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.

H. D. Modanlou, M.D.
Memorial Hospital Medical Center of Long Beach
2801 Atlantic Avenue
P. O. Box 1428
Long Beach, California 90801

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.

Jack A. Pritchard, M.D.
Department of Obstetrics and Gynecology
Southwestern Medical School
The University of Texas Health Science Center at Dallas
5323 Harry Hines Boulevard
Dallas, Texas 75235

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

12.5% of cases, "the trait caused death or greatly contributed to it."

Sears,[4] in his exhaustive review of the literature, concluded that potential hazards "may exist for the sickle cell trait, but they await convincing demonstration by properly controlled studies." The purpose of a brief report such as ours is, in fact, to stimulate controversy and thought so that these properly controlled studies will be performed and we will no longer have to rely on previous dogma or opinion.

We thank the Editors for giving us the opportunity to reply to one eminent teacher of modern obstetrics.

Tung Van Dinh, M.D.
Paul J. Boor, M.D.
Departments of Obstetrics and Gynecology and Pathology
The University of Texas Medical Branch
Galveston, Texas 77550

REFERENCES

1. Whalley, P. J.: Pritchard, J. A., and Richards, J. R., Jr.: Sickle cell trait and pregnancy, JAMA **186:**1132, 1963.
2. Pritchard, J. A., Scott, D. E., Whalley, P. J., Cunningham, F. G., and Mason R. A.: The effects of maternal sickle cell hemoglobinopathies and sickle cell trait on reproductive performance, AM. J. OBSTET. GYNECOL. **117:**662, 1973.
3. McCormick, W. F.: Abnormal hemoglobins. II. The pathology of sickle cell trait, Am. J. Med. Sci. **241:**329, 1961.
4. Sears, D. A.: The morbidity of sickle cell trait, Am. J. Med. **64:**1022, 1978.

One-hour observation period prior to tocolytic therapy

To the Editors:

I should like to take this opportunity to draw to the attention of the readers of this JOURNAL what I consider to be a significant omission in published protocols for the use of β-adrenergic tocolytic agents. This is particularly important at this time because of the current availability of an approved product marketed for this purpose (Yutopar [ritrodrine], Astra Pharm. Products, Inc.).

At the University of New Mexico School of Medicine, we have had over 6 years' experience with the utilization of these drugs for the suppression of labor. Our experience has largely been with terbutaline. Because it has been shown that in any series of patients with suspected premature labor, the condition will subside in at least half of them with the intravenous administration of fluid alone, and because these drugs theoretically could produce more serious side effects in the hypovolemic patient, our established routine prior to the administration of these drugs has been to administer a fluid load. This has generally been planned to represent approximately 10 ml per kilogram of body weight of 5% dextrose in Ringer's lactate over the first hour. This not only provides some fraction of this administered fluid for volume expansion but also necessitates a 1-hour period of observation for the detection of any effects on the patient's contraction pattern prior to the initiation of tocolytic therapy. We also begin tapering the dosage within 2 to 3 hours after cessation of contractions. During a 5-year period of study, encompassing nearly 500 patients, we have seen no case of pulmonary edema develop with this regimen or the subsequent administration of beta-adrenergic drugs. We do not use corticosteroids with any frequency at this institution. We do not use potassium supplementation, except when extreme hypokalemia is observed during therapy (a rare development).

As the only Level III referral center in this large geographic region, we have had occasion to receive in transfer for the development of "toxicity" many patients under tocolytic therapy from other institutions. This toxicity has generally consisted of excessive maternal heart rate in response to usual doses. Our regular observation with such patients is the noticeable lack of appropriate administration of fluid prior to the initiation of the drug. In some cases, simple administration of fluid alone has resulted in the subsidence of not only the signs of toxicity but also the contraction pattern.

We wish to call to the attention of the readership and the drug manufacturers the wisdom of a 1-hour period of appropriate administration of fluid and observation prior to the utilization of β-adrenergic drugs. We consider the lack of such recommendation to be a significant omission in published protocols by the manufacturers for the use of these agents. We recognize the implications of excessive administration of fluid in regard to the development of pulmonary edema,[1, 2] and would advise judicious use of fluid after the drug is started. To date, our regimen has been both successful and safe in common use, with the qualifications mentioned. Steering a safe course between extremes would seem to be most appropriate plan.

Richard P. Perkins, M.D.
Professor of Obstetrics and Gynecology
University of New Mexico School of Medicine
2211 Lomas Boulevard, N.E.
Albuquerque, New Mexico 87131

REFERENCES

1. Katz, M., Robertson, P. A., and Creasy, R. K.: Cardiovascular complications associated with terbutaline treatment of premature labor, AM. J. OBSTET. GYNECOL. **139:**605, 1981.
2. Philipsen, T., Eriksen, P. S., and Lynggard, F.: Pulmonary edema following ritodrine-saline infusion in premature labor, Obstet. Gynecol. **58:**304, 1981.