### Magnesium sulfate is an unfit anticonvulsant in eclampsia

*To the Editors:* I read with great interest the article of Sibai (Sibai BM. Magnesium sulfate is the ideal anticonvulsant in preeclampsia-eclampsia. Am J Obstet Gynecol 1990;162:1141-5). As Sibai pointed out, magnesium sulfate has been recommended in the treatment of eclampsia; however, comprehensive data regarding its reliance and safety are lacking. I believe that patients will not be injured by the drug if the principles of treatment are followed.

There are multiple actions of magnesium sulfate that may be useful or nonhelpful to the fetus and mother. Effects on fetal heart variability and associated low Apgar scores, respiratory depression, hyporeflexia, and hypocalcemia have been reported in the neonates of mothers receiving intravenous magnesium sulfate. These effects were reported in premature infants in association with fetal growth retardation. Such complications may be present in these infants whether magnesium sulfate is or is not used. The perinatal mortality rate was reported to be as much as 17.1%[1] and 45%[2] when magnesium sulfate was used. Our hospital has not used magnesium sulfate, and the perinatal mortality rate is 2.78% (3/108).[3] In contrast, the maternal mortality rate in other large series of patients with eclampsia ranged between 3.3% and 14.4%, and the incidence of intracerebral hemorrhage was as high as 10.7% (Sibai's article). Dr. Sibai's patient was resuscitated and her seizures were controlled with magnesium sulfate. In our hospital taking care of patients with eclampsia without the use of magnesium sulfate has resulted in a maternal mortality of zero (0/106).[3]

Parenteral magnesium sulfate is the drug of choice to prevent convulsions in cases of eclampsia, but it is not suitable as an anticonvulsant in eclampsia. Magnesium sulfate controls convulsions of eclampsia but seriously endangers the health of both the mother and her fetus. We consider magnesium sulfate to be dangerous in our hospital; hence, it is not used.

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2. Ke Y, Lin Q, Wang S, et al. Pathology of obstetrics. ed 2. Beijing: People's Hygienical Publication Society, 1959:73.
3. Luan J. Treatment of eclampsia by early interruption of pregnancy: a 15-year review. Asia Oceania J Obstet Gynecol 1989;15:33-5.

### Reply

*To the Editors:* I do appreciate the interest of Dr. Luan in the subject of eclampsia and magnesium sulfate. Dr. Luan cites some statements from *my* manuscript without indicating the purpose or reason for including them, which makes it difficult to respond to his letter. He then cites a perinatal mortality rate of 2.78% in his experience with eclampsia as an indication not to use magnesium sulfate. He compares it to previous perinatal mortality reported in 1959 (his reference 2) and in 1979 (his reference 1). Again, I fail to see the association since he does not explain what is meant by perinatal mortality. Did he include patients who had stillbirths or neonatal deaths before arrival to the hospital? What are the gestational ages? Did he include patients with gestational ages <28 weeks? Currently, neonatal survival for infants >28 weeks' gestation is almost 100% in Memphis. This has nothing to do with magnesium sulfate. In addition, neonatal survival for preterm babies in Memphis is definitely superior to that in China. He also mentions maternal mortality without use of magnesium sulfate. Again, he does not mention the condition of the patient on admission and how many were excluded before admission.

Finally, he concludes that magnesium sulfate is an unfit anticonvulsant in eclampsia. It is surprising for someone who has never used the drug to make such a statement. In addition, he does not report what drug he uses. It is ridiculous to make such statements without data to support them.

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### The issue of animal rights and human rights

*To the Editors:* I agree with much of what Mister Maharry (Maharry TM. The issue of animal rights and human rights. Am J Obstet Gynecol 1991;164:1543-8) says about medical research being dependent on animal experimentation. I support the use of animals in appropriately screened experiments. I am also a member of the Physicians Committee for Responsible Medicine, and I believe that there are substantial experiments going on in this country that use animals. A number of these experiments are superfluous or do not particularly further the cause of medical knowledge. I believe this type of experiment still needs to be weeded out. Although some animal activist groups have been violent, I believe that in general the cause for protection against cruelty to animals has been advanced. We have all become more sensitive to the injustices that humans have dealt the animal world.

I cannot deny that violence is not an appropriate response, but as an animal rights advocate I believe closer scrutiny of the use of animals in experimentation and closer scrutiny of the usefulness of the experiments themselves is an appropriate response. Do not lump all animal rights activists with the few violent ones. We need to take a close look at our respect for the animal kingdom. Many species have been destroyed through

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 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.

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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 $P_{O_2}$ 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.

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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 Bulletin[2] (>1:8) and two dilutions stricter than the data-based recommen-