

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.

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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 than 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 PO_2 . In spite of maternal oxygen administration the values of umbilical venous and artery PO_2 with reactive non-stress 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 PO_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 CO_2 exchange across placenta membranes or alveoli is much more efficient than the exchange of oxygen.

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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 PO_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, PO_2 , PCO_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 PO_2 values. Therefore for reasons of comparison the absolute PO_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 than 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.

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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 Bulletin² ($> 1:8$) and two dilutions stricter than the data-based recommen-