our carelessness. Perhaps being made more sensitive to their plight will benefit humankind in a way more significant than it may seem at face value. Food for the hungry is important, but survival of the planet to feed all of us is most important.

Fredericka Heller, MD
530 Kenhorst Blvd., Reading, PA 19611

Response declined

Fetal biophysical and umbilical cord gases

To the Editors: The article by Vintzileos et al. (Vintzileos AM, Fleming AD, Scorza WE, et al. relationship between fetal biophysical activities and umbilical cord blood gas values AM OBSTET GYNECOL 1991;165:707-13) is an important contribution to our understanding of some aspects of fetal pathophysiology and its detection before significant morbidity or fetal death. I have some observations about this paper.

First, the title of Table VI should be Umbilical venous blood rather tham Umbilical artery blood.

Second, the authors did not comment on the influence, if any, of the maternal oxygen administration rate of 3 L/min on umbilical cord venous and artery P_{O_2}. In spite of maternal oxygen administration the values of umbilical venous and artery P_{O_2} with reactive nonstress test and fetal breathing movements present appear to be low. Would a contraction stress test have been more useful in identifying impending fetal-neonatal morbidity at a higher value of venous and artery P_{O_2}?

Third, it is not surprising to note that fetal hypercapnia occurred only when fetal movement and tone were absent and was associated with significant fetal acidosis and hypoxia. This occurrence is explained by the physiologic fact that C_{O_2} exchange across placenta membranes or alveoli is much more efficient than the exchange of oxygen.

Houchang Modanlou, MD
2801 Atlantic Ave., P.O. Box 1428, Long Beach, CA 90801-1428

Reply

To the Editors: We thank Modanlou for his interest in our work. We agree with his first comment that the title of Table VI should be umbilical venous blood rather than umbilical artery blood.

In our opinion the relatively low umbilical cord venous and artery P_{O_2} in spite of maternal oxygen administration of 3 L/min was related to the fact that the oxygen administration was of very short duration. This time period was from satisfactory epidural analgesia (when skin incision was made) until delivery of the infant. However, the main emphasis of our study was to investigate the relationship between the presence or absence of the individual fetal biophysical activities and umbilical cord blood gas measurements and to compare the different levels of pH, P_{O_2}, P_{CO_2}, and bicarbonate and base excess at which the individual biophysical activities become compromised. We did not attempt to establish normal umbilical cord venous and artery P_{O_2} values. Therefore for reasons of comparison the absolute P_{O_2} levels were not important because all patients received epidural analgesia and had the same degree of maternal oxygenation. We doubt that a contraction stress test could be useful in identifying impending fetal-neonatal morbidity. In general the appearance of late decelerations of the fetal heart rate in response to uterine contractions may suggest fetal hypoxia caused by uteroplacental insufficiency. However, this concept is valid only during the intrapartum period because the frequency and intensity of the uterine contractions can be monitored with internal monitoring techniques. External monitoring techniques like those used in the antepartum contraction stress test, however, are not capable of measuring the intensity of the uterine contractions and therefore the strength of the stimulus to the fetus. In our opinion any judgment about the presence or absence of fetal hypoxia when the intensity of the stress factor (uterine activity) is unknown is not reasonable and has no scientific basis.

We agree with Modanlou's comment that carbon dioxide exchange across placental membranes is much more efficient that oxygen exchange. We also believe that fetal hypercapnia in the absence of fetal movement and tone may also be related to umbilical cord compression, as is frequently seen in cases in which oligohydramnios is associated with intrauterine growth retardation and compromised biophysical activities.

Anthony M. Vintzileos, MD, Winston A. Campbell, MD, and John F. Rodis, MD
Division of Maternal-Fetal Medicine, Department of Obstetrics and Gynecology, University of Connecticut Health Center, Farmington, CT 06032

Hemolytic disease of the fetus

To the Editors: Weiner et al. (Weiner CP, Williamson RA, Wenstrom KD, et al. Management of fetal hemolytic disease by cordocentesis. I. Prediction of fetal anemia. AM J OBSTET GYNECOL 1991;165:546-53) report a management scheme for hemolytic disease of the fetus based on results from cordocentesis. Although the authors stop short of recommending the abandonment of amniocentesis, it is clear that in their unit they have. Although their results were good, there was no prospective comparison to an amniocentesis-based management scheme. Unfortunately, the authors (and readers) can only speculate on the broad range of relative advantages and disadvantages of each scheme.

My questions regard the selection of patients for invasive testing. The authors state that an indirect Coombs' titer of $\geq$1:8 was selected for testing because in their unit severe hemolytic disease has occurred at 1:8 but not below. This same threshold was reported by Reece et al.[1]; it places the critical titer for their laboratories at 1:4, which is one dilution stricter than that recommended by the American College of Obstetricians and Gynecologists Technical Bulletin[2] (>1:8) and two dilutions stricter than the data-based recommen-

dations of Freda[3] and Queenan[4] (>1:16). As a consequence of this stricter critical titer, more patients require invasive testing or require it earlier in pregnancy.

