eral Food and Drug Administration Obstetrics-Gynecology Devices Panel proceedings. Before discharge, our patients were given thorough instructions in self-palpating uterine contractions, and they also had 24-hour access to nursing support.

Their second concern was our pump dosage protocol. Our regimen was essentially the same as that endorsed by Lam in the January 1989 issue of Contemporary OB-GYN. However, the protocol was modified slightly on the basis of the recommendation of a representative of the pump manufacturer (MiniMed, Inc, Sylmar, Calif) who had extensive experience with terbutaline pump therapy and whose job description included training support personnel on the proper use of the pump. Patients were allowed to self-administer boluses if uterine activity was present, and the mean total dose per day which our patients received was almost identical to the dose used by each of the investigators in their "descriptive" studies of terbutaline pump therapy.

Third, Elliott et al suggest that our patients' labor was in general too advanced when it was arrested with parenteral tocolysis to allow them to benefit from maintenance therapy. Are Elliott et al suggesting that we not use pump therapy for women who have dilatation of ≥3 cm? Elliott et al have never suggested in any of their publications that pump therapy is not efficacious in women with cervical dilatation of ≥3 cm. Our study was specifically designed to include only women who met previously determined criteria for preterm labor, and we were understandably reluctant to enroll women who had only preterm contractions. Patients who are at highest risk for preterm delivery would logically be the best candidates for maintenance therapy. We believe that our observed lack of efficacy in this population is far more telling than would be data from women at lower risk of preterm delivery. It appears that Elliott et al are selectively expressing concerns that support their personal biases. Furthermore, it is our institutional practice to withhold parenteral tocolysis from women at ≥34 weeks' gestation because the risk of major neonatal morbidity at this gestational age is minimal. All the neonates who were delivered between 34 and 37 weeks' gestation did well.

Fourth, the investigators expressed concern that our dropout rate was substantial and that our population was composed largely of indigent women. Does the fact that our patients were predominantly receiving public assistance make our findings less credible? The dropout rates in both the terbutaline and the placebo groups were high. When we compared the outcomes of the women who continued terbutaline pump therapy with those of the women who continued placebo therapy until delivery, active pump therapy resulted in a 2-day-longer prolongation of pregnancy, a clinically insignificant difference.

We concur with the final point of Elliott et al that the rate of major adverse effects with terbutaline pump therapy is low; however, serious complications (including a maternal death) have been reported. We do not believe that any adverse effects are acceptable if an expensive technology with no proven benefit has been used outside properly designed clinical trials.

We understand that Elliott et al have published extensively on their experience with terbutaline pump therapy, but they have published no randomized double-blind trials in peer-reviewed journals that confirm the effectiveness of this treatment. Descriptive studies without appropriate controls do not provide sufficient evidence on which to base expensive and occasionally hazardous therapeutic recommendations. Although we agree that no study is perfect, the current weight of controlled clinical evidence does not support the efficacy of the terbutaline pump for maintenance therapy after preterm labor arrest with parenteral tocolytic therapy. Clearly, The American College of Obstetricians and Gynecologists and the Food and Drug Administration agree with these conclusions. Once again, we urge Elliott et al to demonstrate, in properly designed randomized, controlled trials, a role for terbutaline pump therapy in preventing or reducing premature birth.

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REFERENCES

Reply
To the Editors: Morrison has been a leading proponent of the technology of home uterine activity monitoring with or without the addition of subcutaneous terbutaline pump tocolytic therapy. I therefore welcome the opportunity that his group has provided to again point out the following: (1) Daily nursing contact or daily home uterine contraction monitoring is not the standard of care. (2) Elliott et al must provide data from prospective randomized double-blind trials published in peer-reviewed journals to prove the effectiveness of the terbutaline pump, with or without home uterine activity monitoring. (3) Elliott et al must provide similar data regarding the effectiveness of subcutaneous terbutaline therapy with consideration of individual body mass index, contraction activity, contraction status, and time-of-day dosage sched-
All cases of intrauterine fetal death should be evaluated for parvovirus B19 viral deoxyribonucleic acid

To the Editors: We read with interest the article “Stillbirth Evaluation: What Tests Are Needed?” (Incerpi MH, Miller DA, Samadi R, Settlage RH, Goodwin TM. Stillbirth evaluation: What tests are needed? Am J Obstet Gynecol 1998;178:1121-5). Although economic aspects are of great importance today the essential point, as the authors have shown, is to know your own results before deciding to omit certain laboratory tests.

