors:

We do not doubt that Sanchez-Ramos and associates did a comprehensive review of the literature on the subject in question, but their review of our paper was not diligently done. In our paper, we intended to review only the tracings presented in 23 published pa-