My questions: (1) In their current series, in a first sensitized pregnancy what was the lowest fetal hematocrit observed when the indirect Coombs' titer within 1 to 2 weeks of sampling was ≤1:16? (2) Do the observations in the current series justify invasive sampling in the first sensitized pregnancy when a recent indirect Coombs' titer is 1:8 or 1:16? (3) Can their historic observation of severe fetal hemolytic disease with a titer of 1:8 be explained by (a) its occurrence in an other-than-first sensitized pregnancy, (b) delivery remote from the 1:8 titer with possible intervening unobserved increases in titer, or (c) an unobserved increase in titer caused by invasive sampling after the titer? MacGregor et al.[5] reported a rise from 1:8 to 1:4096 within weeks of cordocentesis.

If the answers to these questions do not support invasive testing in first sensitized pregnancies with 1:8 or 1:16 titers, the authors should acknowledge this and adjust their critical titer. This adjustment would allow the selection of invasive testing only when the fetal risk truly warrants it.

J.A. Spinnato, MD

Department of Obstetrics and Gynecology, University of Louisville, Louisville, KY 40292

REFERENCES

1. Reece EA, Copel JA, Scioscia AL, Grannum PAT, DeGennaro N, Hobbins JC. Diagnostic fetal umbilical blood sampling in the management of isoimmunization. Am J Obstet Gynecol 1988;159:1057-62.
2. American College of Obstetricians and Gynecologists. Management of isoimmunization in pregnancy. ACOG Tech Bull 1990;148:2.
3. Freda JF. The Rh problem in obstetrics and a new concept of its management using amniocentesis and spectrophotometric scanning of amniotic fluid. Am J Obstet Gynecol 1965;92:341-74.
4. Queenan JT. Modern management of the Rh problem. 2nd ed. Hagerstown, Maryland: Harper & Row, 1977: 31-3.
5. MacGregor SN, Silver RK, Sholl JS. Enhanced sensitization after cordocentesis in a rhesus-isoimmunized pregnancy. Am J Obstet Gynecol 1991;165:382-3.

Reply

To the Editors: We thank Spinnato for his inquiry. We expect may questions will arise regarding the proposed management scheme because it is difficult to give adequate detail in an initial publication.

Spinnato asked about the antibody titer at which an invasive evaluation should be undertaken during the first sensitized pregnancy. His specific concern was that we selected 1:8 although the American College of Obstetricians and Gynecologists Technical Bulletin states that a titer of <1:16 indicates that the fetus is not in serious jeopardy. His question is best answered by quoting directly from that technical bulletin. "In general, it is believed that, if the antibody titers remain less than 1:16 in an initial immunized gestation, the fetus is not in serious jeopardy. *It is important to know, however, that this value is somewhat dependent on local laboratory methods, skill of laboratory personnel, and previous experiences related to titers within that institution* [emphasis added]."

Spinnato is not the first to have missed this point. With the decreasing incidence of fetal hemolytic disease, experience with the entity has shrunk. Our critical titer has been and remains 1:8 and is the result of many years of experience in one laboratory with a wide referral area. All physicians involved in the evaluation of fetal hemolytic disease should find the critical titer in their individual laboratories. Obviously this is impossible in a small hospital; regionalization is required.

In our experience patients have frequently been referred with a low indirect Coombs' titer from the local laboratory, only to have a much higher titer when tested by the University of Iowa Hospitals and Clinics blood bank. Whether this reflects a change in sensitization or differences in laboratories, I cannot state.

Our data base does not include information as to whether the pregnancy under evaluation was the first sensitized pregnancy. As a rule we monitored each patient during her first sensitized pregnancy with serial indirect Coombs' titers. We require a titer ≥1:8. Ten nulliparous women have undergone cordocentesis for red blood cell isoimmunization. Two of those fetuses had anemia when first evaluated. In one, sensitization was to D and the indirect Coombs' titer was 1:1024. In the other, sensitization was to Kell and the indirect Coombs' titer was 1:8. However, most of the patients who have required transfusion with low indirect Coombs' titers were not in their first sensitized pregnancies. Thus our observation of severe hemolytic disease with a titer of 1:8 is mostly, but not exclusively, explained by its occurrence in a subsequent sensitized pregnancy. As Spinnato notes, other have had similar experiences with their laboratories.

Spinnato refers to a case report by MacGregor et al., who observed an abrupt increase in antibody titer within weeks of cordocentesis. However, I note that the fetus in question was already anemic when the titer was 1:8. This raises an important point. Cordocentesis is but a procedure. One must not only know what the normal values are, but what to do with them. With our management scheme we have tried to supply a course of action. The procedure can also be misused. To prevent worsening of disease we avoid the placenta in patients with low-grade sensitization by sampling from a midsegment.

In summary, the 1:16 guideline in the American College of Obstetricians and Gynecologists Technical Bulletin was never intended to be engraved in stone. It is clear that at the University of Iowa our critical titer is 1:8. Our experience is that these fetuses are at risk and benefit from accurate testing.

Carl P. Weiner, MD

Department of Obstetrics and Gynecology, University of Iowa Hospitals and Clinics, Iowa City, IA 52242