Recently a report of parvovirus B19 as a significant cause of middle trimester fetal loss has been published. However, nonhydropic fetal loss in the third trimester associated with parvovirus B19 has hitherto not been a dominant issue. In a prospective study of the incidence of parvovirus B19 antibodies in a population of pregnant women, one woman with no B19 immunoglobulin (Ig) G or IgM antibodies was delivered of a nonhydropic, stillborn baby at 37 weeks' gestation in December 1991. Parvovirus B19–specific deoxyribonucleic acid (DNA) was found in the placental tissue as well as in maternal blood samples collected 3 weeks before the death of the fetus and at delivery. The mother did not acquire antibodies against parvovirus B19 until 6 months after delivery. Because no other cause of the intrauterine fetal death was found, we proposed that the parvovirus infection caused the fetal loss, possibly because of a slow immunologic response to the infection by the mother. This case was the incentive for studying parvovirus B19 in all cases of intrauterine fetal death in our department.

Since 1992 all women admitted to Danderyd Hospital with intrauterine fetal death after 28 weeks’ gestation have been examined for IgG and IgM antibodies against parvovirus B19 in serum and for parvovirus B19 DNA by nested polymerase chain reaction in maternal serum and in placental tissue. During the 1992 to 1997 study period 77 cases of intrauterine fetal death occurred. The total number of deliveries during that time was 28,548. Parvovirus B19 DNA was found in placental tissue as well as in maternal serum in a total of 6 cases of third-trimester fetal loss (n = 6/77, 8%). No patient had shown any obvious clinical signs of infection by parvovirus B19.

Parvovirus B19 is known mainly to affect erythropoiesis, causing fetal anemia and hydrops. The virus can affect other systems as well and has a particular affinity for the liver and myocardial cells. In the stillborn baby, however, the autolysis usually makes it impossible to investigate signs of myocarditis, for example.

Viral infections in pregnancy can be menacing to the fetus. In 2 of our 6 cases the period before the women acquired antibodies against parvovirus B19 was considerably prolonged. The question arises whether these fetuses were especially vulnerable because of lack of maternal antibody protection. Estimation of the parvovirus B19 IgG and IgM antibody serum levels was obviously not sufficient to confirm the diagnosis in these cases, because the viral infection was not identified until the maternal serum and placenta were examined for parvovirus B19 DNA. We propose that examination of the placenta and maternal serum for parvovirus B19 DNA should be carried out in all cases of intrauterine fetal death.

At Danderyd Hospital women who have had an intrauterine fetal death are examined according to a standardized protocol after giving consent. The protocol includes examination of maternal blood samples for the Kleihauer-Beck test; antibodies against Toxoplasma gondii, rubella, cytomegalovirus, herpes simplex virus, parvovirus B19, and Listeria monocytogenes; thyroid hormone concentrations and thyroid hormone antibody concentrations. It also includes bacterial culture from the cervix. Chromosomal investigation is performed, and both bacterial and viral cultures from amniotic fluid are examined in most cases. After birth the fetal heart blood is sampled for bacterial culture. A microscopic examination of the placenta is performed, as is an analysis for parvovirus DNA in the placenta and in maternal serum. Autopsy of the fetus is recommended.

When an intrauterine fetal death occurs it certainly helps the parents if a rational explanation can be given. Unfortunately, no explanation has been found by means of our protocol in 30% to 40% of the cases of intrauterine fetal death in our hospital. We believe that the search