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-

pers. It is true that some papers reported several cases of sinusoidal fetal heart rate pattern. Since each author is more prone to publish the most representative tracing, we elected to review only cases with published tracings rather than count all case reports. Sanchez-Ramos and associates appear to have missed this important point.

Sanchez-Ramos and associates with their letter are adding to the already existing confusion on the subject in that they accept all reported cases as "true sinusoidal pattern." Because of the confusion and diverse interpretations by some authors, we decided first, to establish a definition of sinusoidal heart rate pattern; second, to review all published tracings based on that definition (see the original paper for the definition of sinusoidal heart rate pattern); and third, to have my co-author, Dr. Roger Freeman, review the tracings blindly, based on our agreed definition, without knowing the outcome of the patient whose tracing was being reviewed.

We would suggest to Sanchez-Ramos and associates that they develop a definition of sinusoidal fetal heart rate pattern that is acceptable to them and review all 188 cases they may have tracings on. Only through a defined description of the sinusoidal heart rate pattern and strict adherence to that definition can one appreciate the true clinical significance of the sinusoidal heart rate pattern.

Regarding their last point on antepartum or intrapartum sinusoidal fetal heart rate pattern, the same answer applies. With our definition, the sinusoidal heart rate pattern is an ominous sign, except when associated with alphaprodine administration, whenever it appears, antepartum, intrapartum, or during the neonatal period.

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Sickle cell trait and pregnancy

To the Editors:

In their article, entitled "Massive pulmonary embolism following delivery of a patient with sickle cell trait" (AM. J. OBSTET. GYNECOL. 143:722, 1982), Tung Van Dinh and coauthors have described one case of fatal pulmonary embolism in which "a massive 'saddle' pulmonary embolus . . . completely obstructed the pulmonary trunk and the two main pulmonary arteries." They also dwelled at considerable length on the observation that the woman had sickle cell trait.

It is not surprising that an occasional black parturient patient who dies has sickle cell trait since one of 12 black women has sickle cell trait.

What was the purpose of this report by Tung Van Dinh and coauthors? Was it to try to implicate sickle trait in the fatal outcome? If so, the authors appear to have found little supporting evidence in the literature for such a phenomenon. Or, perhaps, was it to emphasize just the opposite, that is, pregnant women with sickle cell trait are extremely unlikely to die suddenly? They comment, "The only reported sudden death in pregnancy presumably associated with sickle cell trait occurred in 1957." That woman had eclampsia.

I strongly suspect that their purpose, unfortunately, was to try to engender fear of adverse outcome for pregnancies in which the mother has sickle cell trait, for in the last sentence they state, "Since it is known that a relationship between sickling and embolization exists, it is important to prevent embolism from occurring, thus emphasizing the importance of a complete prepartum workup to define such high-risk patients with sickle cell disease or trait." I find this sentence very confusing. For example, I know of no firm evidence of an association between sickle cell trait and embolization with or without pregnancy. Do the authors?

Our very extensive experience has been that women with sickle cell trait "perform" as well reproduction-wise as do black women whose red blood cells do not sickle. The only perplexing problem associated with their reproducing is the potential for their offspring to have a sickle cell hemoglobinopathy.

Too long the medical profession has been trying to make the individual with sickle cell trait unhealthy! Fortunately, appropriate data do not exist to justify such a view.

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Reply to Dr. Pritchard

To the Editors:

We wish to thank Dr. Pritchard for his interest in our article. We completely agree with Dr. Pritchard that patients with sickle cell trait fare well during their pregnancies, except for an increased incidence of pyelonephritis.[1, 2]

Our purpose in reporting our case of fatal and massive pulmonary embolism was not "to engender fear of adverse outcome for pregnancies in which the mother has sickle cell trait" (please, trust our good hearts!) but to describe an extremely rare and perplexing case in which sickle cell trait, if not conclusively responsible for death, may have been a contributing factor.

In fact, McCormick,[3] in reviewing a total of 120 autopsies of patients with sickle cell trait, found that in