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Reply

To the Editors:

We appreciate Dr. Wagner's interest in our article. We are encouraged by the strong stance Dr. Wagner has taken supporting our position that a fetal monitor record that uses an autocorrelated ultrasound signal is indisputably different from a fetal heart rate record that uses a direct fetal electrocardiogram as a trigger source. We believe it is important to emphasize this point since the previous scientific reports, except for two case reports,[1, 2] in our opinion did not make this distinction clear. Furthermore, the issue was blurred by marketing statements such as " . . . almost identical to a direct ECG . . . " and " . . . the variability is clinically identical to direct ECG traces . . . " (Hewlett-Packard advertisement). The autocorrelation function tends to reinforce periodic data by correlating the signal with a time-delayed version of itself; it is, in effect, a moving average of a series of ultrasonic fetal sonocardiograms. The fetal heart rate pattern is thereby smoothed so that it is less "noisy" (that is, less displeasing to the eye). Even though it may appear to the noncritical observer to be identical to the fetal heart rate pattern derived from the fetal electrocardiogram, this is categorically not possible because the ultrasound signal is a biomechanical signal with a waveform that varies considerably from beat to beat while the electrocardiogram is a relatively constant bioelectric signal. A major problem associated with monitors that use autocorrelation is that any periodic signal, be it maternal or extraneous transducer noise, will be reinforced and plotted as a fetal heart rate record (spurious data). The clinical consequences of an incorrectly interpreted fetal monitor record need no further discussion. Another weakness of the system becomes manifest in situations where the signal interval changes rapidly, as is the case with variable decelerations (descending and ascending limbs) or fetal arrhythmia. The autocorrelation circuitry tends to reject these legitimate signals as noise, therefore suppressing them from the record (missing data). The so-called "signal quality indicator" light featured in some fetal monitors is of limited usefulness in the face of "spurious" data of "good" quality. The autocorrelated ultrasonic tracing of the jogging man (Fig. 6 in the article) is an example of an extrafetal periodic noise that in our view resulted from the repetitive movement of the ultrasonic transducer against the skin of the abdominal wall. It is unlikely that the abdominal vessels were the sole source of the signals, as suggested by Dr. Wagner, since the jogger's heart rate never exceeded 90 bpm during the exercise. We agree with Dr. Wagner that more work needs to be done in the area of external techniques to record fetal heart rate patterns. Meanwhile, however, we must be aware that underlying a cosmetically pleasing fetal monitor tracing there may be spurious data that may be detrimental to fetal well-being.

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Breast-feeding, antioxidants, and retinopathy of prematurity

To the Editors:

Ostrea et al. (Ostrea EM Jr, Balun JE, Winkler RW, Porter T. Influence of breast-feeding on the restoration of the low serum concentration of vitamin E and β-carotene in the newborn infant. Am J Obstet Gynecol 1986;154:1014-7) observed that breast-feeding raised the serum levels of vitamin E and β-carotene in term infants and speculated that human milk might protect susceptible premature infants against oxygen toxicity via the antioxidant effect of these substances.

Nearly 40 years ago the incidence of retrolental fibroplasia among premature infants in a Chicago nursery was one fifth that of prevailing rates.[1] Sanford et al.[1] observed: "The only difference in care that might account for the lower incidence in our premature group (6%) and others is that we give breast milk as a feeding. Whether this contains some protective substance we do not know." Lately, a review of records of premature births in Philadelphia showed that even partial breast-feeding was associated with half as much retinopathy compared with that found in premature babies given only formula.[2]

Ostrea et al. make an important suggestion, but I offer a caution. Premature infants given human milk also have improved vitamin E status,[3] but it is too early to focus on specific factors to account for its protective effect. Human milk is an extremely complex substance. It is likely that several factors in human milk interact to protect premature infants against retinopathy. Moreover, retinopathy and other manifestations of oxygen toxicity represent just one disease category among several to which premature babies are susceptible. Enteric diseases, sepsis, meningitis, and even hyaline membrane disease appear to be prevented by human

milk.[4, 5] These too should be accounted for when controlled trials with human milk are carried out.

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Reply

To the Editors:

We have presented evidence that breast milk (especially colostrum) contains high concentrations of vitamin E and β-carotene so that breast-feeding in the term infant significantly raises the serum concentration of these two important antioxidants. However, it remains to be determined whether this phenomenon can similarly be observed in the breast-fed premature infant and whether breast-feeding can therefore protect the infant from the damages of oxygen toxicity. Although there is circumstantial evidence to suggest the latter, as pointed out by Cunningham, we tend to agree with him that since milk is such a complex substance, it will be difficult to ascribe whatever beneficial effects milk has to one substance alone. Currently, we are in the midst of a clinical trial to test whether supplementation of vitamin E and β-carotene in low birth weight, premature infants receiving high series of oxygen treatment will prevent the occurrence of bronchopulmonary dysplasia and retinopathy of prematurity. Our results are too preliminary to permit any disclosure at this point.

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Recurrent carcinoma in situ of vulva in skin graft

To the Editors:

In the July 1986 issue of the GRAY JOURNAL, the article by Cox, Coffin, and Kaplan (Cox SM, Kaufman RH, Kaplan A. Recurrent carcinoma in situ of the vulva in a skin graft. AM J OBSTET GYNECOL 1986;155: 177-9) caught my eye. They reported on a case of recurrent carcinoma in situ of the vulva in a skin graft. I too have seen a number of patients with recurrent carcinoma in situ in grafted skin. One patient, who was 45 years old, had a skinning vulvectomy in 1973 and then presented with rather extensive recurrent carcinoma in situ around the clitoral area in the grafted skin in 1980. She was treated by laser vaporization and has remained disease free since that time. The second case involved a 26-year-old woman who also had a skinning vulvectomy in the early 1970s and presented with a single focus of recurrent carcinoma in situ in the graft of the skin of the perineal area. This case was managed by local excision. The third case was in a patient with a skinning vulvectomy in the early 1970s who then presented in 1985 with multifocal lesions around the perineal area and in the graft on the perineum. She was treated by laser vaporization and has remained disease free since that time. The last patient did not really have carcinoma in situ but bowenoid papulosis and grade 2 vulvar intraepithelial neoplasia, with multiple sites on the grafted skin, which had been placed in 1971.

The authors pointed out the possibility of recurrent carcinoma in situ in grafted skin in patients who have these skinning vulvectomies. There has also been one case of invasive cancer occurring in a skinning vulvectomy.

The authors further pointed out most appropriately that other means of treating these patients, such as local excision and vaporization, may be preferable. Also the importance of careful follow-up cannot be emphasized strongly enough.

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Correction of hypovolemia and central venous pressure monitoring in the management of severe preeclampsia and eclampsia

To the Editors:

I refer to the reports by Clark et al. (Clark SL, Greenspoon JS, Aldahl D, Phelan JP. Severe preeclampsia with persistent oliguria: Management of hemodynamic subsets. AM J OBSTET GYNECOL 1986;154:490-4) and Sibai et al. (Sibai BM, Abdella TN, Spinnato JA, Anderson GD. Eclampsia. V. The incidence of nonpreventable eclampsia. AM J OBSTET GYNECOL 1986; 154:581-6.) The importance of central venous pressure monitoring and correction of hypovolemia with colloid to maintain urine output in severe preeclampsia and eclampsia cannot be overemphasized. An aggressive protocol of management at the Royal Women's Hospital, Melbourne, Australia, has virtually eliminated severe renal and cardiovascular complications. The principles of management are as follows:

1. Volume expansion. Warmed 5% stable plasma protein solution is given under central venous pressure control to restore the central venous pressure to the